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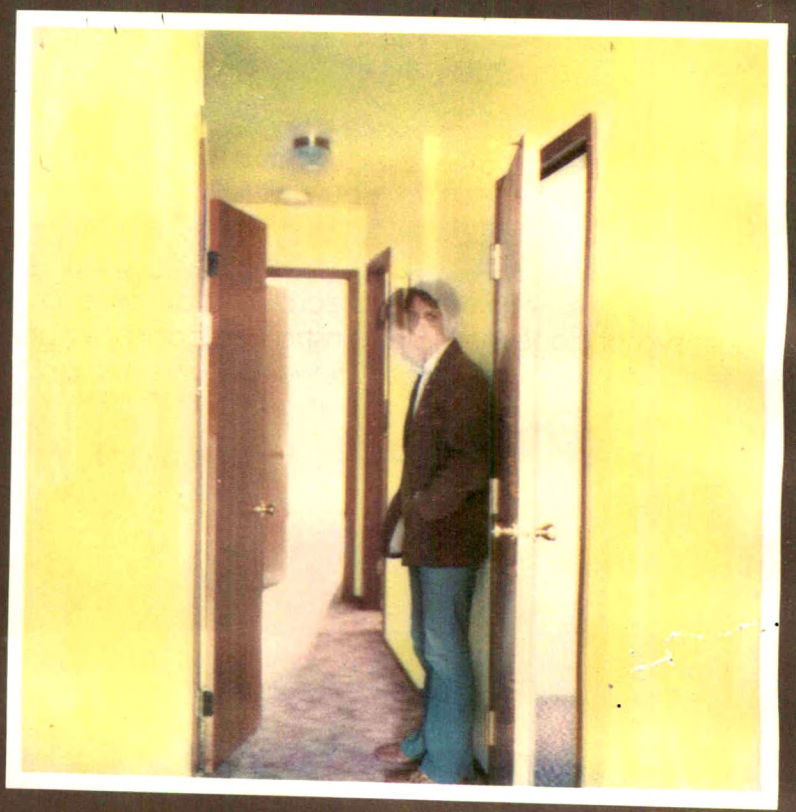
Discriminating Features of Borderline Patients
By John G. Gunderson and Jonathan E. Kolb

Thirteen Years Before the Masthead: The Editor Retires

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Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

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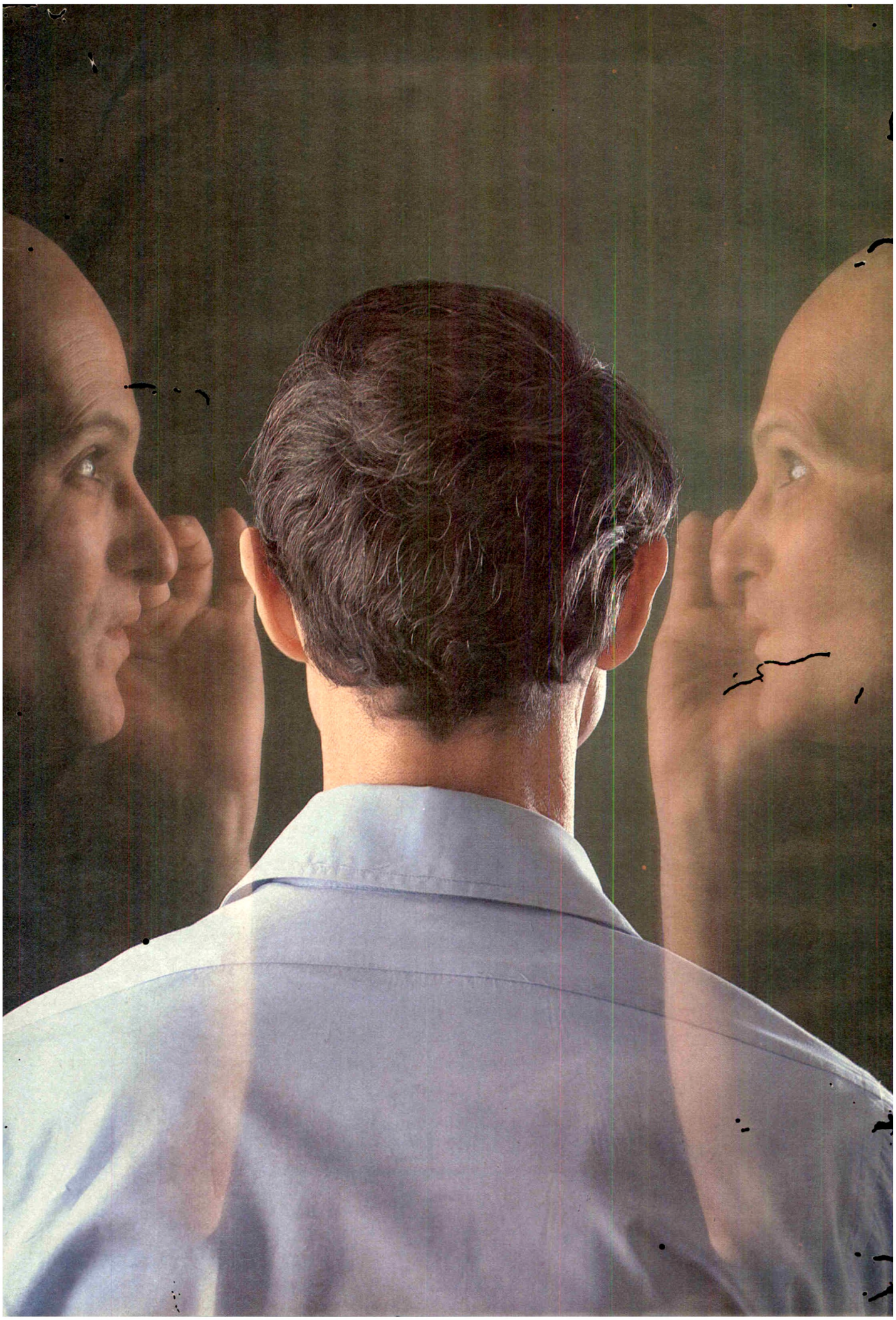
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The August 1978 issue of

The American Journal of Psychiatry

will feature

- Presidential Papers: 1978



VOICES

...accusatory, derogatory, condemning, commanding.

Navane (thiothixene) rapidly controls auditory hallucinations as well as the agitation and hostility patterns they frequently generate.

Rapid control of psychotic symptoms on admission

Navane provides effective, rapid control of the major symptoms of psychosis including hallucinations, delusions, agitation, hostility, and thought disorders.[1-5] Navane promotes rapid progress, usually without the over-sedation or cardiovascular problems that can impede the therapeutic course.

Continued long-term improvement

Because Navane provides long-term outpatient control of psychotic symptoms, it can enable many patients to function effectively at home and on the job and to adjust rapidly to the community.[6]

Effectiveness rarely compromised by adverse reactions

Navane allows patients to remain alert and active, seldom causing over-sedation or drowsiness.[7] Hypotension and other cardiovascular reactions[5,6,8] are seldom reported and the occurrence of unpleasant anticholinergic side effects such as dry mouth or constipation is rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled through dosage adjustments or antiparkinson agents.

Rapid control
Long-term improvement

Navane®

(thiothixene) (thiothixene HCl)

Capsules 1 mg., 2 mg., 5 mg., 10 mg., 20 mg. Concentrate 5 mg./ml. Intramuscular 2 mg./ml.

*For prescribing information, including adverse reactions and contraindications, please see following page of this advertisement.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals
New York, New York 10017

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg
(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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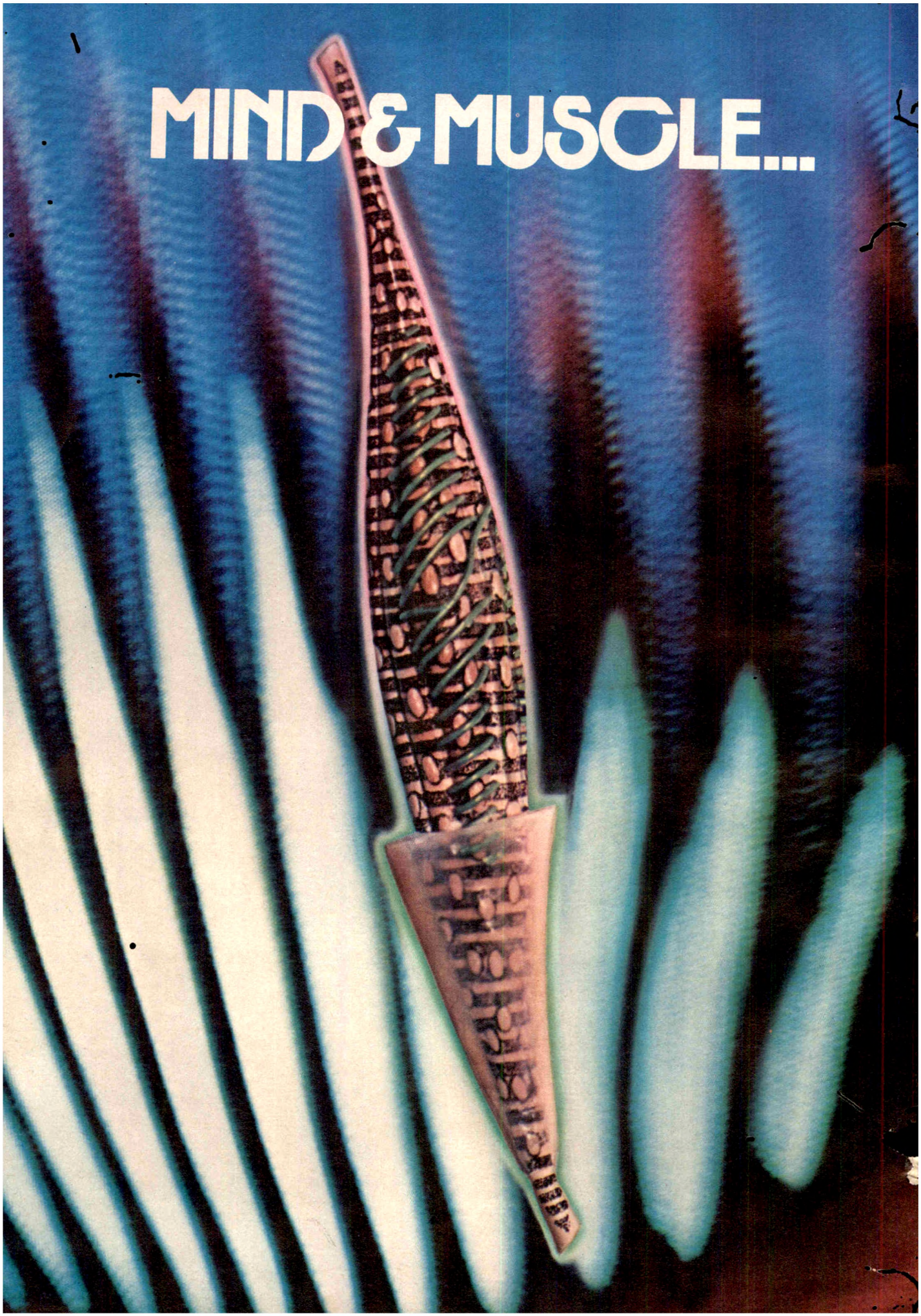
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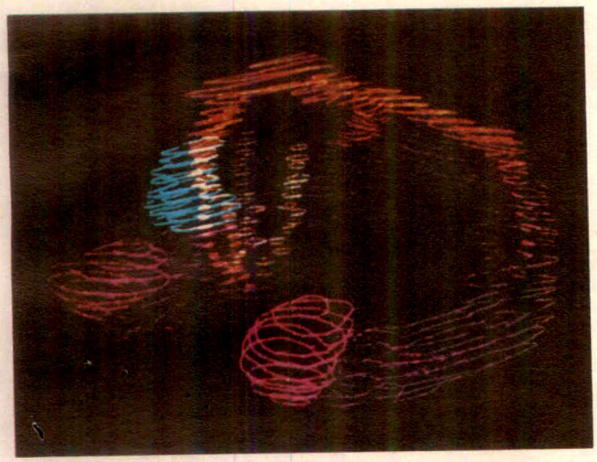
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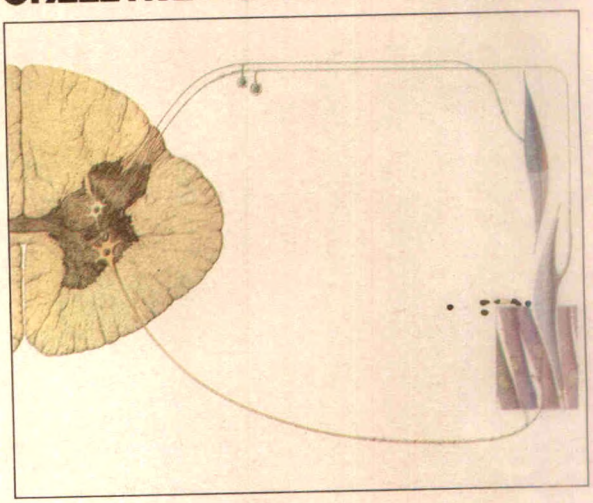
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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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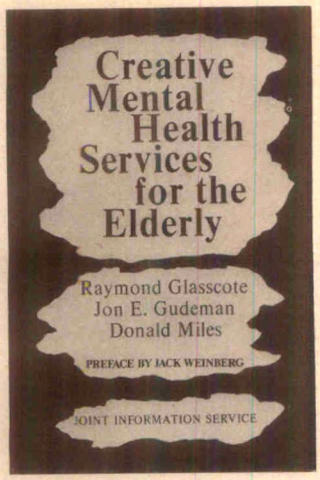
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The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



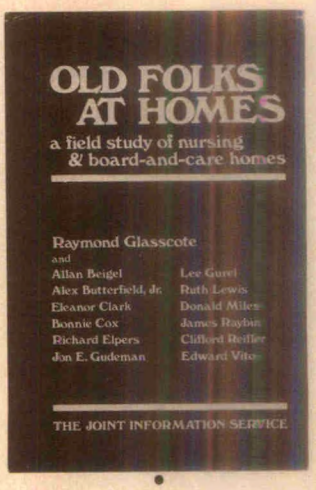
This carefully documented book reports a field study of ten programs, four in America and six in England and Scandinavia, which serve as practical demonstrations of creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elderly residents of several trailer parks
- a high school for the elderly which sends its students abroad for study trips
- a "Human Development Project" that focuses on responding to the psychological needs of the elderly
- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting document becomes a handbook, and, as well, a forum through which some of the world's most experienced practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Social Gerontology, characterizes this volume as "a masterful job of putting everything in relief . . . the guidelines for action are right here."

190 pages. Casebound. Price $8.50.



Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

The teams were surpised to learn that care and quality of life in many of the nursing and board-and-care homes they visited were better—sometimes dramatically better—than that in the mental health facilities and often at lower cost.

This unique publication provides an overview of all facilities visited plus vignettes of approximately half. Federal and state rules and regulations are also expertly synthesized into brief and readable style.

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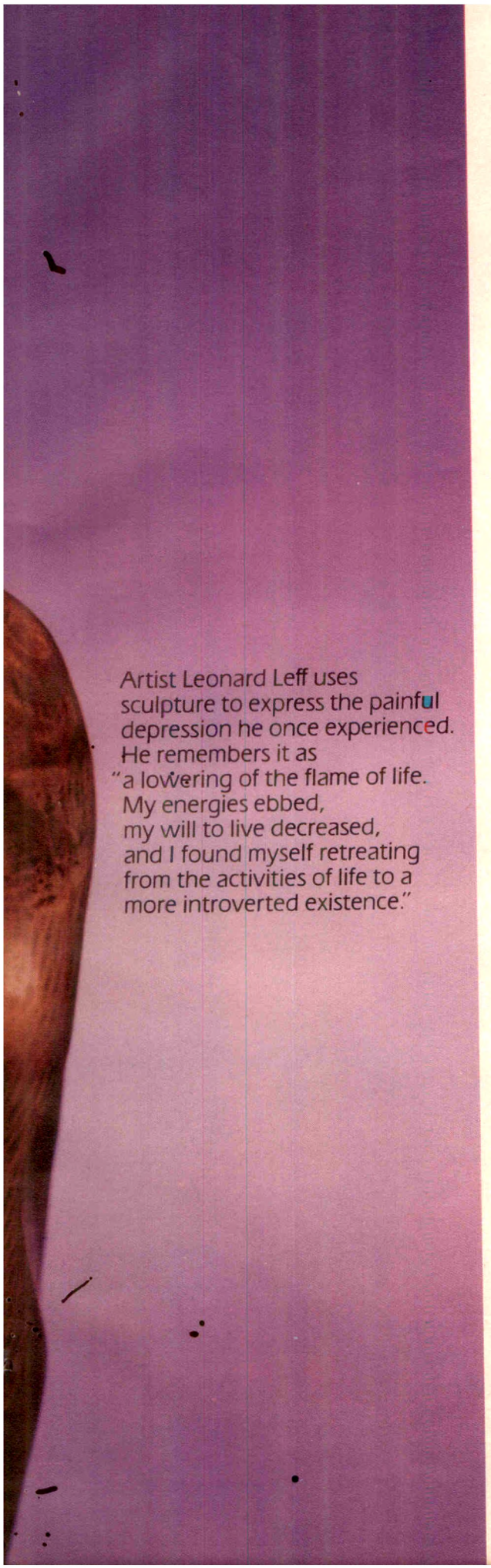
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AJP



Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

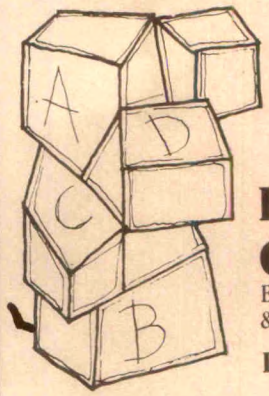
B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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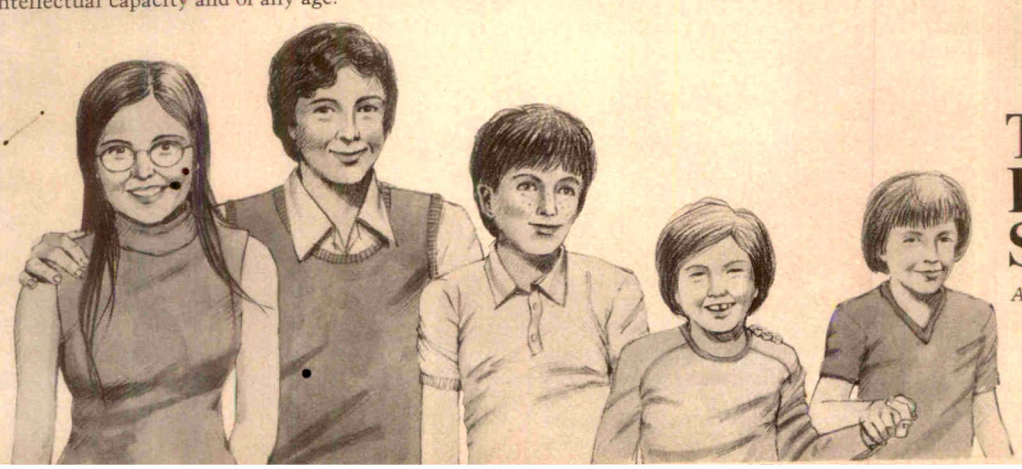
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molindone HCl

- an effective antipsychotic agent.
- not a phenothiazine

P 24, 350

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NO incidence	Tolerance development Hepatotoxic changes Ophthalmological changes Excessive weight gain Impotence Decreased libido Skin pigmentation Thyroid function changes Persistent tardive dyskinesia
LOW incidence	Sedation Adverse drug interactions Hypotension Hematological changes Lactation Amenorrhea Autonomic changes Skin rash Nonpersistent symptoms of tardive dyskinesia
MODERATE incidence	Extrapyramidal reactions

*This table compiled from clinical and field reports, according to the best available information.

Further advantages with MOBAN®

The pharmacological effect from a single oral dose persists for 24-36 hours, permitting *h.s.* dosage, once the daily dosage level is established.

Several clinicians have reported that obese patients on MOBAN® frequently experienced a marked reduction in weight toward normal weight.

How to use MOBAN®

The usual starting dose is 50-75 mg/day. You can increase to 100 mg/day in 3-4 days. Titrate individually up or down depending on patient response. Some chronic, treatment-resistant patients may require an increase to 400 mg/day. (However, the long-term safety of 400 mg/day has not been established.) Please refer to complete prescribing information for further dosage instructions.

For complete prescribing information, please see next page.

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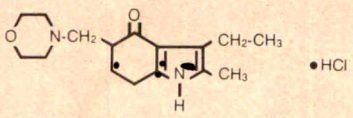
MOBAN®

molindone HCl

5 mg	10 mg	25 mg
Endo	Endo	Endo

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN® (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN® being excreted in urine and feces.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in the pregnant patient have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose—
20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose—
20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose—
5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN® Caution should be exercised where increased activity may be harmful.

MOBAN® does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN® has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological

Extrapyramidal Reactions

Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia

Motor restlessness may occur early.

Parkinson Syndrome

Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome

Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System

Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological

There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN® may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects

Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with MOBAN® therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN®.

Cardiovascular

Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological

Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN® (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN®.

Skin

Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN® usage alone.

MOBAN® (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN® is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia

Although rarely reported with MOBAN® (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50-75 mg/day.

—Increase to 100 mg/day in 3 or 4 days.

—Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.

—An increase to 225 mg/day may be required in patients with severe symptomatology.

—Some chronic, treatment resistant patients, may require up to 400 mg/day (however, the long term safety of 400 mg/day has not been established).

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

Dosages may be administered once a day.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN® in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN® in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles of 100 and 1000 with potencies and colors as follows: 5 mg orange, 10 mg lavender, 25 mg light green.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

MOBAN* is an Endo Registered U.S. Trademark • U.S. Pat. 3,491,093

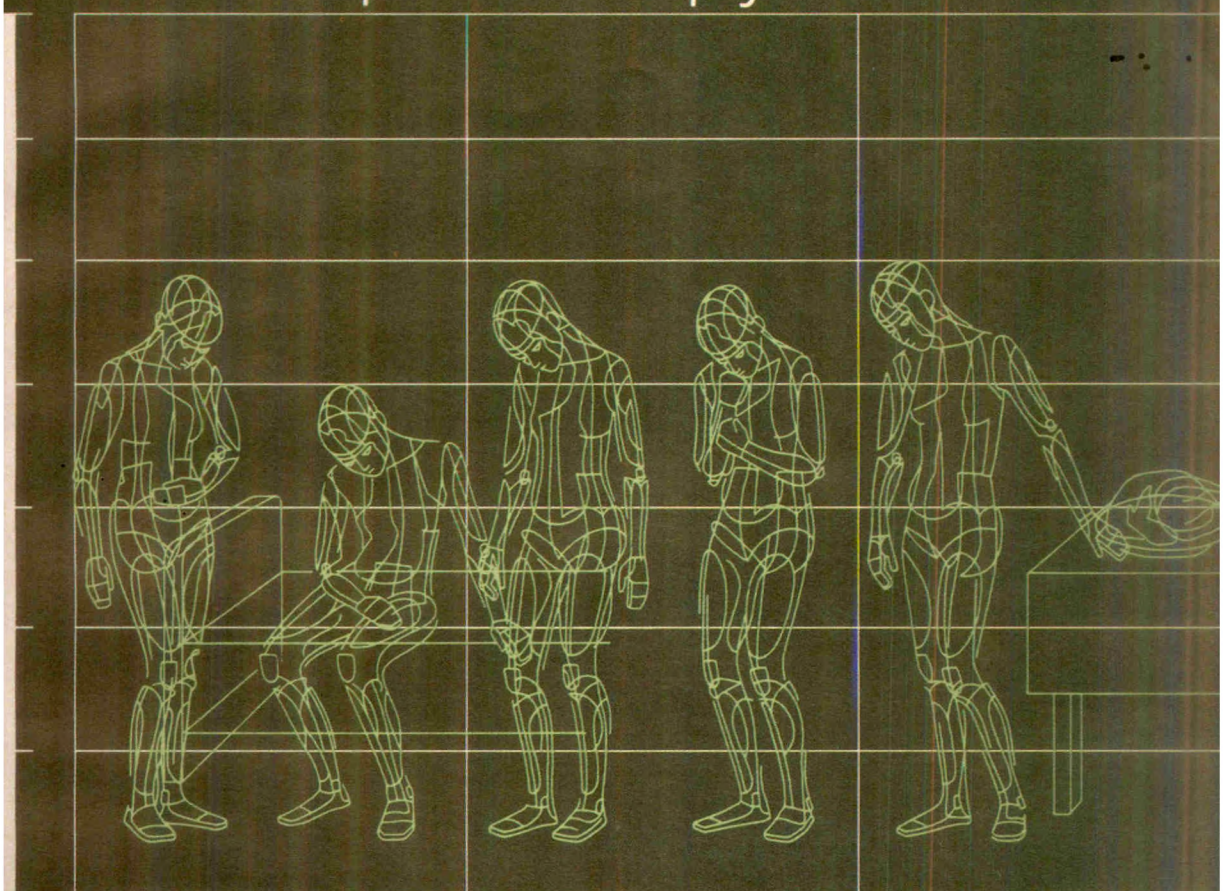
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Garden City, New York 11530

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EDO-014M8-77

Within one week
A better response
For twice as many patients

compared to amitriptyline alone



New Limbitrol® IV

For rapid relief of symptoms in moderate to severe depression and anxiety

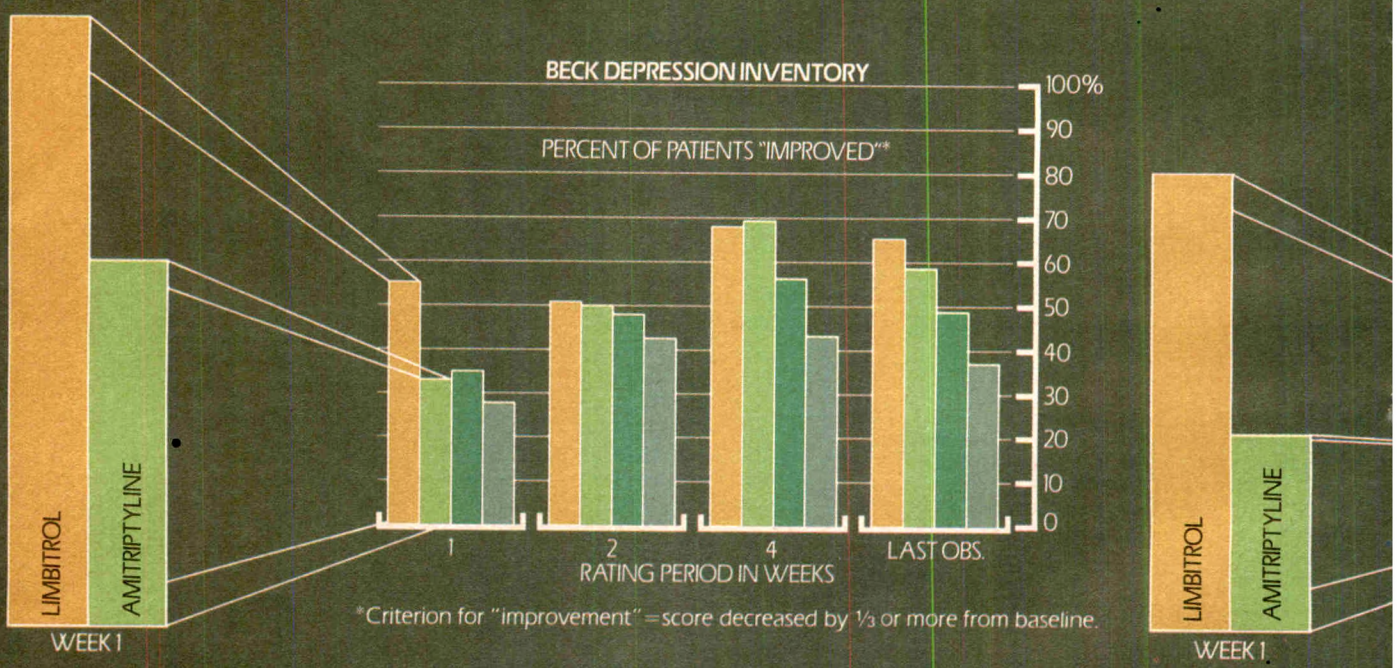
ROCHE

Please see last page of this advertisement for complete product information.

Within one week Twice as many patients improved–



*Criterion for "improvement" = score decreased by ½ or more from baseline.

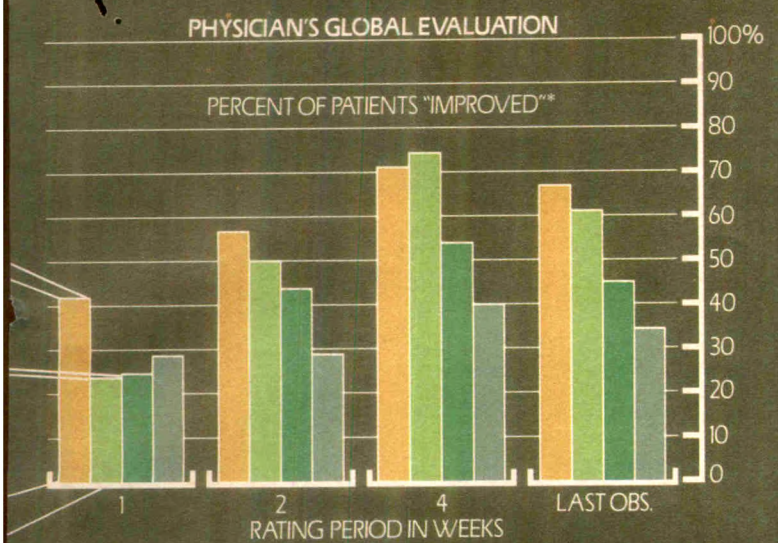


*Criterion for "improvement" = score decreased by ⅓ or more from baseline.

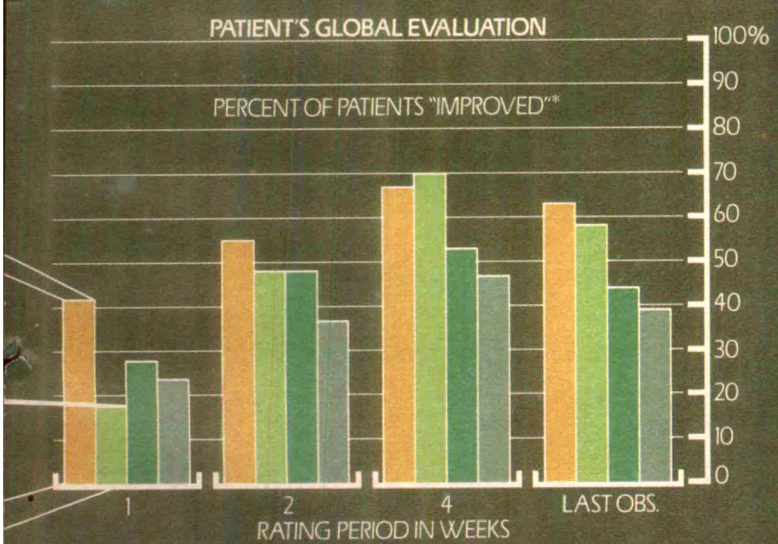
New Limbitrol

Provides the well established antidepressant effect of amitriptyline with the proven antianxiety effect of Librium® (chlordiazepoxide HCl)

sooner than with amitriptyline alone



*Criterion for "improvement" = ratings of "much better" or "very much better."



*Criterion for "improvement" = ratings of "much better" or "very much better."

In a rigidly controlled nationwide multicenter study

The rating scales agree

- ☐ Patients respond faster
  - ☐ Limbitrol—approximately 2/3 of those who will improve will have done so by week one
  - ☐ Amitriptyline—only about 1/3 of those who will eventually respond will have done so by week one
- ☐ Greater overall improvement
- ☐ High correlation between major indices

LIMBITROL
AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO

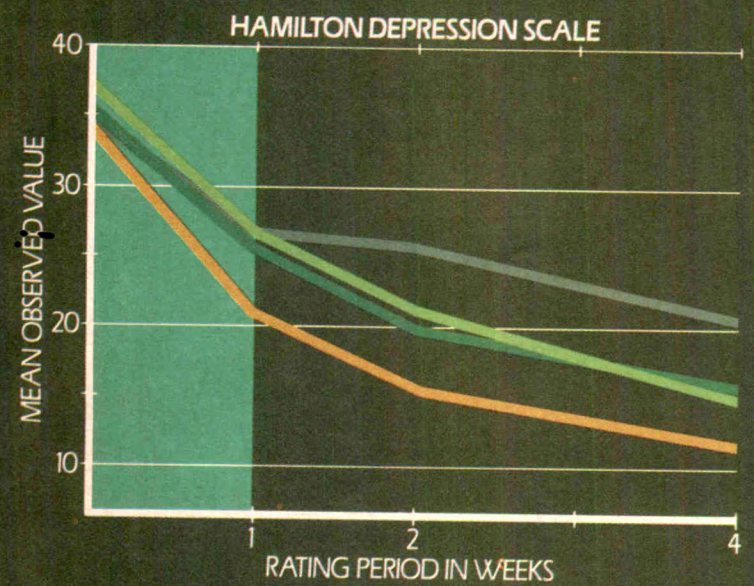
The data presented in this booklet are based on a rigidly controlled nationwide multicenter study of 279 patients with a diagnosis of primary depression and anxiety of moderate to severe degree

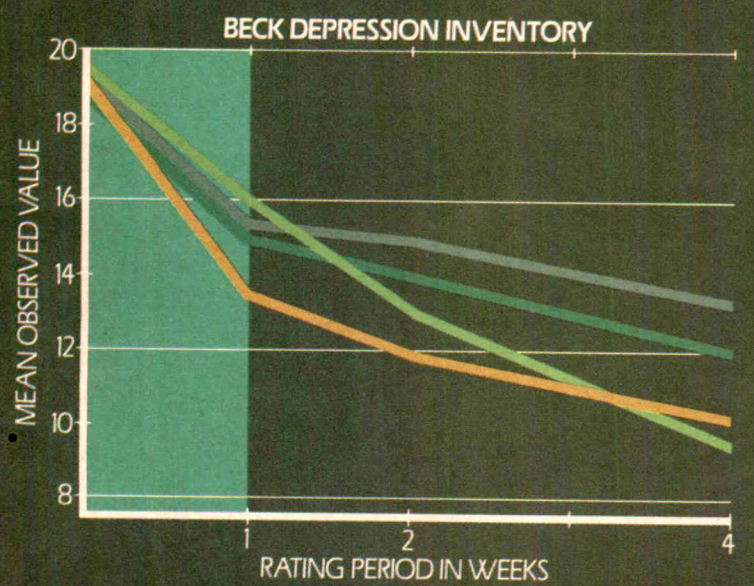
For rapid relief of moderate to severe depression and anxiety

ROCHE

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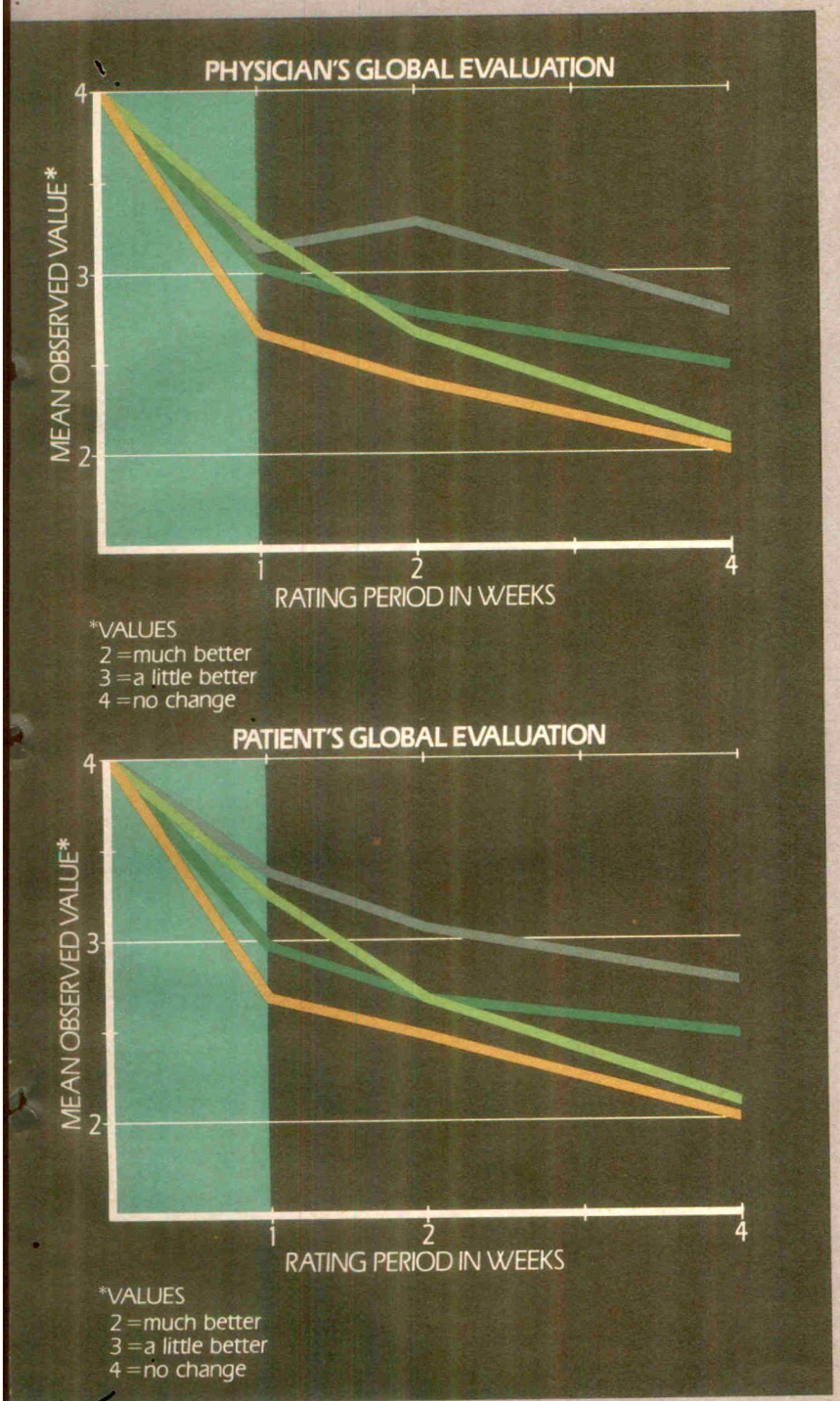
Within one week
Marked reduction in severity of symptoms





New Limbitrol®

–better than amitriptyline alone



In a rigidly controlled nationwide multicenter study

The symptoms subside

Hamilton Depression Scale Items

- ☐ insomnia
- ☐ psychic anxiety
- ☐ somatic anxiety
- ☐ agitation
- ☐ suicidal ideation
- ☐ worthlessness

Beck Depression Inventory Items

- ☐ pessimism
- ☐ dissatisfaction
- ☐ guilt
- ☐ social withdrawal
- ☐ anorexia

Physician's and Patient's Global Evaluations

As measured by the Physician's and Patient's Global Evaluations, Limbitrol was rated superior to amitriptyline.

LIMBITROL
AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO

For rapid relief of moderate to severe depression and anxiety

ROCHE

Please see last page of this advertisement for complete product information.

Within one week
Twice as many patients responded as



New Limbitrol®

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

with amitriptyline alone

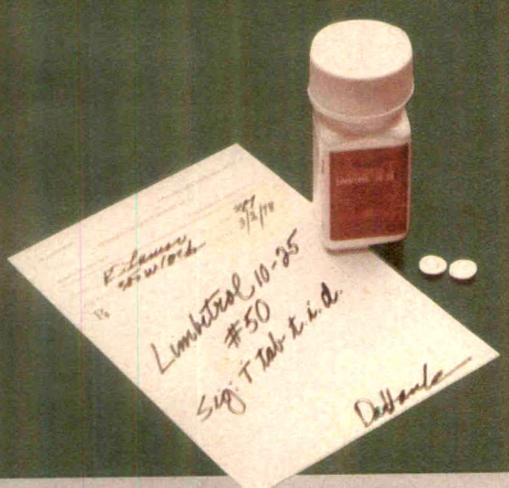
In a rigidly controlled nationwide multicenter study

The therapy succeeds

- ☐ Better patient compliance
- ☐ Lower dropout rate
  - ☐ The dropout rate due to side effects was lower with Limbitrol than with amitriptyline alone (Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)
  - ☐ The dropout rate due to therapeutic failure was lower with Limbitrol (Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)

(While the frequency of side effects was comparable between Limbitrol and amitriptyline, drowsiness occurred more often with Limbitrol.)

- ☐ Free of major phenothiazine drawbacks



How to initiate and maintain therapy

For most patients

☐ Limbitrol 10-25 is recommended in an initial dosage of three or four tablets daily in divided doses.

For elderly patients and patients who do not tolerate higher doses

☐ Limbitrol 5-12.5 is recommended in an initial dosage of three or four tablets daily in divided doses.

For all patients

- ☐ Dosage should be individualized on the basis of symptom severity and response.
  - ☐ Because drowsiness is a common side effect, it may be preferable to give the larger portion of the total daily dose at bedtime. Not only will this help minimize problems with daytime drowsiness, it may also help relieve a major target symptom—insomnia.
  - ☐ Some patients respond to smaller doses or to a single dose at bedtime.
  - ☐ Dosage may be increased up to six tablets or decreased to two tablets daily as necessary.
- ☐ Once a satisfactory response is obtained, reduce dosage to the smallest amount needed to maintain remission.
- ☐ Warning: Since Limbitrol contains chlordiazepoxide, a minor tranquilizer, usage during the first trimester of pregnancy should almost always be avoided.

For rapid relief of moderate to severe depression and anxiety

ROCHE

Please see last page of this advertisement for complete product information.

Limbitrol®
Tablets

Tranquilizer—Antidepressant

Limbitrol 10-25

Each tablet contains 10 mg chlordiazepoxide and 25 mg amitriptyline in the form of the hydrochloride salt.

Limbitrol 5-12.5

Each tablet contains 5 mg chlordiazepoxide and 12.5 mg amitriptyline in the form of the hydrochloride salt.

Description: Limbitrol combines in a tablet for oral administration, chlordiazepoxide, an agent for the relief of anxiety and tension, and amitriptyline, an antidepressant.

Chlordiazepoxide is a benzodiazepine with the formula 7-chloro-2-(methylamino)-5-phenyl-3*H*-1,4-benzodiazepine 4-oxide. It is a slightly yellow crystalline material and is insoluble in water. The molecular weight is 299.76.

Amitriptyline is a dibenzocycloheptadiene derivative. The formula is 10,11-dihydro-*N*,*N*-dimethyl-5H-dibenzo [a, d] cycloheptene-Δ^{5Y}-propylamine hydrochloride. It is a white or practically white crystalline compound that is freely soluble in water. The molecular weight is 313.87.

Actions: Both components of Limbitrol exert their action in the central nervous system. Extensive studies with chlordiazepoxide in many animal species suggest action in the limbic system. Recent evidence indicates that the limbic system is involved in emotional response. Taming action was observed in some species. The mechanism of action of amitriptyline in man is not known, but the drug appears to interfere with the reuptake of norepinephrine into adrenergic nerve endings. This action may prolong the sympathetic activity of biogenic amines.

Indications: Limbitrol is indicated for the treatment of patients with moderate to severe depression associated with moderate to severe anxiety.

The therapeutic response to Limbitrol occurs earlier and with fewer treatment failures than when either amitriptyline or chlordiazepoxide is used alone.

Symptoms likely to respond in the first week of treatment include: insomnia, feelings of guilt or worthlessness, agitation, psychic and somatic anxiety, suicidal ideation and anorexia.

Contraindications: Limbitrol is contraindicated in patients with hypersensitivity to either benzodiazepines or tricyclic antidepressants. It should not be given concomitantly with a monoamine oxidase inhibitor. Hyperpyretic crises, severe convulsions and deaths have occurred in patients receiving a tricyclic antidepressant and a monoamine oxidase inhibitor simultaneously. When it is desired to replace a monoamine oxidase inhibitor with Limbitrol, a minimum of 14 days should be allowed to elapse after the former is discontinued. Limbitrol should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. This drug is contraindicated during the acute recovery phase following myocardial infarction.

Warnings: Because of the atropine-like action of the amitriptyline component, great care should be used in treating patients with a history of urinary retention or angle-closure glaucoma. In patients with glaucoma, even average doses may precipitate an attack. Severe constipation may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressant drugs, particularly when given in high doses, have been reported to produce arrhythmias, sinus tachycardia and prolongation of conduction time. Myocardial infarction and stroke have been reported in patients receiving drugs of this class.

Usage in Pregnancy: Safe use of Limbitrol during pregnancy and lactation has not been established. Because of the chlordiazepoxide component, please note the following:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physicians about the desirability of discontinuing the drug.

Physical and Psychological Dependence: Physical and psychological dependence to Limbitrol have not been reported. However, since physical and psychological dependence to chlordiazepoxide have been reported rarely, caution must be exercised in administering Limbitrol to individuals known to be addiction-prone or to those whose history suggests they may increase the dosage on their own initiative.

Withdrawal symptoms from Limbitrol have not been reported. However, withdrawal symptoms following abrupt cessation of prolonged therapy with either component alone have been reported. With amitriptyline, these have been noted to consist of nausea, headache and malaise; for chlordiazepoxide, the symptoms (including convulsions) are similar to those seen with barbiturates.

Precautions: *General:* Use with caution in patients with a history of seizures.

Close supervision is required when Limbitrol is given to hyperthyroid patients or those on thyroid medication.

The usual precautions should be observed when treating patients with impaired renal or hepatic function.

Patients with suicidal ideation should not have easy access to large quantities of the drug. The possibility of suicide in depressed patients remains until significant remission occurs.

Information for the Patient: Because of the sedative effects of Limbitrol, patients should be cautioned about combined effects with alcohol or other CNS depressants. The additive effects may produce a harmful level of sedation and CNS depression.

Patients receiving Limbitrol should be cautioned against engaging in hazardous occupations requiring complete mental alertness, such as operating machinery or driving a motor vehicle.

Essential Laboratory Tests: Patients on prolonged treatment should have periodic liver function tests and blood counts.

Drug and Treatment Interactions: Because of its amitriptyline component, Limbitrol may block the antihypertensive action of guanethidine or compounds with a similar mechanism of action.

The effects of concomitant administration of Limbitrol and other psychotropic drugs have not been evaluated. Sedative effects may be additive.

The drug should be discontinued several days before elective surgery.

Concurrent administration of ECT and Limbitrol should be limited to those patients for whom it is essential.

Pregnancy: See WARNINGS section.

Nursing Mothers: It is not known whether this drug is excreted in human milk. As a general rule, nursing should not be undertaken while a patient is on a drug, since many drugs are excreted in human milk.

Pediatric Use: Safety and effectiveness in children below the age of 12 years have not been established.

Elderly Patients: In elderly and debilitated patients it is recommended that dosage be limited to the smallest effective amount to preclude the development of ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Adverse reactions to Limbitrol are those associated with the use of either component alone. Most frequently reported were drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Other side effects occurring less commonly included vivid dreams, impotence, tremor, confusion and nasal congestion. Many symptoms common to the depressive state, such as anorexia, fatigue, weakness, restlessness and lethargy, have been reported as side effects of treatment with both Limbitrol and amitriptyline.

Granulocytopenia, jaundice and hepatic dysfunction of uncertain etiology have also been observed rarely with Limbitrol. When treatment with Limbitrol is prolonged, periodic blood counts and liver function tests are advisable.

Note: Included in the listing which follows are adverse reactions which have not been reported with Limbitrol. However, they are included because they have been reported during therapy with one or both of the components or closely related drugs.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergic: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: There has been limited experience with Limbitrol overdosage *per se;* the manifestations of overdosage and recommendations for treatment are based on clinical experience with its components. Primary concern should be with the dangers associated with amitriptyline overdosage. Deaths by deliberate or accidental overdosage have occurred with this class of drugs.

All patients suspected of having an overdosage of Limbitrol should be admitted to a hospital as soon as possible.

Manifestations: High doses may cause drowsiness, temporary confusion, disturbed concentration or transient visual hallucinations. Overdosage may cause hypothermia, tachycardia and other arrhythmias, ECG evidence of impaired conduction (such as bundle branch block), congestive heart failure, dilated pupils, convulsions, severe hypotension, stupor and coma. Other symptoms may be agitation, hyperactive reflexes, muscle rigidity, vomiting, hyperpyrexia or any of those listed under Adverse Reactions.

Treatment: Empty the stomach as quickly as possible by emesis or lavage. In the comatose patient a cuff endotracheal tube should be placed in position prior to either of these measures. The instillation of activated charcoal into the stomach also should be considered. If the patient is stuporous but responds to stimuli, only close observation and nursing care may be required. It is essential to maintain an adequate airway and fluid intake. Body temperature should be watched closely and appropriate measures taken should deviations occur.

The intramuscular or slow intravenous administration of 1 to 3 mg in adults (or 0.5 mg in children) of physostigmine salicylate (Antilirium)[1-3] has been reported to reverse the manifestations of amitriptyline overdosage. Because of its relatively short half-life, additional doses may be needed at intervals of 30 minutes to 2 hours.

Convulsions may be treated by the use of an inhalation anesthetic rather than the use of barbiturates. Cardiac monitoring is advisable, and the cautious use of digitalis or other antiarrhythmic agents should be considered if serious cardiovascular abnormalities occur. Serum potassium levels should be monitored and kept within normal limits by the use of appropriate I.V. fluids. Standard measures including oxygen, I.V. fluids, plasma expanders and corticosteroids may be used to control circulatory shock.

Dialysis is unlikely to be of value, as it has not proven useful in overdosages of either amitriptyline or chlordiazepoxide. Since many suicidal attempts involve multiple drugs including barbiturates, the possibility of dialysis being beneficial for removal of other drugs should not be overlooked.

Treatment should be continued for at least 48 hours, along with cardiac monitoring in patients who do not respond to therapy promptly. Since relapses are frequent, patients should be hospitalized until their conditions remain stable without physostigmine for at least 24 hours.

Since overdosage is often deliberate, patients may attempt suicide by other means during the recovery phase.

References:

1. Granacher RP, Baldessarini RJ: Physostigmine: Its use in acute anticholinergic syndrome with antidepressant and antiparkinson drugs. *Arch Gen Psychiatry 32*:375-380, March 1975
2. Burks JS, Walker JE, Rumack BH, Ott JE: Tricyclic antidepressant poisoning: Reversal of coma, choreoathetosis, and myoclonus by physostigmine. *JAMA 230*:1405-1407, Dec. 9, 1974
3. Snyder BD, Blonde L, McWhirter WR: Reversal of amitriptyline intoxication by physostigmine. *JAMA 230*:1433-1434, Dec. 9, 1974

Dosage and Administration: Optimum dosage varies with the severity of the symptoms and the response of the individual patient. When a satisfactory response is obtained, dosage should be reduced to the smallest amount needed to maintain the remission. The larger portion of the total daily dose may be taken at bedtime. In some patients, a single dose at bedtime may be sufficient. In general, lower dosages are recommended for elderly patients.

Limbitrol 10-25 is recommended in an initial dosage of three or four tablets daily in divided doses; this may be increased to six tablets daily as required. Some patients respond to smaller doses and can be maintained on two tablets daily.

Limbitrol 5-12.5 in an initial dosage of three or four tablets daily in divided doses may be satisfactory in patients who do not tolerate higher doses.

How Supplied: White, film-coated tablets, each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and blue, film-coated tablets, each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

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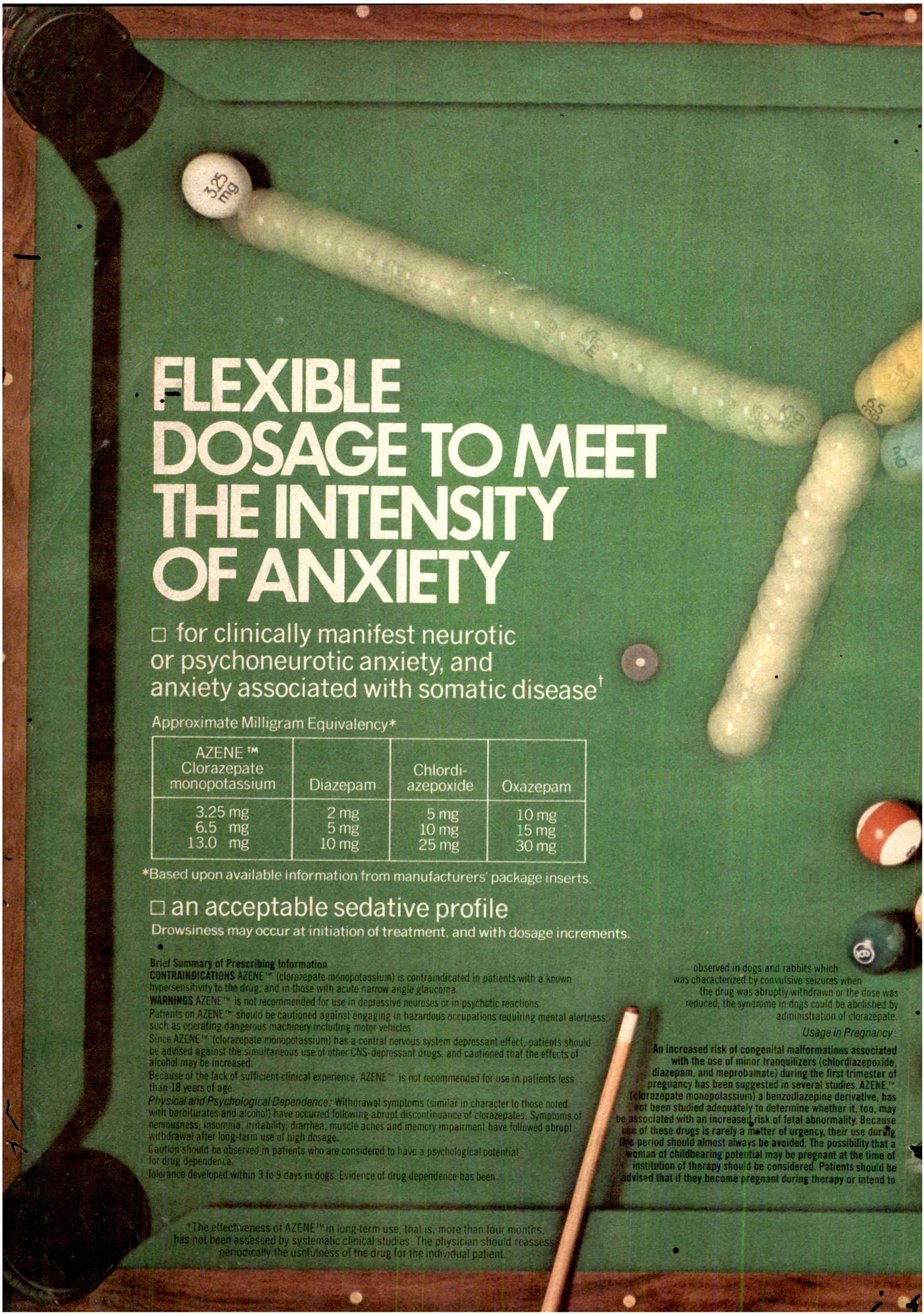
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FLEXIBLE DOSAGE TO MEET THE INTENSITY OF ANXIETY

☐ for clinically manifest neurotic or psychoneurotic anxiety, and anxiety associated with somatic disease†

Approximate Milligram Equivalency*

AZENE ™ Clorazepate monopotassium	Diazepam	Chlordi-azepoxide	Oxazepam
3.25 mg	2 mg	5 mg	10 mg
6.5 mg	5 mg	10 mg	15 mg
13.0 mg	10 mg	25 mg	30 mg

*Based upon available information from manufacturers' package inserts.

☐ an acceptable sedative profile

Drowsiness may occur at initiation of treatment, and with dosage increments.

Brief Summary of Prescribing Information

CONTRAINDICATIONS AZENE™ (clorazepate monopotassium) is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS AZENE™ is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on AZENE™ should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since AZENE™ (clorazepate monopotassium) has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, AZENE™ is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence: Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepates. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

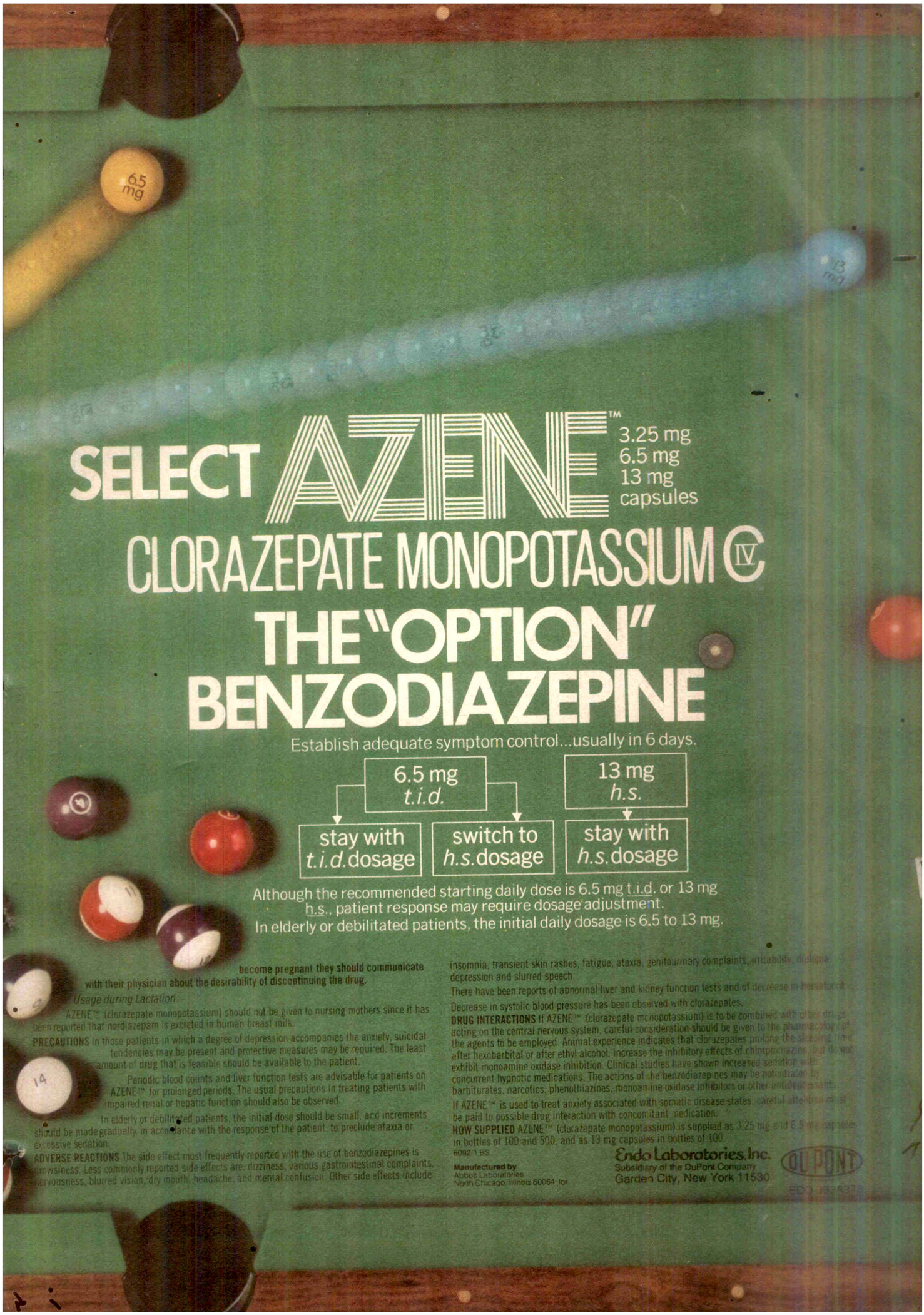
Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Tolerance developed within 3 to 9 days in dogs. Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced, the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. AZENE,™ (clorazepate monopotassium) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to

†The effectiveness of AZENE™ in long-term use, that is, more than four months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.


SELECT AZENE™
3.25 mg
6.5 mg
13 mg
capsules
CLORAZEPATE MONOPOTASSIUM C-IV
THE "OPTION" BENZODIAZEPINE
Establish adequate symptom control...usually in 6 days.
6.5 mg t.i.d.
13 mg h.s.
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switch to h.s. dosage
stay with h.s. dosage
Although the recommended starting daily dose is 6.5 mg t.i.d. or 13 mg h.s., patient response may require dosage adjustment.
In elderly or debilitated patients, the initial daily dosage is 6.5 to 13 mg.
become pregnant they should communicate with their physician about the desirability of discontinuing the drug.
Usage during Lactation
AZENE™ (clorazepate monopotassium) should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.
PRECAUTIONS In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.
Periodic blood counts and liver function tests are advisable for patients on AZENE™ for prolonged periods. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.
In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually in accordance with the response of the patient, to preclude ataxia or excessive sedation.
ADVERSE REACTIONS The side effect most frequently reported with the use of benzodiazepines is drowsiness. Less commonly reported side effects are: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects include insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.
There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.
Decrease in systolic blood pressure has been observed with clorazepates.
DRUG INTERACTIONS If AZENE™ (clorazepate monopotassium) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that clorazepates prolong the sleeping time after hexobarbital or after ethyl alcohol, increase the inhibitory effects of chlorpromazine, but do not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.
If AZENE™ is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.
HOW SUPPLIED AZENE™ (clorazepate monopotassium) is supplied as 3.25 mg and 6.5 mg capsules in bottles of 100 and 500, and as 13 mg capsules in bottles of 100.
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The patient with clinically significant depression

He's getting better...



...and ELAVIL® is helping

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ELAVIL usually relieves a broad range of symptoms associated with depression, including sleep disturbance, one of the most frequently observed in the "constellation of symptoms," and usually the first symptom to respond to therapy.

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ELAVIL (Amitriptyline HCl, MSD) should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

For a brief summary of prescribing information, please see following page.

MSD
MERCK SHARP & DOHME

In clinically significant depression

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, 100 mg, and 150 mg
INJECTION: 10 mg per ml

Elavil®

(AMITRIPTYLINE HCl | MSD)

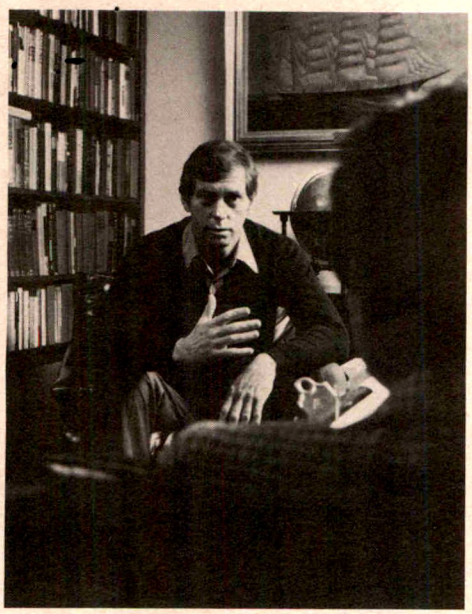
Single-entity antidepressant with an anxiety-reducing sedative component to its action

in clinically
significant depression

Elavil®

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Single-entity antidepressant with an anxiety-reducing sedative component to its action



Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

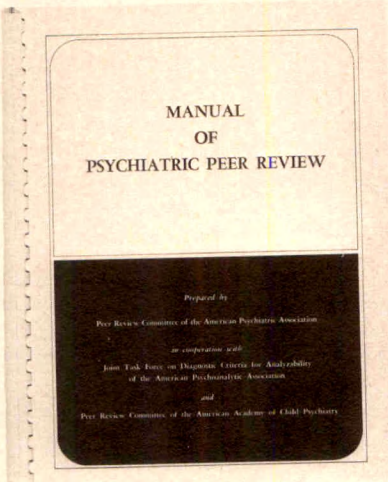
For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486 J7EL18(116)

MSD
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MANUAL OF PSYCHIATRIC PEER REVIEW

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"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

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ANTIPSYCHOTIC EFFICACY... INFREQUENT EXTRAPYRAMIDAL SYMPTOMS



Antipsychotic medication should not interfere with the patient's ability to participate in your total therapeutic program. That is why Mellaril (thioridazine) is an excellent choice. It is highly effective, and although extrapyramidal symptoms are characteristic of this class of drug, with Mellaril (thioridazine) extrapyramidal stimulation—notably pseudoparkinsonism—is infrequent. Adding an anti-parkinsonian agent—which can cause its own side effects—can usually be avoided.

Mellaril (thioridazine) is not habituating and usually does not cause euphoria or undue sedation. (But, warn patients about undertaking activities requiring complete mental alertness.) And Mellaril (thioridazine) is contraindicated in patients with severe hypotensive or hypertensive heart disease.

MELLARIL® (THIORIDAZINE)

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

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Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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The Editor Retires

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FRANCIS J. BRACELAND, M.D.

THE AMERICAN JOURNAL OF PSYCHIATRY

Thirteen Years Before the Masthead—The Editor Retires

WHEN FRANCIS BRACELAND was piped aboard as Editor of the *American Journal of Psychiatry* in July of 1965, the signs were inauspicious. He set sail on his maiden voyage with a crew who, although imaginative and enthusiastic, were entirely new. He carried as cargo a massive backlog of unpublished manuscripts that sank his hull dangerously below the Plimsoll mark. And he was heading into the stormy seas of the late 1960s that were to threaten with shipwreck the leadership of more than one venerable institution. The odds at Lloyd's for a safe return to home port were bleak indeed. They were reckoned, however, without a proper reading of the new skipper and his crew.

The details of the first hectic months of Dr. Braceland's editorship will probably never be fully known, for they are buried in the intangibles of a multitude of critical and sensitive decisions, of the skill and hard work of dedicated editorial staff members, and of the strong loyalties that rapidly developed among the Editor, his associates, and his permanent staff. The results were readily visible in the initial productions of the new regime, and even a cursory glance at the early issues of Volume 122 gives clear evidence of a fresh wind blowing through the editorial office. Rapid review and accelerated publication procedures enabled the *Journal* to carry papers that had been presented only a few months before at the May annual meeting. Articles dealing with a single topic from various points of view were brought together in "Special Sections," and informative reviews provided the reader with careful surveys of clinical and laboratory research in a variety of fields. New and exciting areas of psychiatry made their debut in the *Journal*'s pages—the therapeutic use of lithium in affective disorders, for example, was introduced to many American psychiatrists in the September 1965 issue. Even the Letters to the Editor section took on new life as authors were permitted to respond to their critics in a dialogue that often clarified and enlarged upon the original paper. In all of these innovations are to be found the beginnings of what have since become regular features that have helped to make the *Journal* the flagship of psychiatric publications.

What of the skipper who has piloted his vessel so unerringly true to her course during 13 busy but happy years? In the papers that immediately follow the reader is privileged to hear from two people who know Francis Braceland well—Dr. Howard Rome, his close friend and colleague, and Ms. Evelyn Myers, who, as Managing Editor of the *Journal* throughout Dr. Braceland's editorship, has played a major role in its success. We are grateful to them both for their descriptions of their rich and rewarding associations with him and for so vividly painting for us his erudition, his humor, his kindly warmth, and the integrity, sound judgment, and fairness of his editorial criticisms—characteristics that endear him to us all and make us grateful that, as Editor Emeritus, he will stay aboard as an active hand in the running of the *Journal*.

Tossed on the ceaseless flood of manuscripts that is an editor's lot, Dr. Braceland has never lost his way on the trackless sea of words that has been his home these past 13 years. Many decades ago Frederick Rolfe, that curious and troubled scourge of editors and publishers alike, proclaimed in the peculiarly pungent, frenetic, sometimes vitriolic tones that characterize his style, "I detest, and denounce as criminal that finite mortal man who (having found a groove which fits his atrophied brain and hypertrophied organs) screams to Kosmos, 'This is the way, the only way; and, if you won't mutilate yourself to fit it, then be damned to you, you're dotty, balmy on the crumpet, impossible, and you practise the Bulgarian Heresy!' And so on. 'But the minds of the prudent are flexible: for the language of mortals is voluble; and the discourses thereof are numerous and varied; and vast is the distribution of words here and there.' I hold, with the Primate-Poet, that there is room for all opinions, for all systems, each in its convenient sphere. . . ." (1).

There is a message here for our turbulent, often intolerant modern age—a message that is the star, perhaps above all others, by which Francis Braceland has steered his ship. *Deo volente*, the *Journal* will remain on course.

REFERENCE

1. Rolfe FW: Nicholas Crabbe. London, Chatto & Windus, 1958, pp 145–146

J.C.N.

Francis J. Braceland, the Man

BY HOWARD P. ROME, M.D.

WHEN ONE IS COMMISSIONED to do a portrait of a man, it is expected that he will render his subject realistically. In the style that is de rigueur in these days of investigative reporting, it certainly means not disguising the warts; indeed, all blemishes have to be exposed, and crow's-feet and various other wrinkles have to be noted as the telltale marks of being over the hill. But what does one say when the subject you are asked to portray has for years boldly displayed his wrinkles as the heraldic chevrons they are and furthermore celebrates in numerous writings his "advancing dotage" with scintillating wit and brilliantly humorous one-liners? Although the man in question pretends to be "naught but a hewer of wood and a drawer of water," his industry would be prodigious for a man half his years. And what is more, he actually makes what he does appear to be mundane, hence effortless.

Francis James Braceland, the man, will be an octogenarian in two years. I have to modify his claim that he is "in the metallic age," for, although there may be silver in his hair and gold in his teeth, there is *no* lead in his Sitz-platz. His long list of current activities refutes this public confession about plumbism being responsible for his slowing down. His raillery about his own aging process has become his stock-in-trade as a much sought after speaker on retirement and other aspects of gerontology. This artful dissembling is typical of his tolerant acceptance of life's vicissitudes over a span of 78 years.

EARLY YEARS

Frank's early years could have been the model of those true-grit-shall-prevail, Horatio Alger classics that were famous for exhorting and inspiring disadvantaged young men in the nineteenth century and early decades of this century. His father, John, died when Frank was four years old, leaving his widow, Margaret, to rear their three children. Frank and his younger brothers Joe and Paul learned at an early age to scrimp and scrape for everything. In the economic doldrums before World War I the going was tough.

"Brewerytown," where the Braceland family lived in Philadelphia, was a rough and tumble place in which status and hence respect were earned in the school of hard knocks by fight or flight; the young Frank Braceland learned how to do both. This was quite the opposite of the conducive milieu that Jacques Barzun has said is necessary for a child "to be at home in the House of Intellect," to which the Bracelands aspired.

Frank was 26 years old when, at last, he graduated from La Salle College in Philadelphia; he had to work his way through high school and college. Fifteen years later his alma mater awarded him the first of his twelve honorary degrees. He also has the distinction of being a ten-year member of La Salle's Board of Trustees.

Doctor Braceland graduated from Jefferson Medical College in the Depression year of 1930. In recognition of his literary contributions, Jefferson honored him with the Doctor of Letters degree 35 years later; as a distinguished alumnus, he has been a member of its Board of Trustees. He won a coveted internship and was chief resident in its hospital and after two years, because of a touching experience as a physician caring for a patient in the throes of a depression, decided on psychiatry in preference to other intriguing opportunities that had come his way.

Kirkbride's Hospital, as the 44th Street branch of the Pennsylvania Hospital was known to Philadelphians, had actually been founded by Benjamin Rush. It was a private mental hospital. In the days when Thomas Kirkbride was its superintendent, the 44th Street hospital was out in the country, and in the 1930s, when Frank began his residency, it was still not crowded. The impressive stone walls gave a sense of sanctuary reinforced by spacious, well-kept grounds. In the middle of a plot in front of its Georgian portico there stood a large magnolia tree. The date of its flowering each spring has been duly noted in Spencerian script in the manager's minutes recorded in huge journals. It was a beautiful old place. Frank Braceland fell in love with it and was willing to settle in for the rest of his professional life.

THE SEARCH FOR PHYSICAL AGENTS

His years of intimate association with Doctors Eyman, Bond, Strecker, Appel, Smith, and Palmer coincided with the seeding of modern psychiatry. The bloom that attracted interest was the spectacular discovery of Wagner-Jauregg, whose treatment of paresis was the first to really try to cure a devastating mental illness. It won him the Nobel Prize. Braceland, as a resident physician, quickly learned the knack of keeping a strain of malarial parasites viable by the inoculation of successive luetic patients with whole

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blood drawn at the height of the malarial fever. Naturally, this dramatic treatment lent credence to the search for equally successful physical agents for the treatment of the functional psychoses.

In his omnivorous reading, Frank had read of Cotton's enthusiastic report of treatment by surgical removal of the sites of focal infection but strenuously objected to much of the procedure. He said that making semicolons out of colons would not cure schizophrenia. However, there were always one or two patients on the wards of 44th Street who were being treated with a variant of Klaesi's *Dauerschlaf* (narcosis therapy).

Braceland, an avid and enterprising student of "The New Psychiatry," was enlisted in an enthusiastic search for yet another successful physical treatment of baffling disorders. His first publication, in 1934, was titled "Hematoporphyrin as Therapeutic Agent in Psychoses" (1). Doctors Strecker and Palmer were its senior authors. This study was of a mixed group of long-term chronic psychotic patients who were given both intramuscular and oral doses of hematoporphyrin. The rationale for the treatment was its reported success by European investigators.

A more explicit rationale for the use of these exotic agents appeared as a prefatory paragraph in a 1935 paper titled "Aschner Treatment of Schizophrenia: Therapeutic Note" (2); this was published with Doctors Appel and Farr as the senior authors. Its justification begins,

> For the last 20 years, there has been a revival of interest in the constitutional aspect of disease, first in Germany, more recently in the United States. The leaders of this movement have aimed to integrate the scientific advances of the last 75 years in genetics, morphology, physiology and allied branches.

The conclusions were that "results are . . . disappointing. . . . In the majority of cases [any results were] *only* temporary."

As a bright and enthusiastic young resident, Frank eagerly sought opportunities to interlard his daily routine of psychotherapeutic sessions with an assigned group of patients and the usual administrative chores that are the lot of all tyro-psychiatrists. The congenial atmosphere of the Pennsylvania Hospital encouraged this. Then, too, the time was conducive. Psychiatry was on the move, galvanized by two vastly different currents—psychoanalysis and its variants and, at the opposite pole, what was coming to be biologically oriented research. Our man, F.J.B., was caught in the intellectual turbulence.

By happy coincidence he finished his formal training in time to be chosen as one of several special traveling Rockefeller Fellows. His turmoil in choosing between Zurich and Vienna was resolved by Dr. Earl Bond's advice. He applied for and was accepted as an assistant physician at the Anstalt Burgholzli, Zurich.

While in Switzerland, he met with members of the Jungian school of analytical psychology . . . acquire the patina of a cosmopolite that . . . riched progressively ever since. He . . . his European experience—especially . . . Thomas Mann—as a unique opportuni. . . . his eyes to the world outside of Philade . . . Pennsylvania Hospital. It was rounded o . . . ly rich and stimulating experience as a . . . Dr. Kinnier Wilson's service at the Nat . . . Queens Square, London. While there, . . . draughts of the best of British medicine . . . nonce being unconcerned about the . . . ceived from Dr. Earl Bond an invitatio . . . the Pennsylvania Hospital as Clinical D . . .

THE PHILADELPHIA YEARS

Equipped as he was with the panache . . . fellowship at age 36, Frank's candidacy . . . ademia was complete. His mentors, D . . . Strecker, Appel, and Palmer, saw to tha . . . his place was reserved, first as an appren . . . of a number of "footstools" in the grove . . . en route to what was destined to be a cer . . . of professorial chairs. In short order he . . . an Instructor in Psychiatry at the Unive . . . sylvania Medical School, an Assistant P . . . graduate school and an Associate Pro . . . Women's Medical College, a Visiting Le . . . normal Psychology at Swarthmore Colle . . . Lecturer in Abnormal Psychology at . . . lege, and a psychiatrist on the staffs of s . . . phia hospitals.

The next few years were equally head . . . met, wooed, and married a Main Li . . . nurse's uniform—Hope Jenkins; he beca . . . Diplomate in Psychiatry; he was an act . . . Naval Reserve Medical Group; and he . . . join the private practice at the recent y . . . of the Pennsylvania Hospital and em . . . was planned as a progressive elevation . . . Philadelphia psychiatry. His place in . . . was further assured by his election to the . . . delphia College of Physicians. He rece . . . colade when he became chief of a te . . . the prestigious Philadelphia General . . . Blockley).

As Thomas Wolfe ruefully noted, "... home again." Philadelphia was not for . . . was not to be the life that would stay cert . . . niche no matter what the rewards. He . . . be Dean of the School of Medicine and . . . Psychiatry at Loyola University in Chica . . . was tempting; he was needed, and he fo . . . lenge irresistible.

At a superficial level, he rationalized h . . . Institute by recognizing that the Inst . . . breeding ground for the exportation of its . . . chiatry to the hinterlands. He would be . . .

Philadelphia's emissaries. But Hope and Frank, as well as their many friends, found the departure from Philadelphia a wrenching experience. Their leaving tore deep affective roots. They found a measure of solace in the belief that, after all, Chicago was just a bit beyond West Philadelphia's 69th Street!

Chicago—its medical and university communities—welcomed them with open arms. Its "organized" medicine in the years before World War II was in fact a loosely knit enclave of independent fiefdoms. Coming from Philadelphia with its own boundary problems, Frank found this state of affairs quite familiar. Before he became a dean, he had been and still was a clinical psychiatrist, accustomed to dealing with resistance in all shapes, forms, and guises. This expertise stood him in good stead. In the relatively brief time he remained in Chicago, he succeeded in leaving his unique mark. But his long-term plans for Loyola had to give way to a higher priority—World War II.

ACTIVE DUTY IN THE NAVY

As a Naval Reserve officer as well as a medical school dean, Frank Braceland was a natural to recruit physicians for the Medical Corps of the U.S. Navy. Lt. Commander Braceland MC, USNR, was assigned the duties of Special Assistant to the Surgeon General, U.S. Navy, in 1942. His first job was to solicit physicians to volunteer for active duty. Ultimately, the assignment was formalized when he was ordered to report to the Personnel Office of BuMed; this required a move to Washington, D.C. (His next formal appearance in Chicago was in the 1960s to receive an honorary degree from Northwestern University. Later, Loyola University, Stritch School of Medicine, awarded him the Stritch Medal.

Frank Braceland in Washington was beset by ambivalence. He was frustrated by being kept chairborne, figuratively chained to a desk during years of active duty in the Bureau of Medicine and Surgery. On the other hand, when he became Chief of the Neuropsychiatry Branch he was finally in his element. In retrospect, he sees this assignment as King Henry V saw Agincourt on Saint Crispin's day: "He which hath no stomach to this fight, / Let him depart; his passport shall be made, / And crowns for convoy put into his purse: / We would not die in that man's company / That fears his fellowship to die with us." Frank Braceland, with Bill Menninger in the Army, Bob Felix in the Public Health Service, Don Hastings in the Air Force, Dan Blain in the Coast Guard-Merchant Marine, and their associates ("shall be remembered,/ We few, we happy few, we band of brothers") were responsible for the organization of the nation's psychiatric services during World War II.

Frank's special contribution to the naval war effort was the unique accomplishment of humanizing a military organization that prided itself on its tradition of iron men and wooden ships. Frank's approach to the old salts of the line—Navy admirals and Marine generals—was pragmatic. If they required, as their motto had it, to keep as many men at as many guns for as many days as possible, they would have to concede a little expertise to the Neuropsychiatry Branch of BuMed. Mirabile dictu! They did, and Frank Braceland was authorized to institute what amounted to far-reaching reforms in the selection of personnel in the Naval Training Stations as well as in the care, treatment, and discharge from hospitals of the Navy's psychiatric casualties. His Legion of Merit and promotion to Rear Admiral are testament to the acknowledgment by superiors of his meritorious contributions.

THE MAYO CLINIC

As trying as those years were, they actually served as a second apprenticeship at age 46 for what was to come following Frank's discharge from active duty. Not for long was he without an equally challenging billet. The Mayo Clinic had for years eschewed the creation of a Section of Psychiatry. The reason was said to be that the Doctors Mayo were as mindful of the limited facilities available in Rochester, Minn., in the prewar years as they were conscious of the magnitude of the psychiatric problems in the population their clinic served. Their successors were also aware of the changes that were taking place in the delivery of medical services.

Frances Braceland, M.D., now a civilian, was invited to join the staff of the Mayo Clinic as a member of the new Section of Neurology and Psychiatry. The initial affiliation with neurology was intended to make use of the established reputation of the distinguished and respected neurologists as the thin edge of the wedge to accommodate an Auslander.

In short order, the innovations for which F.J.B. is noted began to appear. He convinced the Sisters of St. Francis, who operate St. Marys Hospital, that as a general hospital purporting to render comprehensive services it needed a defined and separate psychiatric unit. He collaborated with the Section of Pediatrics in the establishment of the Rochester Child Health Project, for which he recruited Adelaide Johnson and Ben Spock, the latter recently released from active duty as a Navy psychiatrist. Frank established a liaison-consultation service in both the clinic and its affiliated hospitals. He persuaded the Mayo Graduate School to assign fellows (residents) in internal medicine, neurology, and neurosurgery to the Section of Psychiatry for a three-month full-time stint. In addition, he carried a full schedule of clinical responsibilities.

As an extracurricular activity, he extended his support to both the local and state communities and was involved in the exposé of the Minnesota state hospital problems. In undertaking these community actions, he launched another long and illustrious career as a mem-

ber, officer, national and international consultant, and adviser to myriad professional and governmental organizations—at last count, the number he has served over the past 38 years was 184.

THE INSTITUTE OF LIVING

Frank Braceland's appointment in April 1951 as Psychiatrist-in-Chief of the Institute of Living, Hartford, Conn., was noted, in proper Navy parlance, as "A New Helmsman Comes Aboard" in a chapter of a book celebrating the sesquicentennial anniversary in May 1972 of the founding of the Institute (3). The death of his predecessor, Dr. C.G. Burlingame, 12 months previously had left Eli Todd's Hartford Retreat adrift without a rudder. After a few stormy months the patients, the staff, the City of Hartford, and the State of Connecticut became aware that the Institute was again on the course set by Dr. Todd on May 22, 1822; a man of equal national stature, far-reaching vision, and a gentle but firm hand was now in charge. Although Frank stepped down as Psychiatrist-in-Chief in 1965, he continues on as Senior Consultant, maintaining the indefatigable pace that had helped make the Institute the exemplar of a comprehensive mental hospital. Emerson's observation that "an institution is the lengthened shadow of one man" is realized in the lasting foundation on which others have built and limns Braceland and his vision of the place of psychiatry in and of the community.

"A MAN OF PARTS"

Last year was a Braceland typical. In addition to his biweekly trips to Washington, "the mendicant" (as he calls himself) continued an enormously successful money-raising campaign in behalf of the Institute of Living's rebuilding program. He was requested to write nine papers during the year. He winnowed numerous invitations and finally consented to present 15 formal lectures; among the institutions were seven colleges here and in Canada. In what he extravagantly calls his "spare time," he reviewed books for no less than 23 journals that ran the gamut from the *New England Journal of Medicine* and *Science* to the *Psychoanalytic Quarterly* and *Theological Studies*. Verily, the man Braceland is a man of parts.

Frank is an inveterate idealist. Every place he has ever worked is the best of all places. He is nostalgic about each of them and talks longingly about his earnest desire to return—but he never has. Instead, he goes on to other challenges, leaving in his wake lasting ties of friendship and sincere regrets, both his and theirs. Each of the places he has left is much the better for his having "tarried awhile in their midst," as he would put it. Each of these places became his home. A close friend once remarked that Frank Braceland's alter ego must be that mythical Nigerian go[at] ue has his feet point forward while his he[ad] look back to where he has been.

In the years I have known him, o[nly] seen him come close to losing his compos[ure] during his tenure as Secretary of the A[m] of Psychiatry and Neurology. The holde[r] is pestered by trivia and inundated [with] Frank and his long-time friend David Boy[d] job to that of a cross between a servant a[nd] boy (4). On the day Frank came close [to] cool, he was at his desk in the Board o[ffice] scientious secretary, Clarice Johnso[n] screened the numerous phone calls to a[llow] to do what only the boss could do. A fre[e] sistent caller was giving her a particula[r] demanding a positive answer to a series [of] she had said repeatedly were not with[in] tary's power to answer. In desperation, [she] Frank and asked, "What shall I tell him [to] pursed his lips, paused (I suppose to [collect] and then replied, "Tell him to walk cast [and] floats."

Frank and David Boyd wrote a delig[htful] the trials and tribulations of being Se[cre]tary Board (4). I am sure of Frank's hand in [choosing] its Latin subtitle: "Apologia Pro Vita Su[a]" only to the redoubtable Robert Burton, [the] author of medical treatises who so de[lighted] quote Latin—phrases, aphorisms, vacet[iae].

Frank's close friends are privileged to [share] pose. He would describe himself as "[a] gentleman who loves to walk beaches." [Let] him regale congenial listeners by the ho[ur] shady, uproarious shaggy-dog stories [or] deliberately, his special brand of Nav[y] "with-a-twist-with rocks-if-you-please." days (and on occasion now) he would, i[n] into a soft-shoe routine à la Ray Bolge[r] ly older ladies who "knew him when." choice as a dancing partner for a fast [one] when he can convince the orchestra to [play] for Two."

Not the least of his talents, one for w[hich] membered with great affection as "Un[cle] incomparable prestidigitator," is his [ex]pertise is able to leave young children slack-j[aw] ment when, with a flourish of an empty h[and] to extract a coin from their ears or pock[ets]. And what is more, make it move over [their] fingers "by magic." Frank mimes W.C. [Fields] fection in pretending to be all thumbs w[hile] with one of his young grandchildren in [the] diapering. And yet, if asked, he proudly [has] latest snapshots of every one of them fr[om] pocket wallet.

The real key to Frank Braceland's pers[onal] ing optimism is his passionate religious c[onvic]tion. is devout. His wife, Hope, who is equa[lly] tously captured the essence of the man [in]

this way: "His early rising, his respects to and chats with his Maker betray his acceptance of what is, and his trust and faith in what can be, and what can be done 'with a little bit of luck' and with a little bit of help."

George Raines, an old Navy shipmate, feigning incredulity, used to say: "You can't ever follow the Dean through a revolving door for he is sure to come out after you. He's just got to be last!" Frank said it indirectly through the words of the poet Terence: "Humani nil a me alienum puto." Significantly, this was the last sentence of his Presidential Address to the 113th annual meeting of the Association in Chicago, Ill., in May 1957.

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Francis J. Braceland, the Editor—The Music Will Play On

BY EVELYN S. MYERS, M.A.

IN APRIL 1977 Dr. Francis J. Braceland wrote Dr. Robert W. Gibson, then APA President, requesting that a search committee be appointed to find a new Editor of the *American Journal of Psychiatry*. In his letter to Dr. Gibson, Dr. Braceland observed that the *Journal* was going along very well. "Why, then, am I leaving?" he asked. "Only one reason: I shall be nearly 78 by the time my replacement arrives. . . . Samuel Johnson said, 'At 77 it is time for a man to be in earnest,' and besides, there are a number of things I still want to do. . . . Two thoughts play around in my mind. The first is that 'it is hard to go home while the music is still playing.' But this is cancelled out by a line from the bard: 'Reluctant lags the veteran on the stage.' "

The many letters received after Dr. Braceland's decision was reported to the Board of Trustees and in *Psychiatric News* reflect in some measure how he is regarded inside and outside of psychiatry. Some of the letters touch on traits and characteristics that have made him an outstanding Editor. From the vantage of an association with Dr. Braceland that spans the entire 13 years of his editorship, I would like to quote and comment on extracts from a few of those letters.

A former member of the *Journal*'s Editorial Board wrote

> In a very real sense your decision marks the end of an era, probably one of the most dramatic chapters in the history of our culture. . . . You are fortunate in being able to age gracefully and to leave the arena upright and with dignity. None of us could hope for more.

The strong sense of history implied in these sentences has been a characteristic of the Braceland editorship. We on the staff who met with him each time he came to Washington on his biweekly trips from Hartford became keenly aware of this as, meeting with us, he mused on how to solve a knotty editorial problem. Often he would reach back to the days when he was a resident at the Pennsylvania Hospital, when some of the somatic treatments were being applied to psychiatric patients for the first time; or to the days when as a Rockefeller Fellow he was learning from Jung at the Bürgholzli in Switzerland; or when, during World War II, he worked hard to educate the Navy "brass" about the need for a humane approach to young sailors with psychiatric problems. Dr. Braceland's ultimate decision about whether to accept or reject a manuscript was based on criteria that met the needs of the 1970s—Is this new and important? His decision was always tempered with knowledge of what had gone on before.

Another side of Dr. Braceland's personality came through in a letter from the editor of a literary magazine:

> Few privileges in this world are more welcome (to me) than the discovery of a well-turned phrase, a philosophy of medicine, your approach, and a sense of humor.

His sense of humor is of course one for which Dr. Braceland is famous. One can appreciate this trait when one is in his presence at a table of guests at dinners where he has been the honoree or after-dinner speaker can attest. We staff members have been treated to a goodly measure of his famous-world-figure jokes, and an assortment of stories that defy categorizaton. The readers of the *Journal* have not had the same opportunity to enjoy his occasional response to a letter to the Editor that called for a well-turned phrase. For example, a letter in the February 1971 issue took Dr. Braceland to task for his "In his plea for shorter papers for our *Journal*, F.J.B. wrote, 'But psychiatrists who want to publish in our journal will simply have to condense their productions and write more succinctly.' Couldn't F.J.B. have given us a more succinct example, such as, 'But psychiatrists who want to publish in our journal will have to write more briefly.' F.J.B.'s response was pure Braceland: 'Our Editor, already dehydrated, hence forced to succincts and discard his briefs.' "

In a quite different vein, Dr. Braceland was especially pleased to receive a letter from a psychiatrist who, shortly after Dr. Braceland took office, had objected to the way his manuscript had been handled. Looking back on his attitude at the time and expressing his regret, the psychiatrist wrote that he "admired, very much, the excellent job you have done as editor, and I don't have to tell you how difficult your job was." The details of what had happened with the manuscript are unimportant—what is important—and what I think the psychiatrist was pleased to say in his letter—was a recognition that his manuscript had been given a fair hearing in relation to its merit.

This quality—fairness—has been a hallmark of Dr. Braceland's editorship that I

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most. In evaluating manuscripts he has bent over backward to ensure that a decision was based not on bias or on his own special interests but on informed judgment. This sometimes meant asking another member of the Editorial Board to share in the final decision if a paper under consideration reflected a point of view with which Dr. Braceland disagreed. It sometimes meant reconsidering his decision to reject a manuscript when the author presented cogent arguments at variance with the comments of the experts who reviewed the paper. He also tried—even when he found it necessary to reject their papers—to encourage neophyte authors, often sending them lengthy suggestions so that they would have a better chance the next time.

KEEPING UP WITH THE TIMES

A professor and chairman of a department of psychiatry wrote Dr. Braceland:

> You have left a significant mark on the *American Journal of Psychiatry,* which has grown, during your tenure, to a genuinely stimulating publication that compares favorably with any superior journal in our own or any other specialty. Some of the heightened interest, of course, is a reflection of the exciting new research that has recently shed new light on old psychiatric syndromes. The changes in style, format, and organization and the new features which you have introduced, however, are the factors that make this journal the one which I await each month with eager anticipation.

What comes through here is a reflection of Dr. Braceland's willingness to keep up with the times. Basically a conservative person (one of his favorite expressions is, "Go not from the troubles you know to the troubles you know not of"), he has been willing to accept change when he was convinced that change was warranted. This applied to format, type style, size of the *Journal,* and the introduction of such features as special sections and topical groups of papers devoted to a subject of importance, overview and other special lead articles, and, more recently, our new Clinical and Research Reports section. He has wanted the *Journal* to be as good as possible, and he has given thoughtful consideration to all suggestions for change, whether they came from an author, a member of the staff, an Association member he met on one of his frequent trips around the country, or anyone else who had something to say. The *Journal* has never been far from his thoughts. Reading manuscripts on planes, at home in the evenings and on most weekends, and during his so-called "vacations"—this has been his way of life for the past 13 years. I have never known a person who has been more dedicated to his job than Dr. Braceland.

The final quote, which refers back to Dr. Braceland's letter to Dr. Gibson, needs no comment from me. From a current member of our Editorial Board, it says it all:

> You are more than entitled to do the reading and other things you want to do without worrying that thousands of psychiatrists will be mad at you for not continuing to take care of us! But you have spent a marvelous lifetime preparing us to negotiate our selfishness. By all means go home while the music is playing—it will play forever for you.

These letters are a testament to some of Dr. Braceland's better-known qualities. I would also like to touch on one or two other qualities that have particularly impressed me.

The first is pride—pride in being a physician and especially a psychiatrist. This is a pride associated not with arrogance but with the possibilities of service to one's fellow man. Dr. Braceland has been pleased in recent years—and this has permeated his writings—that psychiatry has moved closer to the rest of medicine. He sees psychiatry's principal mission as taking care of the mentally ill. The "medical model" means different things to different people; to Dr. Braceland, if my perception is correct, it means the well-trained, compassionate physician putting the needs of the patient before all else. In selecting material for the *Journal* he has kept uppermost in his mind: Will this help psychiatrists with their patients?

His pride has a wry twist. He is understandably proud of the honors his beloved profession has bestowed on him—head of the Navy's Neuropsychiatric Branch during World War II, Secretary of the American Board of Psychiatry and Neurology, President of the American Psychiatric Association, and, most recently, Editor of APA's official journal. But there is another side. "I'm always afraid when I'm introduced to a psychiatrist for the first time," he has been known to say, "that I may have sent him as a young medical officer to Attu, or signed the letter telling him he failed his Boards, or rejected one of his brainchildren."

"Rejecting brainchildren" brings me to another of Dr. Braceland's qualities as Editor. The *Journal* can accept fewer than a quarter of the articles submitted to it. For Dr. Braceland, some decisions to reject—when the paper was clearly below standard—were not difficult. But others brought agony. His concern was not so much the fear of losing a good article (rejected authors sometimes sent him a reprint of their article when it was published elsewhere and this pleased him) as concern over the author's wounded feelings. Many authors received special letters from Dr. Braceland rather than the form letters a large journal is often obliged to use. These letters reflected the Editor's character—his charm, wit, and compassion. They also transmitted his genuine regret, and a number of authors have written him to say, "That was the nicest rejection I ever received."

Finally, a more personal note. When Dr. Braceland and I began working on the *Journal* 13 years ago, neither of us knew quite what to expect of the other. The relationship—and I think this is true with most important relationships—took a while to develop to a point

where we were both comfortable with it. Part of the reason for its success was that we both were committed to do the best job possible, and each of us respected that commitment in the other. But also of great importance was Dr. Braceland's extraordinary (uncanny, almost) sensitivity to the feelings of those within his orbit. Within two minutes of his walking into the *Journal* office on his visits from Hartford he could tell how things were going with us on the editorial staff, and if there was a problem he offered his assistance in solving it. The other members of the staff did not only have respected Dr. Braceland as a distinguished Editor but have come to love him as a kind and sensitive human being.

One could search *Bartlett* and discover a quotation that might capture some of Dr. Braceland's special qualities. I prefer a verse from Proverbs:

> Wisdom is the principal thing, therefore get wisdom.
> Yea, with all thy getting, get understanding.

Discriminating Features of Borderline Patients

BY JOHN G. GUNDERSON, M.D., AND JONATHAN E. KOLB, M.D.

Borderline patients were compared with schizophrenic patients, neurotic depressed patients, and a group of patients with differing diagnoses. The purpose of this comparison was to find out whether borderline patients could be discriminated from other psychopathological groups and whether a discrete list of recognizable characteristics discriminating borderline patients could be isolated. According to the results of the comparison, borderline patients can be discriminated with high accuracy from matched comparison groups with whom diagnostic confusion is common. Seven criteria provided a clinically sensible and practical means of approaching the diagnosis of borderline disorder.

THE FIRST STEP in defining a clinical syndrome is to demonstrate that it is a recognizable entity that can be discriminated from other disorders. This is particularly relevant to borderline disorder because of its presumed position between psychosis and neurosis and the arguments that persist as to whether it is really a subtype of schizophrenia or a variant of affective disorder. In a previous report (1) the descriptive literature on borderline patients was reviewed and those characteristics of borderline patients about which there is consensus were summarized. We have also demonstrated which of those consensus characteristics were actually found and which were not found in a systematic study (2). In this report we ask two new questions. First, can borderline patients be discriminated from other psychopathological groups with whom diagnostic confusion often exists? The failure to do so would cast doubt on the rationale for continuing use of borderline disorder as a separate diagnostic entity in our clinical parlance. A positive result could lend support to the inclusion of borderline disorder in our standard nomenclature. The second question addressed in this study is, Can a discrete list of readily recognizable characteristics that are highly discriminating be isolated? Such a list could be useful in selecting a sample of borderline patients in a replicable way.

Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

Dr. Gunderson is Assistant Professor, Department of Psychiatry, and Dr. Kolb is Instructor in Psychiatry, Harvard Medical School, Boston, Mass. Dr. Gunderson and Dr. Kolb are also Assistant Psychiatrists, McLean Hospital, 115 Mill St., Belmont, Mass. 02138.

The authors wish to acknowledge the statistical assistance of Murray Dalziel.

METHOD

All of the patients included in this study were given clinical diagnoses by the admitting physician at McLean Hospital. All of the patients were hospitalized, were between the ages of 16 and 35, and had no evidence of organicity. Only patients given an initial diagnosis that was considered certain for schizophrenia, neurotic depression, or borderline personality were included. Patients given a primary diagnosis of alcoholism or drug habituation, even if they were considered to have a borderline personality organization, were not included. Each sample was further screened using independent research criteria and/or clinical diagnoses to prevent overlapping diagnoses (3). The samples were matched for age, sex, race, and marital status (2) (see table 1). The patients were generally young, white, middle-class people from the Boston area; approximately two-thirds of them were women.

After giving informed consent, the 64 patients were interviewed within one week of admission using the Diagnostic Interview for Borderlines (DIBs). The DIBs consists of 123 inquiries ("items"), from which 29 of the characteristics of borderline patients found in the literature (each presented as a "statement") are scored. These statements are distributed in the 5 areas ("sections") of the interview as follows: 4 for social adaptation, 5 for impulse/action patterns, 5 for affects, 8 for psychosis, and 7 for interpersonal relations. The statements are added and converted into a scaled score for each of these 5 sections. Reliability and scoring characteristics of this instrument have been reported elsewhere (4).

A stepwise discriminant function analysis was used to compare the borderline sample (N=31) with three different comparison groups.[1] The first comparison group was composed of 22 schizophrenic patients. The second comparison group was composed of 11 patients with neurotic depression. The third comparison group, called "all others," consisted of a combination of the schizophrenic and neurotic depressed patients plus a

[1]This method uses Rao's V to indicate the separation between groups. The first variable entered is one that maximizes V. This proceeds sequentially unless the variable correlates highly with all other variables in the function. In that case the V will decline and that variable is excluded.

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TABLE 1
Demographic Variables and Diagnoses of 64 Inpatients*

Variable	Diagnosis		
	Borderline Disorder (N=31)	Schizophrenia (N=22)	Neurosis (N=11)
Mean age	23.7	23.7	25.5
Sex			
Men	9	8	4
Women	22	14	7
Marital status			
Ever married	11	4	2
Never married	20	18	9

*All of the patients were white except 1 black patient, who was in the borderline group.

mixed group of 9 patients with other diagnoses. For each of these three comparisons discriminant functions were done on two sets of variables. The first discriminant function was done on the 29 summarizing statements. The second analysis was a discriminant function using the scores from each of the 5 sections of the interview. One further discriminant function analysis was done on the comparison between the borderline patients and all others. This function was done on 10 items selected because we have found them especially helpful in our clinical experience. Other reasons for selecting these items were that they reflected independent areas of function and were among the most significantly discriminating according to t tests. The best overall discriminators were then synthesized on the basis of clinical and conceptual overlap into a discrete list of clinically sensible discriminating characteristics.

RESULTS

The borderline patients were first compared with the schizophrenic patients, then with the neurotic depressed patients, and then with the group of patients with differing diagnoses.

Borderline Versus Schizophrenic Patients

Together, the 8 statement variables listed in table 2 were able to discriminate the borderline sample from the sample of schizophrenic patients with 100% accuracy. The most useful discriminator was flat affect. It was unusual to find flatness in our borderline sample, but it was common in the schizophrenic patients. This difference was more obvious in terms of affect within the interview than as it was reported in the patients' recent histories. As found previously (5, 6), although schizophrenic patients actually evidenced considerable depression and reported recent histories of other affects, unlike borderline patients they were often flat at the time of the interview.

The variable of devaluation/manipulation reflects qualities of the close interpersonal relationships of borderline patients that are less evident in schizophrenic patients. In fact, as the "loner" variable in-

TABLE 2
Standardized Coefficients in Comparison of Borderline Sc zo-phrenic Patients and DIBs Statement and Section Sc

Variable	Standard
Statement description	
Flat/elated**	
Devaluation/manipulation	
"Loner"**	
Derealization***	
Drug abuse	
Psychotic experiences on drugs	
Aggressiveness	
Active social life	
Section description	
Interpersonal relations	
Impulse/action patterns	
Social adaptation	
Affects	

*For the DIBs statements, Wilks' Lambda was .200 the the borderline patients was .776, and the group cent phrenic patients was −1.012. For the DIBs sectio .418, the group centroid for the borderline patients wa centroid for the schizophrenic patients was −.863
**A low score on this variable was expected to cha tients.
***This variable was higher in the comparison group (direction).

dicates, the schizophrenic patients were be socially isolated and to lack the ships of the borderline patients. The zation was more frequent in the schizop than in the borderline patients was also vious study using other samples (5). Be siveness of drug abuse and the fact th borderline patients had psychotic exp while on drugs were also useful discr final two statement variables reflect the of social aggressiveness and more act volving groups of people found in the tients than existed for the schizophren

Although all 5 areas of assessment we in locating the most discriminating grou the psychosis section did not provide u information when the section total sc lyzed (see table 2). These 4 variables w criminate the two groups of patients wit racy. The most useful section totals personal relations and impulse/action p which reflect more enduring behavio analysis suggests that the least useful discriminating borderline from schizop can be expected to come from the s g information.

Borderline Versus Neurotic Depressed

The 14 scored statements listed in t to discriminate the borderline from t pressed sample with 95.45% accuracy. parison with the schizophrenic pati abuse and the occurrence of psychot while using drugs were helpful discrim ly discriminator from the affect evalu

TABLE 3
Standardized Coefficients in Comparison of Borderline Versus Neurotic Depressed Patients and DIBs Statement and Section Scores*

Variable	Standardized Coefficient
Statement description	
Drug abuse	.411
Dysphoria/anhedonia	.307
School/work achievement**	−.541
Devaluation/manipulation	.353
Paranoia	.233
Past therapy regressions	.443
Antisocial	.297
Psychotic experiences on drugs	.281
Difficulty being alone	.222
Instability	.357
Hallucinations/delusions***	.281
Mania, delusional***	.195
Past therapy relationships	.211
Sexual deviance	.228
Section description	
Impulse/action patterns	.590
Interpersonal relations	.621
Psychosis	.236

*For the DIBs statements, Wilks' Lambda was .167, the group centroid for the borderline patients was .546, and the group centroid for the neurotic depressed patients was −1.489. For the DIBs sections, Wilks' Lambda was .530, the group centroid for the borderline patients was .410, and the group centroid for the neurotic depressed patients was −1.119.
**This variable was higher in the comparison group (i.e., not in the expected direction).
***A low score on this variable was expected to characterize borderline patients.

TABLE 4
Standardized Coefficients in Comparison of Borderline Patients Versus All Others and DIBs Statement and Section Scores*

Variable	Standardized Coefficient
Statement Description	
Devaluation/manipulation	.163
Drug abuse	.460
Flat/elated**	.176
"Loner"**	.214
Paranoid	.283
Manic, delusional**	.380
School/work achievement	.185
Hallucinations/delusions**	.253
Derealization***	−.257
Sexual deviance	.324
Dependency/masochism	.219
Past therapy regressions	.222
Manipulative suicide	.222
Angry, hot-tempered	.155
Section description	
Impulse/action patterns	.485
Interpersonal relations	.468
Psychosis	.194
Affects	.175
Social adaptation	.154

*For the DIBs statements, Wilks' Lambda was .309, the group centroid for the borderline patients was .965, and the group centroid for all of the other patients was −.706. For the DIBs sections, Wilks' Lambda was .537, the group centroid for the borderline patients was .790, and the group centroid for all of the other patients was −.578.
**A low score on this variable was expected to characterize borderline patients.
***This variable was higher in the comparison group (i.e., not in the expected direction).

borderline patients reported more frequent and sustained dysphoria and anhedonia. Borderline patients were more frequently found to have a sexually deviant pattern (usually promiscuity) or antisocial pattern, and they had lower school or work achievement records. Borderline patients were more intolerant of being alone, but their interpersonal relations were more unstable and marked by more devaluation and manipulation. Such problems were often evident in their previous treatment experiences, which indicated countertransference problems, staff splitting, and frequent reports of "getting worse." A final major area of difference is that the borderline patients were more likely to report brief paranoid experiences and occasionally had experiences that resembled hallucinations but proved difficult to evaluate. The neurotic depressed patients were more likely to have had possible manic episodes.

With respect to the section scores (see table 3), once again more enduring patterns (impulse/action patterns and interpersonal relations) proved more valuable than signs and symptoms. The section scores discriminated the two groups of patients with 84.09% accuracy. Neither social adaptation nor affects added much when the other three sections were taken into consideration.

Borderline Patients Versus All Others

The comparison between borderline patients and all of the other patients sharpened the focus on those characteristics of borderline patients which are the best overall discriminators for use in a variety of differential diagnostic problems. The 14 DIBs statements shown in table 4 were able to discriminate the borderline patients from all other patient groups with 86.84% accuracy.[2] Because of clinical and conceptual overlaps within the 14 variables they can be condensed into the following 7 criteria:

1. *Low achievement.* The typical level of the school and work achievement in the two years before hospitalization was quite low in borderline patients. This was so despite apparent talent or ability to do better and was quite similar to the achievement levels attained by schizophrenic patients.
2. *Impulsivity.* The principal form this took was a pattern of serious alcohol or drug abuse. Sexual deviance was highly related to impulsivity because it consisted largely of the promiscuous behavior of borderline patients under the influence of alcohol or other drugs.
3. *Manipulative suicide.* This refers to those suicide efforts or gestures which seemed designed to exact a "saving" response from a significant other. This commonly occurred as wrist slashing or overdosing and was the most frequent precipitant for hospitalization.
4. *Heightened affectivity.* Borderline patients dis-

[2]Application of this function to an additional independent sample of 29 clinically diagnosed borderline patients accurately identified 76%. In another analysis, 81% of 27 patients who were classified by this function as having borderline disorder had been given this clinical diagnosis.

played multiple intense affects. Anger may be the most discriminating of these. Even more important in discriminating borderline patients from others was the absence of flat affect and the relative absence of satisfied feelings.

5. Mild psychotic experiences. These most commonly took the form of drug-free paranoid ideation, derealization experiences, and a history of regressions or getting worse in previous treatment. Occurrence of any of these in the absence of severe widespread psychotic symptoms of any type in the patient's past life was a strong indicator for the borderline diagnosis.

6. High socialization. Borderline patients were definitely not socially isolated ("loners") and were, in fact, intolerant of being alone, i.e., compulsively social.

7. Disturbed close relationships. Devaluation, manipulation, dependency, and masochism characterized and caused instability in the intense attachments of borderline patients. Devaluation refers to the tendency to discredit or undermine the strengths and personal significance of important others. Manipulation is used here to describe those efforts by which covert means were used to control or gain support from significant others. Some typical ways included somatic complaints, provocative actions, or misleading messages. Masochism refers to the pattern in which borderline patients repeatedly, knowingly, and avoidably found themselves hurt in their close relationships (i.e., they saw themselves as victimized). The dependency issues of borderline patients were frequently manifest in receiving actual caregiving or directions from their important other.

A discriminant function by section scores showed that for this comparison all 5 areas provided valuable discriminatory information (see table 4). The section scores discriminated the patient groups with 81.58% accuracy. As in the previous comparisons, sign and symptom areas (psychosis and affects) were less helpful than the impulse/action patterns and interpersonal relations.

The third function on this comparison was derived from 10 highly specific items that had shown differences on t tests. The 7 items included in the function are shown in table 5 in the form in which they are asked on the DIBs. This function discriminated the borderline patients from all others with 85.35% accuracy. This is very similar to the function that was derived from statements. Sadism reflected a habitual pattern of being hurtful in relationships despite conscious knowledge of the effect. Two other items had to do with the fact that the delusions reported by borderline patients were circumscribed and ego alien (patients felt disturbed about them, i.e., they were experienced as symptoms). Loneliness and emptiness were very frequent complaints and quite specific to the borderline patients. Another useful discriminator was the frequency with which borderline patients reported having formed special relationships with previous therapists or others in caregiving positions. Other items that resembled aspects of the previous functic history of self-mutilation and drug depe

TABLE 5
Standardized Coefficients in Comparison of Borderline All Others and DIBs Items*

Item Description	
Do your significant others complain that you mean (e.g., teasing, beating, withholding)?	
Have you ever hurt yourself deliberately—othe than suicide?	
(After making inquiries for delusions) How do ye explain (the delusion) to yourself? How do you e perience the belief that (the delusion)? (Judge whether ego dystonic)	
Do you suffer chronic feelings of emptiness or lone ness?	
(After inquiries for delusions) How does your be in (the delusion) affect you? (Judge whether dec sions are widespread)	
Have you ever been dependent on any drug?	
Have you developed special relationships with of the staff or psychotherapists you have been in volved with?	

*Wilks' Lambda=.382; the group centroid for the .823; the group centroid for all the other patients w

DISCUSSION

This study has shown that borderline discriminated with high accuracy from parison groups with whom diagnostic common. The fact that there were fewe with slightly higher power for discrim line patients from schizophrenic patie neurotic depressed patients may reflec the diagnosis of neurotic depression is more difficult differential diagnosis. If s apply only to hospitalized neurotic depre who are a much more disturbed gr hospitalized neurotic patients.

The advantage of a list of the best ov nators developed from items over one de statements is that the items are more dis pler to evaluate or quantify. The list derived from the statements was conde teria that provided a clinically sensible means of approaching the diagnosis of order. These have the advantage of bei ly applicable and conceptually enlighte

Two unresolved questions about this degree to which the discriminating va this study are generalizable to other set diagnostic habits may differ and to wh would hold up if other comparison gr ployed. A potentially more difficult would be one made up of patients with orders not considered borderline. Even ing would be to examine a sample fro

admissions or a cross-section of the general population.

There are several ways in which we foresee these results can be used. The acceptance of a reasonable and replicable approach to the use of the term "borderline" can help clinicians anticipate certain clinical problems and subtypes. Among these are the probable contraindications for unmodified psychoanalytic treatment (7, 8), the questionable advisability of antipsychotic medications (9), the risks of staff splits (10), and the temptation to underestimate or overestimate the patient's potential level of functioning. A major implication of several of the recent reports (5, 11) on the social functioning of borderline patients also targets the low level of work and school achievement as a major problem for such patients and points to the role that social learning programs (groups, rehabilitation, milieu) can have in comprehensive treatment planning.

A second advantage of a practical and replicable guide for diagnosing borderline patients is that it would allow research to proceed into questions whose answers are controversial or unknown. Among these questions are the following: 1) Are borderline patients a heterogeneous group containing subgroups that could more appropriately be placed in other standard diagnostic categories? 2) Do borderline patients have common genetic linkages with chronic schizophrenic patients, as suggested by the adoptive studies (12)? 3) Do formulations employing splitting and projective identification as the predominant defenses (13) or abandonment depression (14) reflect our understanding of these patients; or, as others have suggested, are different formulations required (15)? 4) Do these patients have specific psychopharmacological sensitivity to antidepressants or lithium (9)? 5) Is an expressive supportive psychotherapeutic approach (16) preferable to a more limited ego-directed approach (7, 17)? 6) Are there characteristic interactional patterns or parental psychopathology in the families from which these patients originate? 7) Does the course of these patients parallel that of schizophrenic patients (5, 11)? 8) Do the children of patients designated borderline by these criteria have a high risk for growing up to be chronic schizophrenics (18, p. 25; 19)?

These results provide evidence supporting those clinicians who have argued that borderline patients have a recognizable personality disorder. As Grinker and associates (20, 21) have shown, these results also indicate that a definite syndrome exists. The results clarify what the most discriminating aspects of that syndrome are and provide a replicable means for identifying borderline patients. Further studies that use these criteria are under way to search for external validators in terms of treatment responsivity, course, and etiology; such studies could further establish this syndrome as a coherent psychiatric disorder.

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Personality and the Prediction of Long-Term Outcome of Depression

BY MYRNA M. WEISSMAN, PH.D., BRIGITTE A. PRUSOFF, M.P.H., AND GERALD L. KLERMAN, M.D.

In a follow-up study of 150 women who had undergone treatment on an outpatient basis for acute depression, it was found that the most important predictor of their long-term clinical outcome (8, 20, and 48 months after the acute episode) was personality as measured by the Neuroticism Scale of the Maudsley Personality Inventory (MPI-N). Age, race, social class, marital status, religion, number of previous depressions or suicide attempts, diagnosis, history of early deaths of or separations from significant others, history of neurotic traits as a child, amount and type of stress in the 6 months before onset, and severity and pattern of pretreatment symptoms were not predictive of outcome.

ALTHOUGH CLINICIANS and psychotherapists have long emphasized personality as a major variable in case discussions and theoretical discourse, personality measures usually have not been included as predictors of long-term outcome of depression. Most of the studies of depressive disorder that attempt to predict long-term outcome focus on the patient's pretreatment severity or patterns of symptoms or on diagnostic subtypes (e.g., the psychotic-neurotic, endogenous-reactive, or primary-secondary distinctions). A recent unpublished review of these studies by Shapiro and Klerman,[1] which distinguished between long- and short-term outcome, found few predicte
past history, sociodemographics, a
showed few consistent relationships to
measures of personality were rarely use
indicators.

In the Boston-New Haven Collabora
Depression, 150 women with depressio
with drugs and/or psychotherapy for 8
ing symptomatic improvement of thei
sive episode. Among the various pred
assessed at the start of treatment, th
Scale (N Scale) of the Maudsley Perso
(MPI) proved to be the single most impo
of chronic poor outcome with psychot
drug treatment, as will be described. Th
the end of 8 months of maintenance tre
follow-up 20 and 48 months after the a

METHOD

The full methodological details, inclu
the selection of patients and for the
change, have been described elsewher
cipal criterion for admission into the a
phase was a Raskin depression score
At entrance into treatment, conside
patient's social and psychiatric status

The Patients

The patients were all women whose
the late 30s and who were usually from
lower-middle social classes. Diagnost
typically neurotic depressive patien
bulatory, nonpsychotic, nonbipolar
enced either no previous episodes of d
ly one.

Treatment Phases

Patients who entered the acute treat
ceived 100–200 mg of amitriptyline a
responded after 4–6 weeks of acute tre
the maintenance phase, which took the
balanced factorial design, conducte
(New Haven and Boston). In the mai
all patients were seen monthly by the t
trist in brief interviews for assessment
In addition, half of the patients were

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This work derives from the Boston-New Haven Collaborative Depression Group. Alberto DiMascio, Ph.D., was a senior collaborator.

[1]Shapiro RW, Klerman GL: Predictors of outcome of affective disorders (unpublished manuscript).

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signed to weekly individual psychotherapy. All patients continued to receive amitriptyline for a transitional phase of 2 or more months and were then further randomized into one of three drug procedures. Within each contact group, one-third of the patients continued on amitriptyline, one-third were withdrawn double-blind onto placebo, and one-third were overtly withdrawn from medication. Treatment continued in these six treatment groups for a further 6 months, a total of 8 months, and then ended. Patients were then no longer under the direct clinical responsibility of the two research clinics.

Follow-Up

Twenty and 48 months after maintenance treatment had ended the patients were again located and interviewed. A psychiatrist and a research assistant reviewed with the patients their clinical course and treatment experience since the previous contact. At 20 months, 149 patients were located and followed; at 48 months only the first 94 patients were followed due to the termination of the project.

Criteria for Chronicity

A crucial aspect of the follow-up study depended on assessing the patient's longitudinal clinical course and developing objective criteria for chronicity. Using the information collected at each interview, a month-by-month rating of the patients' clinical status on a 7-point global scale was reconstructed for each month during the follow-up period. On the basis of these ratings each patient received a blind assignment to one of three groups: asymptomatic, moderate, and chronic. The asymptomatic patients were almost entirely symptom free over the period assessed and never rated higher than a 2 more than 1 month out of 12. Patients with a chronic course were never asymptomatic during the period assessed and maintained a continual moderate level of symptoms (mean rating of 3). Patients who did not meet either of these criteria were assigned to the moderate group.

Personality Assessment

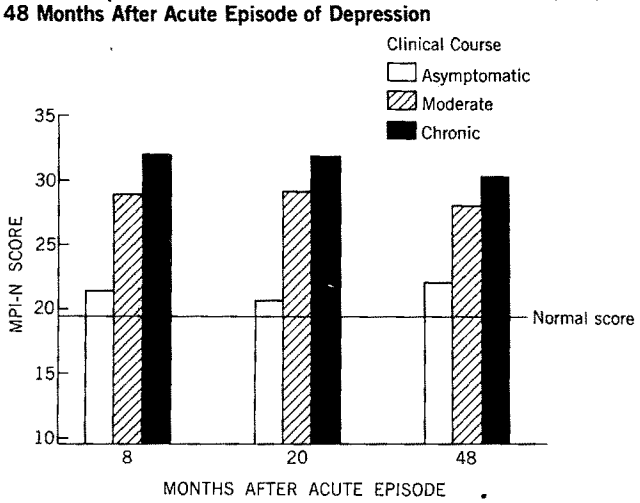
Personality was evaluated using the Maudsley Personality Inventory (MPI), a 48-item self-report scale developed by Eysenck in Great Britain and extensively used as a general mode of personality assessment (3–5). Two scales were derived from the MPI: the N Scale (neuroticism), which is a general index of the patient's predisposition to neurotic illness, and the E Scale, which is related to the introversion-extraversion concept and relies heavily on ideas of Jung. The MPI has been used extensively in Western Europe and the United States, particularly in research on depressed patients, where it has been shown to be related to different types of depression and to differentiate between depressed patients and other diagnostic groups.

The MPI-N was completed by the patients after 1 month of treatment, when they had shown a symptomatic response. This procedure was followed in order to minimize the effects of illness on reporting (6). Subjects were instructed to answer according to how they usually acted or felt. Appendix 1 shows a sample of items from the N and E Scales.

TABLE 1
Clinical Course of Depressed Patients 8, 20, and 48 Months After Acute Episode (in percents)

Clinical Course	Months After Acute Episode 8 (N=150)	20 (N=149)	48 (N=94)
Asymptomatic (complete remission)	31	28	24
Moderate or fluctuating symptoms	50	60	46
Chronic symptoms	19	12	30

FIGURE 1
Relationship Between MPI-N Scores and Clinical Course 8, 20, and 48 Months After Acute Episode of Depression



RESULTS

Clinical Course

Table 1 presents the clinical courses of depressed patients 8, 20, and 48 months after the acute index episode. The 8-month assessment covered the previous 6 months, while the patients were on maintenance therapy. The 20-month period covers the past year since termination of maintenance treatment, and the 48-month period covers the past 2 years. We used the 2-year period because we felt that a longer time span might result in poor recall. In all three periods about one-third of the patients were in complete remission and between 12% and 30% were chronically but mildly symptomatic.

Sociodemographic, Historical, and Clinical Characteristics

Appendix 2 lists the social, historical, and clinical variables that *did not* differentiate the chronic group. The amount of stress experienced in the past 6 months was measured using the Recent Life Events Scale. The

patients' pretreatment symptoms were assessed by a modified version of the Hamilton Rating Scale for Depression, the Raskin Depression Scale, and by two self-report scales, the Inventory of Psychiatric and Somatic Complaints and the Mood Scales (1).

Personality

Of the two scores derived from the MPI, the N score for neuroticism showed a difference, but the E score did not, supporting our conclusion that our findings were not due to a halo effect or to nonspecific response set factors. Compared with the other clinical outcome groups, the chronic patients had higher neuroticism scores initially and at 8, 20, and 48 months (see figure 1). Although the differences did not reach significance in the 48-month period, the trends were in the same direction. The chronic patients were initially more neurotic. The personality scores on the N Scale at the beginning of treatment were predictive of clinical outcome. In fact, there was a gradient in that the chronic patients had the highest initial scores (all above 30). The patients who were asymptomatic had the lowest initial scores (between 21 and 23, which is close to the reported score of about 20 in normal populations). The intermediate MPI-N scores of the moderately symptomatic group were above the normal.

DISCUSSION

These findings show that personality assessment is a useful predictor of the clinical course and long-term outcome of the ambulatory nonbipolar depressed patient. The clinical significance of these findings is that patients with long-standing neurotic maladaptations as measured by the MPI-N are at risk for persisting depressive difficulties. Such patients are particularly vulnerable to poor long-term outcomes. These findings also raise methodological questions about the assessment of personality, as well as clinical questions about the relationship between personality, clinical course, and treatment.

The Assessment of Personality

The relationship between personality and depression has been discussed in the clinical literature for over five decades. Chodoff (7) reviewed the psychodynamic and interpersonal theories related to depression in 1972. Most theories emphasize the etiological relationship between personality and depression and seek personality factors that may be an antecedent to the depression. In this framework, emphasis has been placed on such factors as dependency, guilt, and hostility turned against oneself as well as general neuroticism. A second area of interest in the personality-depression relationship has been the attempt to relate personality patterns to symptom profiles in such a way that relationships have been shown between such patterns as hystericism and obsessionality (8). Relatively little attention, however, has been paid in the research literature to the role of personality as a predictor of clinical course and long-term outcome.

The Maudsley Personality Inventory, the scale used in this research, is a very broad measure designed to assess dimensions of personality, such as neuroticism, hostility, dependency, feelings of guilt, and [illegible] features, that have been identified in the literature as related to predisposition to depression. Future follow-up studies of depressive subjects should include, in addition to the MPI personality measures, some of the newer measures developed specifically for depressed patients and being tested by the staff of the Collaborative Project on the Psychobiology of Depression (9).[2]

State, Trait, Response Set

There has been controversy about the use of personality measures as assessments of trait rather than how much they merely reflect clinical state. Two questions are raised by this controversy: 1) Is personality a direct measure of severity of clinical depression? 2) Does personality assessment reflect response set?

In regard to the first question we found that severity of depressive symptoms and the presence of a previous history of depression did not predict long-term outcome, i.e., the patients who were the most sickest did not have the poorest outcome nor were patients with the most neurotic personality disturbed. Therefore, severity of depression and personality seem to be separate dimensions.

In regard to the second question, the personality measures completed 1 month after the depression still predicted outcome after 8, 20, and 48 months. Therefore, if response set was operating, it is long term and enduring. The fact that the initial scores predict outcome argues against a general response set.

Personality, Course, and Treatment

Although the detailed data are not presented here, there was suggestive evidence that personality may be influenced by treatment. As a general information of the effect of personality on treatment, multiple regression formulas were calculated for the control group and for each of the treatment groups in the maintenance study. Of interest was the fact that in the regression equation for the control group, who received neither drugs nor psychotherapy, the correlation on the MPI-N Scale but not on the E Scale was highly significant. Of the MPI-N scores had the single highest weight in the multiple regression equation. Treatment with therapy and with drugs reduced the correlation of MPI-N score with outcome, suggesting that the effect of treatment was to ameliorate the negative influence of personality variable on outcome.

[2]Hirschfield RMA, Klerman GL, Gough HG, et al. [illegible] interpersonal dependency (unpublished manuscript)

To further search for pretreatment variables that may have predicted the chronic outcome at the 20-month follow-up, we examined all patients who had been assigned to the no-maintenance-treatment group (N=50). We divided these 50 patients as to their clinical course in the follow-up: chronic (N=12) and nonchronic (N=38) (10). We compared the two groups as to social history and pretreatment clinical status. From the large pool of variables, only neuroticism as measured by the MPI-N significantly differentiated the chronic patients. The chronic patients in the no-maintenance-treatment group were more neurotic, suggesting that the "neurotic" patient who receives no maintenance therapy after an acute depression is at greater risk for developing chronicity in the subsequent months. This latter finding was not replicated after 48 months, perhaps due to the reduced sample size or the presence of other intervening factors. Therefore, it must remain speculative.

Together these findings suggest that many chronically depressed patients, who for the most part have neurotic personalities, are patients who have partially recovered from an acute episode and have mild unresolved symptoms. Moreover, these findings highlight the utility of personality assessments in the longitudinal study of depressive subjects. Future longitudinal studies of depression that emphasize prediction should include personality measures in addition to the usual symptom and diagnostic ratings, and further attention should be given to the development of personality measures that include a broad range of dimensions.

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APPENDIX 1
Sample of Items from the Maudsley Personality Inventory Neuroticism and Extraversion Scale

Neuroticism Scale Items

Does your mind often wander while you are trying to concentrate?
Are you sometimes bubbling over with energy and sometimes very sluggish?
Are you often troubled about feelings of guilt?

Extraversion Scale Items

Do you usually take the initiative in making new friends?
Are you inclined to be quick and sure in your actions?
Do you prefer action to planning for action?

APPENDIX 2
Variables That Did Not Differentiate Chronic Depressed Patients from the Other Groups 8, 20, and 48 Months After Acute Episode

Age
Race
Social class
Marital status
Religion
Number of previous depressions
Number of suicide attempts
Early deaths of or separations from significant others as a child
Neurotic traits as a child
Amount and type of stress in the 6 months before onset of acute episode
Severity of pretreatment symptoms

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Side Effects in the Neonate from Psychotropic Agents Excreted Through Breast-Feeding

BY JAMBUR ANANTH, M.D., M.B.B.S., D.P.M.

Neuroleptics, antidepressants, lithium, anxiolytics, and hypnotics may be excreted in breast milk. Because of the danger to the neonate, drugs such as diazepam, lithium, bromides, reserpine, and opium alkaloids should not be given to lactating women, and barbiturates, haloperidol, and penfluridol should be administered with caution. The side effects produced as a result of breast-feeding of the infant by mothers consuming psychotropic drugs are reviewed and possible preventive measures are discussed.

PSYCHOPHARMACOLOGICAL agents are used frequently during pregnancy to control nausea and vomiting and during labor for analgesic purposes, as well as after delivery for anxiety and depression. Thus they may reach infants of nursing mothers receiving these medications. Even though there are many reports of neonatal toxicity resulting from breast-feeding by mothers treated with psychopharmacological agents, no definitive answer to the problem is currently available. In this paper the available literature is reviewed with the following aims: to provide guidelines as to what drugs to avoid giving breast-feeding mothers, to point out the need to anticipate side effects and observe the baby carefully if the mother is taking any drugs, and to point out the importance of monitoring the infant carefully if the mother is taking certain drugs known to produce side effects in the neonate.

BREAST-FEEDING AND DRUG SIDE EFFECTS

Sedatives and Analgesics

Barbiturates and alcohol. The amount of barbiturates excreted in breast milk has not been found to affect the nursing infant (1). Sapeika (2) described a report of drunkenness in an 8-day-old bre
with alcohol in his blood; the mother ha
of port wine within 24 hours. Howeve
amount of alcohol ingestion by the n
does not necessarily have adverse effect
into consideration that a woman who dr
ly may not even want to breast-feed he
the emotional security and gratification
an alcoholic drink may outweigh any dis
cial drinking cannot be treated as tabo
mother. Common sense should dictate
drinking is to be avoided.

Kesaniemi (7) measured alcohol and a
the milk and peripheral blood of lac
drinking ethanol. The results of the stud
er findings that ethanol is found in hu
concentration similar to that in the pe
and that it decreases with decreasing c
of the blood (8, 9). On the other hand, n
was found in the infants' blood even
erable amounts of alcohol were measure
of ethanol that a suckling child could
maternal ethanol metabolism was sig
tively low and unlikely to cause harmf
maternal use of alcohol is temporary. S
demonstrated that the activity of alcoh
hyde dehydrogenases is extremely low
fetus and young infants (10, 11), they a
tible to alcohol than adults are.

Opiates. Although opiates are dete
milk and theoretically could lead to
newborn infant, the amounts in milk a
the risk is relatively small. The average
adone by a nursling whose mother is
this drug has been estimated to be on
too low a dose to produce any effect
peak levels of methadone can occur i
when the maternal methadone level s
lem can be avoided by giving the mo
after the infant's evening feeding and
mentary bottles after the intake of me
and associates (13) found that the pl
adone ratio was 1:0.83 on an average
tained on 10–80 mg of methadone (ave
ly). They concluded that breast-feedi
mitted if desired, even though in 2 of
breast milk concentrations of methad
than plasma methadone levels.

Regarding maternal narcotic addict

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heroin) and its effect on the newborn infant, Cobrink and associates (14) found a positive correlation between the amount of the narcotic in the milk and the mothers' intake of it. Several others (2, 5, 15, 16) have reported that therapeutic doses of morphine to nursing mothers result in the excretion in the milk of small quantities of morphine that do not harm the child. However, narcotics are consumed by addicted mothers in amounts higher than therapeutic doses. With such higher doses there is always the danger of the nursling developing side effects. In one study (14), 13 of 22 breast-fed infants of mothers who had taken 2–45 mg of heroin per day were reported to have manifested withdrawal symptoms. One case report involved an infant breast-fed by a mother who became addicted to heroin after delivery. The baby showed signs of narcotic withdrawal every time the mother was deprived of the drug or whenever the drug was withheld for therapeutic reasons (17). Gradual weaning is one of the treatments proposed for such addicted infants (18). Meperidine has also been reported to be excreted in breast milk, but in quantities too small to affect the nursing child (19).

Propoxyphene. Another commonly used analgesic is propoxyphene (Darvon), which has been found to be excreted in the breast milk of animals. In one study (3) the amount of propoxyphene in the stomach of a nursing rat during the suckling period was only .04% of the total amount administered to the lactating rat. Catz and Giacoia (20) reported in a personal communication from Eli Lilly and Company that following a suicide attempt with propoxyphene by a nursing mother, the quantity of the drug in the breast milk was half the amount in the mother's plasma. A theoretical calculation based on the above shows that an infant weighing 5.4–7.3 kg could ingest up to 1 mg of the drug per day—a significant amount—if the mother were to consume the maximum recommended dose around-the-clock.

Anxiolytic Sedatives

Phenobarbital. Phenobarbital is an important member of this drug group even though it is not used as frequently as in the past. For many years it has been known that this drug is excreted through breast milk (21), and it appears that therapeutic doses of barbiturates to the nursing mother have little or no effect on the infant (2, 4–6, 15, 19). However, one questionable report of toxicity in an infant has been found in the literature (22). In contrast to these findings, Finch and Lorber (23) reported a well-documented case of toxicity involving methemoglobinemia, drowsiness, ecchymosis, and collapse in the newborn breast-fed daughter of an epileptic mother who was taking 390 mg of phenobarbitone and 400 mg of phenytoin daily. The toxicity improved with the administration of 250 mg of whole blood, recurred upon resumption of breast-feeding, and improved when donor's milk was used. In this case the side effects seemed to be fairly well established, but they cannot be attributed to phenobarbitone alone.

Although in general only quantities of barbiturates insufficient to directly affect the infant are excreted through breast milk, even these small quantities are enough to increase the activity of drug metabolizing enzymes in the liver. Phenobarbital in concentrations high enough to produce enzyme induction has been reported in animals (24). It must be stressed that the influence of inducing agents is not limited to a specific group of enzymes. In infants this increased enzyme activity may affect other drugs and substances passed through breast milk, even endogenous corticosteroids.

Diphenylhydantoin. This drug is used to treat epileptic psychiatric patients as well as nonepileptic psychiatric patients. It is excreted in the milk in limited quantities (25, 26). It is generally assumed that no side effects develop in the nursing infant (27, 28); and only one case of methemoglobinemia has been reported (23).

Benzodiazepines. Therapeutic doses of chlordiazepoxide are excreted in breast milk in quantities insufficient to affect infants (4, 19, 20), but central depression similar to that produced by diazepam has been reported (29). Diazepam, after intramuscular injection, has been reported to cross the placenta rapidly (30). The drug is not metabolized at all in the fetal liver or small intestine, and thus the infant is susceptible. Diazepam administered during labor has been reported to affect the temperature regulation of neonates (31).

Diazepam has also been reported to cross into breast milk (4, 19, 32, 33). Patrick and associates (32) described lethargy and weight loss in a nursing infant whose mother was taking diazepam. Erkkola and Kanto (33) reported that from the fourth day on, there was a remarkable decrease in the level of diazepam and its metabolites in the neonatal blood, indicating that the first four days of the neonatal period may be the most susceptible time. Furthermore, these researchers also noted that detoxification of diazepam is accomplished by its conjugation with glucuronic acid, the same mechanism by which free bilirubin is eliminated. It is possible, therefore, that if infants receive diazepam through breast milk or from other sources, physiological jaundice may be prolonged and hyperbilirubinemia may occur. Indeed, this side effect has been noted in the infants of mothers who took diazepam during labor (34). On the basis of the current evidence, it is advisable not to give diazepam to nursing mothers. Chlorazepate may cause drowsiness and should not be administered to nursing mothers, based on the manufacturer's advice (35).

Meprobamate. Concentration of this drug in breast milk is two to four times that in maternal plasma (19). Infants should be monitered for possible signs of drug intoxication during meprobamate therapy.

Beta-receptor blocking agents. Karlberg and associates (36) have indicated that propranolol is excreted freely in breast milk and the actual milk-to-plasma ratio is greater than 1:1. Hence under no circumstances

should this drug be administered to a nursing mother, and no mother taking this medication should be permitted to breast-feed her infant.

Antidepressants

Several studies (3, 4, 15) have reported that imipramine could not be found in breast milk after 5 days of administration. However, Vorherr (37) found that imipramine was excreted in breast milk and that while the drug level in plasma was .2–1.3 mg/100 ml, the drug level in milk was .1 mg. Therefore only .1% of the drug is excreted in the milk per day, and the neonate fed with breast milk should not be affected. Desipramine and amitriptyline are also not excreted in milk in appreciable quantities (4, 19). Therapeutic doses of tranylcypromine are excreted in breast milk in quantities too small to affect the child (4, 15). Therefore antidepressants of both groups can be safely given to a nursing mother. Of 103 nursing mothers who received dextroamphetamine sulfate for postpartum depression, there were no instances in which a baby showed any evidence of stimulation or insomnia (3). Attempts to demonstrate the presence of the drug in milk have not been successful (19).

Lithium. Lithium passes from maternal blood to breast milk. Infants breast-fed by women on lithium therapy do ingest a certain amount of the drug. In fact, Schou and Amdisen (38) have shown that in one sample the lithium concentration in breast milk was about half that in the nursing mothers' blood. Simultaneous measurement of the infants' serum lithium concentrations indicated that the levels were about one-half those of the mothers during the first week of life and about one-third during subsequent weeks. Based on these findings, they commented,

> Arguments can be presented both for and against the view that a lithium-treated woman should be permitted to breast-feed her child. On the one hand, during the pregnancy the child has been exposed to the same lithium concentration as the mother for many months, and it seems unlikely that exposure to a lower lithium concentration for a few more months will do any harm.

However, I feel strongly that any mother on lithium should not breast-feed her baby for the following reasons. Even though the serum lithium concentration in the infant is one-half to one-third of the mother's serum lithium level, the infant's regulatory and excretory mechanisms are not well developed; and tolerance of lithium in intrauterine life does not automatically provide proof of safety during the early extrauterine life. Other researchers (37, 39) have noted lithium levels in breast milk similar to those reported by Schou and Amdisen (38). Perhaps the findings of Tunnessen and Hertz (40) lend support for not permitting the mother on lithium to nurse her baby. They reported a breast-fed baby who had a serum lithium level of .6 mEq/liter, compared with a serum level of 1.5 mEq/liter in the mother and who manifested cyanosis and poor muscle tone. An ECG of the infant showed T-wave changes in the lateral pericardi... were compatible with drug effect.

Neuroleptics

Phenothiazines. Various phenothia... tions including chlorpromazine (3, ... fluoperazine (3), prochlorperazine (3, 15 ... zine (42), mesoridazine, and piperacet... excreted in breast milk. It may be possible ... nothiazine preparations are excreted in ... but no significant neonatal side effects ... observed. Vorherr (37) noted that the ... chlorpromazine excreted in breast milk ... one-third the quantity in plasma. Uhlif ... reported that the daily dose of chlorprom... 200 mg or more for the drug to ente... Blacker and associates (43) found that ... high a dose as 1200 mg, the presence o... zine in breast milk was barely detecta... Carmichael (44) studied 7 women who ... babies while on chlorpromazine therapy ... low-up of 16 months to 5 years, they fou... to be normally developed and healthy. S... have been reported in animal studies ... would seem that the quantities of pher... creted in breast milk are probably harm... nate.

Other Drugs

Lundberg (46) reported that oral ad... haloperidol to rabbit mothers during the ... week induced certain behavioral abno... offspring. Using more specific beha... same group of investigators (47) has s... istration of penfluridol to nursing ra... the first postnatal week impaired the ... conditioned avoidance response in ... weeks after birth (48). To my know... been no clinical documentation of side ... breast-fed neonates of mothers receiving ... O'Brien (19) reported that reserpine c... lactorrhea.

CONCLUSIONS

An important issue in the field of ... clinical pharmacology is the recogni... born infant may be the unintended ... administered to the lactating mother ... compounds have been observed as ha... detrimental, or no visible consequence... Perhaps these contradictory findings ... fact that only certain drugs may produ... effects, and these are contingent ... pharmacokinetics, and individual sus... ever, the question most frequently aske... continue breast-feeding while on medi... psychiatrist involved in treating post...

should be aware of the potential side effects to the neonate resulting from breast-feeding.

Most drugs are excreted in the milk in small quantities, but this fact cannot be too comforting because of the possibility of cumulative effects, slow or absent detoxification, enzyme induction, and limited excretion in the neonate. In addition, even though side effects are not apparent, certain subtle behavioral problems may develop. The physician, then, has two choices. If the mother requires prolonged medication, especially with drugs known or suspected to be dangerous, she should be advised to bottle-feed her infant. If the drug ingested by the mother is not known to be dangerous, she should either be persuaded to bottle-feed or to keep strict vigilance over the infant being breast-fed. It is wise not to prescribe *any* medication to the lactating woman unless it is definitely needed.

With our present inadequate knowledge, it is difficult to prepare a list of drugs that are safe or are harmful to the breast-fed infant. However, we do know that drugs such as diazepam, lithium, bromides, reserpine, and opium alkaloids are to be avoided and that barbiturates, haloperidol, and penfluridol should be administered with caution. Finally, since our knowledge in this area is very limited, more research is needed. Taking a complete drug history and monitoring the infant for possible side effects will ensure comparative safety to the infant and perhaps throw more light on this topic. In all suspected drug-related or unexplained reactions in breast-fed infants, examination of the mother's drug history should be a priority, along with the cessation of breast-feeding, at least on a temporary basis.

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Presidential Papers to Appear in August

The 1978 Presidential Address, the Response to the Presidential Address, and the biography of Dr. Weinberg will appear in the August issue.

Imipramine Response in Deluded Depressive Patients

BY FREDERIC QUITKIN, M.D., ARTHUR RIFKIN, M.D., AND DONALD F. KLEIN, M.D.

The authors examined how delusions and other psychotic features influenced treatment outcome with imipramine in patients with primary depression. Global improvement scores indicated that delusions or other evidence of psychosis do not contraindicate imipramine treatment. This finding does not support a recent report suggesting that deluded depressive patients should not be treated with imipramine. Possible explanations of the discrepancy between these two studies are discussed.

ALTHOUGH A HIGH PERCENTAGE of depressed patients respond favorably to antidepressants, no clear relationship between therapeutic effect and specific psychopathological features of depression has been identified. Glassman and associates (1) reported that the presence of delusions in patients with primary depression is negatively correlated with successful imipramine treatment. Furthermore, they suggested that such patients should be considered diagnostically different, that they represent a separate subgroup of primary depression for which the treatment of choice is ECT. This article elicited a correspondence in which Ziegler and associates (2) suggested that the treatment of choice for depressed delusional patients is amitriptyline. Ulett (2) recommended combined tricyclics and phenothiazines, and Glassman and associates (3) reasserted that all deluded depressed patients should be treated with ECT.

Our clinical experience suggests that the presence of delusions or other psychotic features does not contraindicate imipramine treatment of primary depressive illness. In order to test this we reexamined data from an earlier study testing the effect of imipramine in primary depressive illness.

METHOD

The sample was part of a large placebo-controlled investigation of imipramine and chlorpromazine effects done in the early 1960s. The subjects were inpatients who had failed to respond to nonsomatic hospital treatments. The study design has been presented elsewhere (4).

Data reported here are confined to those obtained from the depressed patients who were randomly assigned to imipramine or placebo. Diagnoses of depression were made by the study psychiatrist (D.F.K.) if the clinical picture consisted of dysphoric mood, persistent anhedonia, loss of interest, and other phasic depressive features. Patients with Kraepelinian features of schizophrenia or with evidence of organicity were excluded. Thirty-four patients received a diagnosis of manic-depressive, depressed type. (The concept of unipolar versus bipolar affective disorders was not prevalent at the time the diagnosis was made.) All patients would almost certainly have met the Research Diagnostic Criteria (5) for major depressive disorder. The mean age of the sample patients was 35.9; there were 12 men and 22 women.

All patients were drug-free for at least four weeks before the study. The LORR Multidimensional Scale for Rating Psychiatric Patients (MSRPP) was completed on all patients before and after the study. In addition, the Global Improvement Scale (GIS) was rated at the end of treatment by the study psychiatrist, who was unaware of which treatment the patients received. Maximum improvement was scored 15; no change, 10; and maximum deterioration, 5. Patients were randomly assigned to placebo or imipramine for a six-week period. Dosage was increased gradually to 300 mg/day on a fixed schedule.

Glassman and associates (1) did not present the specific criteria they used to define their study patients as delusional. There are no psychopathological features universally considered to be associated with the delusional or psychotic form of primary depressive illness. Therefore, we decided to first divide our sample on the basis of a preexisting factor so that as many patients as possible with evidence of psychosis could be separated from those who lacked such features. Since patients had been rated on the LORR by a blind rater, we decided to use the LORR MSRPP Factor F, Perceptual Distortion, to divide the sample into patients with and without evidence of psychosis before treatment. We selected Factor F because it was a preexisting scale and thus free of experimenter bias that might influence post hoc item selection. This decision was made before we examined the frequency distribution of baseline or final scores.

Factor F consists of the following items: implausible

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belief, belief in hallucinatory voices or visions, disorientation for people, and disturbed by hostile impulses. Of all the MSRPP factors it included the most items measuring the type of psychotic state seen in depressed patients. Patients with a positive rating on any item on the F scale were considered psychotic.

The sample was divided into four groups: 1) imipramine-treated patients with no pretreatment evidence of psychosis, 2) imipramine-treated patients with pretreatment evidence of psychosis, 3) placebo-treated patients with no pretreatment evidence of psychosis, and 4) placebo-treated patients with pretreatment evidence of psychosis. A two-way analysis of variance was performed. The hypothesis that the presence of delusions or other evidence of psychosis contraindicates the use of imipramine in depressive disorders would be supported if there was a main effect for drug as well as an appropriate interaction with pretreatment evidence of psychosis.

In an identical manner every item in the LORR MSRPP that could be considered to measure a delusion (i.e., implausible belief, conviction of persecution, belief in external control, and conviction of sinfulness) was analyzed individually using the procedure described for Factor F. We also analyzed for item combinations that seemed particularly relevant to the concept of "psychotic" depression. We believe this would allow a clinician to decide which item or combination of items contraindicates use of imipramine and which do not.

As a further test of whether delusions or other psychopathological features of the psychotic state were specifically associated with poor response to imipramine treatment, pretreatment scores on MSRPP items measuring the psychotic state were correlated with GIS scores within each drug group. Other items from the MSRPP such as depressed mood were also correlated with GIS scores to allow perspective on the extent to which other nonpsychotic features of the depressive syndrome are associated with favorable outcome.

RESULTS

Thirteen patients were randomly assigned to placebo and 21 to imipramine. The 4 placebo-treated psychotic patients (i.e., those having a positive score on any F scale items) had a mean GIS score of 10.5±.58, and the 9 placebo-treated nonpsychotic patients had a mean GIS score of 11.67±2.18. The total mean score of these 13 patients was 11.31. The 9 imipramine-treated psychotic patients had a mean GIS score of 12.56±2.79, and the 12 imipramine-treated nonpsychotic patients had a mean GIS score of 12.92±1.16. The total mean score of these 21 patients was 12.76. The total mean score of the 13 psychotic patients was 11.92; for the 21 nonpsychotic patients it was 12.88.

A two-way analysis of variance indicated a significant main drug effect (p=.032) (see t... interaction of drug effect and pretreat... state was not significant. This indicate... mine is an effective treatment in depres... gardless of pretreatment evidence of p...

As a further test of the usefulness o... depressed psychotic patients, the subg... treated with imipramine was studied a... fect. There was no significant difference... for the imipramine-treated group with... evidence of psychosis compared with t... out pretreatment evidence of psych... t=.405). Imipramine was useful in patie... not there was pretreatment evidence o...

Data were analyzed and presented... fashion for items measuring different m... delusions and other psychopathologic... ciated with psychosis; that is, the sam... vided into four cells on the basis of ... imipramine plus or minus any evidence... belief and treatment with placebo plu... evidence of implausible belief (see tab...

The 4 imipramine-treated patients w... plausible belief had a mean GIS score ... and the 17 imipramine-treated patients ... sible belief had a mean GIS score of 1... total mean score of these 21 patients w... placebo-treated patient who showed im... had a mean GIS score of 10.00, and t... pl...

TABLE 1
Analysis of Variance of GIS Scores of 13 Psychot... psychotic Depressive Patients and Imipramine Versus ...

Source of Variation	Sum of Squares	df	Mean Square	
Diagnosis (psychotic-nonpsychotic)	4.20	1	4.20	
Treatment (imipramine-placebo)	19.67	1	19.6...	
Diagnosis × Treatment	1.17	1	1.1...	
Error	116.14	30	3.87	

*One-tailed.

TABLE 2
Analysis of Variance of GIS Scores of 5 Patients wi... lief and 29 Patients with No Implausible Belief and ... sus Placebo

Source of Variation	Sum of Squares	df	Mean Square		
Presence or absence of implausible belief	.009	1	.009		
Treatment (imipramine- placebo)	18.34	1	18.346	4...	1...
Presence or absence of implausible belief × Treatment	6.23	1	6.23	1...	2
Error	111.15	30	3.70		

*One-tailed.

treated patients with no implausible belief had a mean score of 11.42±.93. The total mean score of these 13 patients was 11.31. The total mean score of the 5 patients who showed implausible belief was 13.20; for the 29 patients who showed no implausible belief it was 12.03.

An identical analysis was done dividing the sample on the basis of presence or absence of conviction of sinfulness (see table 3). The 11 imipramine-treated patients who showed conviction of sinfulness had a mean GIS score of 12.64±2.34, and the 10 imipramine-treated patients with no conviction of sinfulness had a mean score of 12.90±1.60. The total mean score of these 21 patients was 12.76. The 7 placebo-treated patients who showed conviction of sinfulness had a mean GIS score of 11.71±2.36, and the 6 placebo-treated patients with no such conviction had a mean score of 10.83±1.17. The total mean score of these 13 patients was 11.31. The total mean score for the 18 patients with a conviction of sinfulness was 12.28; for the 16 patients without this conviction it was 12.13.

The same analysis was done dividing the sample on the basis of presence or absence of conviction of persecution (see table 4). The 3 imipramine-treated patients who showed this conviction had a mean GIS score of 12.67±1.15, and the 18 imipramine-treated patients without this conviction had a mean score of 12.78±2.10. The total mean score of these 21 patients was 12.76. The 3 placebo-treated patients who showed this conviction had a mean score of 10.67±.58, and the 10 who did not had a score of 11.50±2.12. The total mean score of these 13 patients was 11.31. The total mean score of the 6 patients who showed a conviction of persecution was 11.67; for the 28 patients who did not it was 12.32.

The same analysis was done dividing the sample on the basis of presence or absence of implausible belief and/or belief in external control (see table 5). The 4 imipramine-treated patients who showed either of these beliefs had a mean GIS score of 14.0±.82, and the 17 imipramine-treated patients who showed neither belief had a mean score of 12.47±2.07. The total mean score for these 21 patients was 12.76. The 2 placebo-treated patients who showed either or both of these beliefs had a mean score of 10.50±.71, and the 11 who did not had a mean score of 11.45±2.02. The total mean score of these 13 patients was 11.31. The total mean score of the 6 patients who showed the beliefs was 12.83, and the score of the 28 who did not was 12.07.

The same analysis was done dividing the sample on the basis of presence or absence of any evidence of psychosis (see table 6). The 16 imipramine-treated patients who showed evidence of psychosis had a mean GIS score of 12.75±1.98, and the 5 who did not had a mean score of 12.80±2.17. The total mean score of these 21 patients was 12.76. The 9 placebo-treated patients who showed any evidence of psychosis had a mean score of 11.44±2.13, and the 4 who did not had a mean score of 11.0±1.41. The total mean score of these 13 patients was 11.23. The total mean score of the 25 patients who showed any evidence of psychosis was 12.28; for the 9 who did not it was 12.00.

In all of these analyses there was a main effect for drug (patients given imipramine improved more), no main effect for presence of delusions or other psychotic features, and no significant interaction, indicating that imipramine-treated patients with these psychopathological features improved as much as patients in

TABLE 3
Analysis of Variance of GIS Scores of 18 Patients with Conviction of Sinfulness and 16 Patients with No Conviction of Sinfulness and Imipramine Versus Placebo

Source of Variation	Sum of Squares	df	Mean Square	F*	Significance
Presence or absence of conviction of sinfulness	.761	1	.761	.19	p=.33
Treatment (imipramine-placebo)	17.84	1	17.84	4.54	p=.02
Presence or absence of conviction of sinfulness × Treatment	2.61	1	2.61	.66	p=.21
Error	117.70	30	3.92		

*One-tailed.

TABLE 4
Analysis of Variance of GIS Scores of 6 Patients with Conviction of Persecution and 28 Patients with No Conviction of Persecution and Imipramine Versus Placebo

Source of Variation	Sum of Squares	df	Mean Square	F*	Significance
Presence or absence of conviction of persecution	1.08	1	1.08	.273	p=.30
Treatment (imipramine-placebo)	13.06	1	13.06	3.29	p=.03
Presence or absence of conviction of persecution × Treatment	.63	1	.63	.16	p=.34
Error	118.94	30	3.96		

*One-tailed.

TABLE 5
Analysis of Variance of GIS Scores of 6 Patients with Belief in External Control and/or Implausible Belief and 28 Patients with Neither and Imipramine Versus Placebo

Source of Variation	Sum of Squares	df	Mean Square	F*	Significance
Presence or absence of belief in external control and/or implausible belief	.36	1	.36	.09	p=.37
Treatment (imipramine-placebo)	22.66	1	22.66	6.10	p=.01
Presence or absence of belief in external control and/or implausible belief × Treatment	6.85	1	6.85	1.84	p=.09
Error	111.46	30	3.71		

*One-tailed.

TABLE 6
Analysis of Variance of GIS Scores of 25 Patients with Any Evidence of Psychosis* and 9 Patients with No Evidence of Psychosis and Imipramine Versus Placebo

Source of Variation	Sum of Squares	df	Mean Square	F**	Significance
Presence or absence of any evidence of psychosis	.24	1	.24	.06	p=.40
Treatment (imipramine-placebo)	15.46	1	15.46	3.86	p=.026
Presence or absence of any evidence of psychosis × Treatment	.329	1	.329	.09	p=.36
Error	120.02	30	4.0		

*Includes implausible belief, belief in hallucinatory voices, conviction of persecution, belief in external control, conviction of sinfulness, and irrelevant speech.
**One-tailed.

whom these features were absent. That is, depressed patients with pretreatment evidence of implausible belief, conviction of sinfulness, conviction of persecution, or belief in external control benefited from treatment with imipramine.

To further test whether the presence of delusions contraindicates the use of imipramine, the mean GIS score of imipramine-treated delusional patients was contrasted with that of imipramine-treated nondelusional patients by means of two-tailed t tests. This was done for each of the specific forms of psychopathology. In all cases the differences between means was nonsignificant. For presence or absence of implausible belief the t was .802, for conviction of sinfulness the t was .301, for conviction of persecution the t was .040, for implausible belief and/or belief in external control the t was .802, and for any evidence of psychosis the t was .060. In this sample, delusional depressive patients treated with imipramine did as well as nondelusional imipramine-treated patients.

Table 7 presents correlations between pretreatment scores on MSRPP items and GIS scores within the placebo and imipramine groups. Since a positive correlation indicates that high pretreatment pathology is associated with improvement, support of the hypothesis that imipramine treatment is contraindicated if delusions or other aspects of psychosis are present requires significant negative correlations. Six items were associated with the psychotic state. For imipramine-treated patients the 6 correlations tended to be either positive or essentially zero (see table 7).

The item "ideas of reference," which we believe is not pathognomonic of psychosis, had an insignificant negative correlation. This analysis is consistent with the analysis of variance and suggests that the presence of delusions does not contraindicate imipramine treatment in primary depressive illness.

Caution is required in generalizing from these data to other groups of deluded depressive patients. Delusions and hallucinations were not prominent features

TABLE 7
Correlation Between GIS Scores and Pretreatment S[illegible] Items

Item	Patients Given Placebo (N=13)
Suicidal preoccupation	.18
High hostility	.59*
No mood swings	−.20
Overinhibited	−.01
Belief in hallucinatory voices	none
Implausible belief	−.21
Feelings of external control	−.05
Ideas of reference	−.12
Conviction of sinfulness	.25
Frequent use of complaints	−.38
Extreme body concern	−.54†
Belief in hallucinatory visions	none
Conviction of persecution	−.16
Irrelevant speech	−.21
Emotionally unexpressive	none
Slow reactions	.09
Undertalkative	−.24
Self-depreciatory	−.11
Depressed mood	.06
Emotionally unresponsive	−.10

*p<.001, two-tailed.
**p<.05, two-tailed.
***p<.01, two-tailed.
†p<.10, two-tailed.

TABLE 8
Analysis of Variance of Age of 23 Patients W[illegible] Patients Without Delusions and Imipramine Ver[illegible]

Source of Variation	Sum of Squares	df	Mean Square
Presence or absence of delusions	118.85	1	118.9
Treatment (imipramine-placebo)	635.80	1	635.8
Presence or absence of delusions × Treatment	1.22	1	1.2
Error	5204.48	30	173.4

*Includes any positive rating for implausible be[illegible] secution, conviction of sinfulness, or belief in ex[illegible]
**One-tailed.

of the symptoms of these patients. [illegible] these psychopathological features w[illegible] slight to mild. It is possible that in pa[illegible] sions and hallucinations are more p[illegible] mine might be ineffective, but no po[illegible] this effect has yet been presented.

Since most of the deluded depres[illegible] ported by Glassman and associates [illegible] poorly with imipramine were wom[illegible] exists that there might be some inter[illegible] lusions, and response to tricyclics. T[illegible] tions of pretreatment scores on items [illegible] chosis with GIS scores were examin[illegible] men and women within drug groups

There were no significant effects or even trends suggesting that sex affected treatment outcome. We also examined the ages and sex distribution of patients with delusions to determine if there was anything distinctive about each of these groups. For this analysis any evidence of implausible belief, conviction of persecution, conviction of sinfulness, or belief in external control qualified the patient as delusional. Seven men and 16 women met these criteria, and 5 men and 6 women did not. We examined the ages of these patients by treatment and presence or absence of these psychopathological features. There was a nonsignificant trend for patients receiving imipramine to be older, but there was no difference in age of patients with and without delusions (see table 8). The 14 delusional patients treated with imipramine had a mean age of 38.42±14.7, and the 7 nondelusional patients treated with imipramine had a mean age of 42.50±9.68. The total mean age of these 21 patients was 40.29. The 9 nondelusional patients given placebo had a mean age of 28.44±8.5, and the nondelusional patients given placebo had a mean age of 33±20.3. The total mean age of these 13 patients was 29.85. The total mean age of the 23 delusional patients was 34.09; for the 11 nondelusional patients it was 39.04. The two-tailed t test contrasting the means for the patients who received imipramine showed insignificant differences (t=.774).

COMMENT

The data presented in this paper do not support the hypothesis that imipramine is contraindicated in the treatment of primary depressive illness if delusions or other evidence of psychosis are present.

What possible explanations are there for the discrepancy between our data and those of Glassman and associates (1)? Their sample consisted of 12 women and 1 man; the average daily dose for the women was about 200 mg (3.5 mg of imipramine/kg of body weight). Hordern and associates (6), using a maximum dose of 200 mg/day, also found that the presence of delusions was associated with poor response to treatment with imipramine and amitriptyline.

In the present study all patients received 300 mg/day of imipramine. Previous studies contrasting tricyclic dosage regimens of 300 mg/day and 150 mg/day in depressed inpatients found the higher dosage regimen more effective on a variety of measures (7, 8). It is possible that doses of 200 mg/day are inadequate for deluded depressive patients. In addition, the patients of Glassman and associates received treatment for four weeks and the patients of Hordern and associates for four (an unspecified number of Hordern and associates' patients received treatment for six weeks), but patients in the present study received imipramine for five weeks before a final evaluation. Perhaps delusional patients require longer than four weeks on drug to improve.

Our clinical observations suggest that the manner in which some depressed patients treated with tricyclics improve implies the presence of a threshold that, once reached, is followed by marked clinical improvement. Before achieving the threshold there is little improvement. It is possible that delusional depressed patients are more severely ill, have a higher threshold, and, therefore, require more than 200 mg/day of tricyclics. Another, more remote possibility is that delusional depressive patients metabolize tricyclics in a manner necessitating higher doses of tricyclics to reach therapeutic levels.

Glassman and associates stated that delusional depressed patients should be regarded as diagnostically different from other severely depressed patients. However, other lines of evidence indicate that the presence of delusions does not differentiate two classes of patients with endogenous or major depressive disorder. Although not absolutely consistent, the trend in factor analytic studies has been to differentiate between endogenous and neurotic depressive patients rather than between psychotic and nonpsychotic primary depressive patients. Kiloh and associates (9), in a factor analytic study of 145 depressed patients in Australia, concluded that they had replicated the findings of an earlier study of 143 patients by Kiloh and Garside (10) in Newcastle. Their major finding was that endogenous and neurotic depressive patients can be differentiated and that neurotic depression is probably a dimensional disorder and heterogeneous. However, the endogenous form of the illness appears to be a condition with "a restricted range of clinical manifestations, consistent with an imputed genetic or biochemical basis" (9). Kiloh and associates did not find that the endogenous group split along a psychotic versus nonpsychotic dimension.

Guze and associates (11) divided a group of patients with primary affective disorder into those with and those without psychotic features depending on the presence or absence of delusions or hallucinations. They compared these patients on 40 demographic, family history, or parental variables and 75 symptom variables. No consistent meaningful differences were found between those with and those without psychotic features. We mention this sample of studies of the phenomenology of depressive illness to show that considerable data suggest that the presence of delusions does not indicate a separate class of depressive patients.

Our study fails to corroborate the claim that classifying affective illness into psychotic and nonpsychotic subtypes has differential treatment implications. Nosological formulations should be subject to periodic reevaluation in the light of new information. To clarify the existing ambiguities, studies of delusional depressive patients treated with different doses of imipramine for adequate periods while monitoring drug plasma levels should be done. Until these studies are carried out, asserting that delusional depressive patients form a special subgroup who require special treatment is premature.

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Parent Assessment in Research on the Vulnerability of Children and Families to Mental Disorder

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The research methodology for a current study of the assessment of family factors as they relate to the development of psychopathology in high-risk children is described. Factor analysis of preliminary data from 83 families led to the construction of 6 rating scales on the emotional health of the patient, spouse, and child; the family; and the marriage; these should prove valuable in predicting the outcome of the index child and his family.

THE PROCEDURES and results described in this report are part of the project on Children and Families Vulnerable to Mental Disorder at the University of Rochester School of Medicine and Dentistry. This project, following a high-risk paradigm, uses a convergent, longitudinal design whereby a number of research teams prospectively study a population of families in which one parent has in the past been hospitalized for emotional illness and in which there is a male child of appropriate age. Collaboration of the research teams permits the investigation of a range of variables potentially relevant to the subsequent development of psychopathology in the index children.

METHOD

Sample

This analysis is based on 83 families in which one parent had previously been hospitalized for psychiatric illness and in which there was a male child of age 4, 7, or 10. All families were white and fell into social classes I to IV on the Hollingshead index (1). Families were not included in the analysis if the ill parent's hospitalization was for organic disorder, alcoholism, or drug abuse. The average age of the patients was 37.83 years. In 62 of the 83 families the ill parent was the mother. The diagnoses of the ill parent were distributed as follows: schizophrenia, 24%; affective psychosis, 10%; intermediate, 19%; and nonpsychotic, 47%.

Our investigative team engaged only the parents of the subject families and was generally concerned with documentation of interpersonal and personal (parent and caretaker) characteristics and the nature of caretaking patterns of behavior; characterization of parental-object relations; and analysis of the ill parent's illness, clinical course, and diagnosis.

Procedure

Two techniques were used to gather data—a semistructured interview and a form consistng of 36 adjective pairs (termed the object relations schemata). The data-gathering procedure was as follows: Both the patient and spouse were seen in a session lasting approximately 2 hours. One of the investigators (J.R.) conducted an interview in which the patient was first seen for about 50 minutes, the spouse for about 25 minutes, and then both of them for about 25 minutes. All of the interviews were videotaped. They focused on a number of issues, including the patient's past illness and current health, the effect of the illness on the patient's various social roles and on the family, child-rearing practices and experiences, the marital relationship, and the family interactions. The interviewer followed a preestablished format in that he systematically directed attention to a succession of topics. The interaction regarding these topics was as spontaneous and informal as the relationship with those being interviewed allowed.

While the patient was being interviewed, the spouse, using the object relations schemata, gave descriptions of the following objects: self, patient before illness, patient now, typical spouse, index child, typical child, and other children. Then, when the spouse was being interviewed, the patient gave descriptions that were similar, except that the "before illness" and "now" qualifiers applied to self.

Within several weeks after the data-gathering session the investigators watched a playback of the videotaped interviews. Notes were taken and subsequently used to prepare a narrative summary of the interview. The investigators separately scored each of 107 vari-

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ables according to whether it was judged present (+), absent (−), or unratable (0); for a handful of variables (such as the probable type of emotional illness of a relative), choices were made among descriptive terms. The investigators' judgments were then compared and a consensus reached on variables not rated similarly. The overall agreement between the two investigators and one research assistant was approximately 83%. A diagnostic assessment of the patient at the time of the key admission was made at this time, as determined both from the interviews and available hospital records. There was also a diagnostic assessment of the current status of both patient and spouse. On the basis of this information a judgment was also made regarding the accuracy of the patient's previous hospital diagnosis, and in cases of disagreement, a rediagnosis was made. The current level of functioning of both patient and spouse and the patient's level of functioning during the illness were rated on the Global Assessment Scale of Endicott and associates (2). Finally, ratings were made on work and social competence, the degree of frankness in the interview, and the general orientation of each parent toward difficulties and problems.

The 107 variables, which cover those discussed in the literature (3–29) as well as those developed by the investigators, deal with the marital situation, caretaking, and parental roles, functions, and attitudes.[1] A number of variables were derived from the process described above. In addition to the separate variables rated from the interviews, a series of six desirability ratios were calculated from sets of items referring to the major aspects covered in the interviews (marital relationship, spouse, parenting, family, patient, and extrafamilial aspects). Each of the items in these subsets has been classified as desirable or undesirable according to our combined judgment as to whether the trait or behavior in question would be likely to support or undermine mental health. Variables for which such judgments could not be made with confidence were not included in the ratios. Each ratio score was then calculated by dividing the sum of variables rated in the desirable direction by the number of desirable and undesirable variables that were rated as either present or absent.

The object relations schemata comprise 36 bipolar descriptive items that permit a response on a 1–5 scale, with 3 falling midway between the two poles. The scale has 6 items in each of 6 categories: mood, thinking, task competence, activity-passivity, anger, and interpersonal relations. One item from each category refers specifically to the family. Test-retest reliability for the form was determined by retesting 7 randomly chosen subjects (5 patients and 2 spouses). The average interval between testing was 34 days. Average test-retest correlations (over 36 items) for descriptions of "patient before illness," "patient now," "spouse," and "index child" were .79, .84, .80, and .79, respectively. Prior to use in this study the ob schemata items were given weights acco classification by a lay population in ter bution of emotional illness. These weig used in the calculation of an emotiona each description. Descriptions made b wife with the object relations schemat tercorrelated to indicate the degree ceptions regarding themselves and the this point the data were in the form discrete variables. These variables wa related to outcome data on the index these become available and to variables vestigative teams assess on the same lation. The next phase of this study relating variables by multivariate analys the structure of the underlying dimen

A factor analysis performed on the 83 59 variables yielded 8 factors. Six of the to be of major importance in delineat the family situation. The factors term illness demands by children" and "c sistency" were represented by high loa variables that their further usefulness tionable and they were put aside.

Rating Scales

Although the factors that emerged analysis were made to yield factor sco another approach was taken to pro each family on the 6 primary dimens the factor analysis. The 3 or 4 highest lo on each of the 6 major factors were equivalent to their factor loadings a scales. Scores on each scale could th culated by using a family's data for the The scales can be viewed as approx original factors. This procedure effec saving in the number of variables that tors would be required to measure sho use the dimension employed in this st portant, then, to determine how clo scores (based on 3 to 4 variables) ap factor score coefficient counterparts variables. Correlations between the over 83 families were .78, .91, .85, suggesting an excellent fit for each fi scales are presented in appendix 1. It that the names of the scales were bas analysis and thus represent more va used in the scales.

Scale 1 refers to the spouse's emoti tributing to it are the investigators' jud flected by the Global Assessment Scal tional illness indices of both parents' their spouse. It was not assumed tha tors' judgment was entirely sufficie from the husband and wife's descr spouse added important strength to th on this scale will be highest or lowest v

[1] A sample list of the 107 variables is available from Dr. Romano on request.

sources are in agreement, although it should be recognized that the emotional illness indices reflect an indirect attribution, since they are secondary scores derived from descriptions of the spouse. The factor coefficient counterpart of this scale also loaded highly the spouse desirability ratio, which was not used for the scale because it requires many judgments from interview data.

Scale 2 represents the investigators' judgment of the patient's emotional health. It comprises variables referring both to the past and present. Because the judged intensity of past illness and pre-illness competence are substantially correlated with the current Global Assessment score, this scale can be viewed as referring to the patient's present condition.

Scale 3 consists of items that reflect the patient's self view, its emotional illness index, and its relation to views of the index child and the spouse's view of the patient.

Scale 4 presents the spouse's view of the patient's emotional health. The time focus can be taken as current, since the spouse's view of the patient before the illness appears to be highly related to his or her current view of the patient. This scale differs from the previous one in that the patient's view is not represented, whereas in scale 3 it is the major variable.

Scale 5 refers to the emotional health of the index child. It comprises variables based on the investigators' judgments and the parents' views so that, again, concordance of the three sources will produce the most extreme scores on this scale.

Scale 6 involves the family and the marital situation. The variables derive solely from the investigators' judgments. The 4 variables each reflect a plus (for present) or minus (for absent) rating given after reviewing the videotaped interviews of the family. If insufficient information or clarity is encountered, then the variable is rated zero and not subsequently counted for that family. By and large, the 4 variables on this scale were covered well by the interviews and could be rated for virtually all of the families in the sample. Lack of satisfaction in parenting was inferred from the way the mother described and talked about her activities as a parent. For this item to be scored as present the mother would have expressed essentially no enthusiasm or pleasure regarding her parenting role, and often she would elaborate on its burdens and difficulties.

Freedom of expression between the parents was rated as present when both parents indicated that their relationship included bringing up and discussing their problems and concerns. An absent rating was given when at least one parent indicated that such concerns were not generally aired or discussed.

Substantial conflict between the parents was judged present when they indicated that the expression of disagreement occurred or was a threat on a constant basis. The conflict had to be frequently expressed and had to be continuing so that it appeared to be a virtually permanent aspect of their relationship.

The presence or absence of a capacity to resolve child-rearing issues was inferred from parental descriptions of such issues and how they resolved them. Families that did not identify child-rearing issues of much concern to them were nevertheless rated as having the capacity to resolve such issues. Lack of such capacity was inferred from the presence of unresolved conflict that was an aggravation to at least one of the parents.

DISCUSSION

The data-gathering procedures of this study are pointed toward two types of subsequent analysis. In the first type, single variables (e.g., the effect of a shadow of an emotionally ill ancestor, the spouses assuming new roles in order to maintain the family after the patient's illness, the presence of support from members of the extended family) can be related to child outcome or to other variables. The variables rated from interview material were put in the present-absent format in order to avoid producing multidimensional response alternatives in one item (e.g., the parent's relationship is trusting, subject to frequent disruptions, or based on mutual dependence). Although the present-absent format requires clear ideas of where to draw the line between presence and absence, it was felt that it involves judgments that are simpler, closer to the relevant observational data, and that provide a more sound basis for subsequent statistical treatment, as in regression analysis.

The second type of analysis involves the results of the variable interrelationships represented by the scale scores. The 6 scales provide a multidimensional view of the structure of a family. Each dimension can be thought of as a strength or weakness. However, it should be noted that the dimensions preclude the coherent interpretation of the single variables that comprise them. The dimensions can be related to other variables singly or patternwise. For example, if the spouse's emotional health were a weakness for a family, the presence or absence of other weaknesses could be crucial to the outcome for the index child. Thus a pattern of weakness (low score) on scale 1 would have implications different from those of a pattern of weakness on scale 1 and scale 2 (patient's emotional health).

In summary, this report of work in progress gives data on a partial sample of 83 families studied. As of July 1978 we have completed the study of 148 families and have as our goal the study of 150 families. However, factorial structure has not changed significantly in the analyses of data from 40 families, 60 families, and 80 families. As is evident, we do not yet have any outcome data and therefore no definitive findings; this is essentially an exposition of methods. Factor analysis of our data led to the construction of 6 scales that we anticipate will be useful in predicting the outcome of the index child and his family.

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APPENDIX 1

The Six Rating Scales Used in Predicting the Outc... ...Child and His Family

Scale 1: Current Emotional Health of Spou...	*M... ...gr...*
Global Assessment Scale score for spouse	.8
Emotional illness index for spouse's view of se...	.6
Emotional illness index for patient's view ... spouse	.6
Correlation between spouse's view of self a... patient's view of spouse	.6
Scale 2: Investigators' View of Patient's Em... tional Health	
Intensity of patient's illness in acute phase	).7
Patient diagnostic grouping	).7
Global Assessment Scale score for patient n...	).6
Pre-illness social competence	).6
Scale 3: Patient's View of Own Emotion... Health	
Emotional illness index of patient's view of ... tient now	).8
Patient's view of self now correlated with ... tient's view of index child	).7
Emotional illness index of spouse's self-vi... subtracted from patient's view of self	).8
Scale 4: Spouse's View of Patient's Emotio... Health	
Emotional illness index of spouse's view of ... tient before illness	).7
Correlation of spouse's view of typical spo... with view of patient now	).6
Emotional illness index of spouse's view of ... tient now	).6
Correlation between spouse's view of pa... before and spouse's view of the typical spo...	).6
Scale 5: Emotional Health of Index Child	
Emotional illness index of patient's view o... dex child	.0
Correlation of patient's view of typical ch... with view of index child	.0
Current diagnosis of index child	.0
Correlation of spouse's view of self w... spouse's view of index child	0.5
Scale 6: Marital and Family Stability	
Mother shows lack of satisfaction in parent ...	0.5
Freedom of expression between parents	0
Substantial conflict between parents	0
Parents have lacked capacity to resolve ch... rearing issues	0.5

Explaining the Increased Arrest Rate Among Mental Patients: The Changing Clientele of State Hospitals

BY HENRY J. STEADMAN, PH.D., JOSEPH J. COCOZZA, PH.D., AND MARY EVANS MELICK, PH.D.

The arrest rates of nearly 2,000 psychiatric patients released from psychiatric centers in 1968 and those of nearly 2,000 patients released in 1975 were compared with the arrest rates of a group of 5,000 patients studied 25 years earlier. In general, arrest rates among the two recent samples were considerably higher than general population rates, primarily because of the large proportion of patients previously arrested. Other characteristics related to subsequent arrest were age and admitting diagnosis. One additional finding was that the number of patients with prior arrests had increased markedly over the years.

VERY RARELY is the research literature as consistent in its findings as has been the case in the studies of arrest rates of former mental patients. Investigations reported before 1965 (1–4) noted that in every offense category former patients were arrested less often than were members of the general population. Since 1965 research studies (5–9) have found that in certain offense categories, varying from study to study, former patients were arrested more often than were members of the general population. Most distressing from the policy standpoint was that the offenses for which the patients tended to be most often arrested included assault, robbery, and rape.

All of these studies represent substantial work with unwieldy data that are most difficult to obtain. However, there are some limitations in each study that have restricted their policy applications. For example, the important work by Zitrin and colleagues (8) dealt with but one catchment area in one city (New York City), while another recent study (9) reported statewide data but these were from a highly rural state (Wyoming), thus limiting possible generalizations to more populous areas.

A further difficulty in interpreting these studies is that for the most part, their data relate to only one study group for one time period. This limitation is important, since these data have been interpreted as suggesting some unintended consequences that may have been precipitated by changes in mental health treatment orientations and practices. Unfortunately, the inferences about changing behavior patterns among former patients have rested on comparing one study's data with others, rather than having multiple time periods in the same location(s), which would directly address the changing arrest rates as alterations in the treatment systems were occurring.

The research reported here offers comprehensive data on a statewide sample of persons released from state psychiatric facilities at two different times. Moreover, since the setting of the research was New York State, we were able to compare our results with those obtained 30 years earlier by Brill and Malzberg (4). As a result, long-term trends in the characteristics and composition of patients in New York facilities could be measured. These add support to the recent data on higher arrest rates among persons released from state hospitals than for the general population. However, these data also provide an explanation of these changing rates based on the changing characteristics of persons provided services within the New York State system.

METHOD

Our two major interests in undertaking this research were to obtain comprehensive statewide data as current as possible while still having a reasonably long follow-up period and to obtain the same information on a group that would have been released before the effects of deinstitutionalization were manifest in the New York State system. To meet the first need we selected a sample of former patients who were 18 years of age or older and who were released from psychiatric centers between April 1, 1974, and March 31, 1975.[1] During this time there were approximately 35,000 patients released from state psychiatric centers. We selected every 18th release, which produced a 1975 study group of 1,938 subjects. By following these individuals through March 1976 we had on the average 19 months of risk.

Since the New York State program of reducing the number of inpatients began in earnest during the peri-

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[1]This included patients who were discharged, escaped, or were put on leaves exceeding 5 days.

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od 1965–1966[2], we wished to draw our second study group as close to that period as possible. Because of restrictions in the Department of Mental Hygiene data processing systems, the oldest complete data set available was that for persons released between April 1, 1967, and March 31, 1968. Accordingly, we selected every 14th person released from psychiatric centers during this period to produce a second study group of 1,920 individuals.

Two types of data were gathered for both study groups. First, all hospitalization histories were obtained, together with a range of diagnostic and sociodemographic items stored in the Department of Mental Hygiene's data processing system. Second, the criminal records of those individuals who had prior and/or subsequent criminal records were obtained from the New York State Division of Criminal Justice Services. The division also provided the data from which the arrest rates of the general population were compiled.

FINDINGS

The first data we examined dealt with how many of our two groups of nearly 2,000 released mental patients were arrested after release and particularly how many for violent crimes. Of the 1,920 ex-patients in the 1968 study group, 131 (6.9%) were arrested during their first 19 months after release. Of the 1,938 subjects in the 1975 study group, 183 (9.4%) were arrested. Looking specifically at violent crimes (murder, manslaughter, and assault), 17 of the 1,920 ex-patients in the 1968 group (0.9%) were arrested for such offenses, compared with 33 of the 1,938 in the 1975 study group (1.7%). It is quite important to recognize how few of the persons released from state mental hospitals are arrested (6.9% and 9.4%, respectively, in the 1968 and 1975 groups) and even how many fewer are arrested for violent offenses (N=17 [0.9%] and N=33 [1.7%], respectively). Too often the infrequency of such events is forgotten in the heat of the particularly bizarre or violent behavior of a former patient, which is given considerable media coverage.

However, such absolute numbers are not sufficient to make the equally important comparisons with the arrest rates of the general population, members of which are also seldom arrested. To examine such questions, these absolute figures must be converted to base rates of the number of persons in the various groups who were arrested for every thousand people in each group. The data in table 1 make such a conversion for our two study groups and for the general population of New York for the same years.

As is evident, our data show statewide patterns fairly similar to what Zitrin and colleagues (8) found locally in one catchment area in New York [illegible] all other post-1965 studies in this area [illegible] strated. The major difference between our [illegible] others' is that in both our study groups [illegible] tients were not only arrested more often [illegible] was the New York State general population [illegible] also were arrested more often for every [illegible] gory but one. The only exception to this [illegible] slightly lower rates for sex crimes in the [illegible] For total arrests, the 1968 group was arrested [illegible] as often than the general population [illegible] group was arrested 3.0 times as often [illegible] vidual offense categories, excluding the [illegible] for sex crimes, the former patients were [illegible] 2.1 times as often for potentially violent [illegible] the 1968 group to 7.0 times as often [illegible] crimes in the 1975 group, compared with [illegible] population.

Having found these substantial differences [illegible] ex-mental patients and the general population [illegible] tinued to analyze our data to determine [illegible] tain characteristics of the released patients [illegible] lated to their arrests after release. Our findings [illegible] strated that the overall rates in table 1 [illegible] misleading.

TABLE 1
Annual Arrest Rates per Thousand for 1968 and 1975 [illegible] and the General New York State Population*

Item	Arrest Rate per [illegible]		
	1968 Sample		
	General Population	Patient Sample	[illegible]
Total subsequent arrests	27.53	73.50	
Violent crimes (murder, manslaughter, assault)	2.29	5.58	
Potentially violent crimes (robbery, arson)	1.85	3.94	
Sex crimes (rape, sodomy, sexual abuse)	0.42	0.33	
Drug crimes	2.32	14.77	
Crimes against property (burglary, larceny, forgery)	4.50	23.95	
Minor crimes (vagrancy, public intoxication)	10.19	20.34	

*Per 1,000 persons 18 years of age and older.

Factors Related to Subsequent Arrest

In examining a wide variety of variables [illegible] that there were only three factors that [illegible] tically significant contributions in explaining [illegible] the former patients were subsequently arrested [illegible] der of importance, they were total prior [illegible]

[2]The decrease is apparent in the number of resident patients at the end of the following fiscal years: 1960—93,855; 1965—89,216; 1970—66,529; and 1975—34,032.

TABLE 2
Relationship Between Prior and Subsequent Arrests for 1975 Patient Samples During 19-Month Follow-Up*

Number of Total Subsequent Arrests	Total Prior Arrests: 0 (N=1,428)		1 (N=187)		2 (N=97)		3 or more (N=226)	
	N	%	N	%	N	%	N	%
0	1,387	97.1	158	84.5	66	68.0	144	63.7
1	32	2.2	23	12.3	26	26.8	45	19.9
2 or more	9	0.6	6	3.2	5	5.2	37	16.4

*χ^2=370.72, p<.001, ϕ=.31.

and admitting diagnosis.[3] Table 2 displays the relationship between prior and subsequent arrests for the 1975 study group. As we will do below in our discussion of the relationship between diagnosis and arrest, in the interests of clarity of presentation we have omitted the 1968 data since they are very similar both in the size and directions of the relationships found in the 1975 data. It is more instructive to examine this table than to simply view the .351 zero-order correlation between the two factors (.371 for the 1968 group). From table 2 it is apparent that the strength of the relationship comes from the steadily increasing probability that those with more prior arrests will have more subsequent arrests. Whereas 97.1% of the 1975 group without prior arrests were not arrested, with one arrest this percentage decreased to 84.5%, to 68.0% with two prior arrests, and with three or more prior arrests to 63.7%. Thus the factor of a prior arrest record is systematically and strongly related to subsequent arrest.

As expected, age was also related to subsequent arrest. The median age of those subsequently arrested in the 1968 sample was 42, compared with 51 for those not arrested. The average age of the 1975 former patients who were subsequently arrested was 35, compared with 45 for those not arrested. The zero-order correlation between age and subsequent arrests was .112 for the 1968 ex-patients and .142 for the 1975 group.

The only other factor that was significantly related to subsequent arrest was admitting diagnosis. Table 3 presents this relationship for the 1975 group. What is most apparent is the significant difference in the percent arrested among those given a diagnosis of substance abuse and personality disorder compared with all other diagnostic groupings. Whereas no more than 8% of the subjects in any other major diagnostic category were subsequently arrested, 18% of those with either alcohol or drug diagnoses and 28% of those with personality disorders were arrested. One of the factors apparently causing this relationship was that persons particularly prone to subsequent arrest were younger individuals diagnosed as substance abusers. This interactive effect ultimately indicated that the more important factor statistically was age. Nevertheless, the role of alcohol and drugs in re-arrest rates found in earlier works (9) appears to receive some further support from our data.

[3]The multiple R for these variables for the 1968 group was .400 and, for the 1975 group, .372.

TABLE 3
Relationship Between Subsequent Arrest and Admitting Diagnosis for the 1975 Released Patients*

Admitting Diagnosis	Total Subsequent Arrests: 0		1		2 or more	
	N	%	N	%	N	%
Organic brain syndrome (N=179)	171	95.5	4	2.2	4	2.2
Other psychoses (N=116)	109	94.0	5	4.3	2	1.7
Schizophrenia (excluding paranoia) (N=598)	561	93.8	27	4.5	10	1.7
Paranoid states (N=393)	363	92.4	21	5.3	9	2.3
Neurosis (N=159)	146	91.8	9	5.7	4	2.5
Substance abuse (N=345)	283	82.0	43	12.5	19	5.5
Personality disorder (N=56)	42	72.4	10	72.4	6	10.3

*χ^2=69.90, p<.001, ϕ=.14.

Number of Prior Arrests

After completing our preliminary analyses showing a history of prior arrests to be the most important factor associated with subsequent re-arrest, we recalculated the rates of subsequent arrest for the two study groups, separating the released patients with one prior arrest and those with two or more prior arrests from the patients with no prior police record. These data, reported in table 4, are the most important for properly understanding the arrest rates of former patients.

In fact, the arrest rate in every offense category but one (property offense) for those patients released from New York State facilities in 1975 with no prior police records is lower than that of the general population. Overall, 22.1 per 1,000 former patients with no prior arrests were subsequently arrested, compared with the general population rate of 32.5 per 1,000. Again, the ex-patient with no prior police record was subsequently arrested for violent crimes at a rate of approximately 2.2 per 1,000, compared with 3.6 per 1,000 for the general population. Those patients returning to the community who had no police record before admission had substantially lower rates of arrest than did the general population.

However, when we examine those released patients with 1 arrest before hospitalization, the total rate jumps to 138 per 1,000 for all arrests and to 3.4 per 1,000 for violent crimes. Even more dramatic are the rate increases for persons with multiple prior arrests. Their rate of re-arrest for all offenses leaps to 413.5 per 1,000 and to 60.5 per 1,000 for violent offenses. Thus the arrest rates for all former patients are considerably higher than those of the general population primarily due to those individuals who have been arrested before they entered state facilities. Those with multiple prior arrests are disproportionate contributors. It is only in the multiple prior arrest group (2.9% of all 1,938 sampled patients) that the rate of arrest for vio-

TABLE 4
Annual Arrest Rates per 1,000 for the General Population, Total 1975 Patient Sample, and Patients with Zero, One, and Two or M

Item	General Population (N=12,320,540)	Ex-Patients with No Prior Arrest (N=1,428)	Ex-Patients with One Prior Arrest (N=187)	Ex-Patients with Two or More Prior Arrests (N=323)
Total arrests	32.51	22.06	138.00	413.50
Violent crimes	3.62	2.21	3.37	60.46
Potentially violent crimes	2.83	0.88	3.37	31.21
Sex crimes	0.45	0.44	6.74	9.75
Drug crimes	2.54	1.76	20.21	11.70
Crimes against property	5.13	8.38	30.32	161.89
Minor crimes	19.97	7.45	57.27	118.98

TABLE 5
Comparison of Findings by Brill and Malzberg and Findings from 1968 and 1975 Released Male Patients

Item	Brill and Malzberg 1947 Sample	1968 Sample	1975 Sample
Number of male former patients	5,354	1,075	1,088
Length of follow-up	5.6 years	19 months	19 months
Prior arrest before current hospitalization	803 (15.0%)	343 (31.9%)	435 (40.0%)
Those with prior arrest subsequently arrested	273 (34.0%)	107 (31.2%)	128 (29.4%)
Those without prior arrest subsequently arrested	92 (2.0%)	15 (2.0%)	26 (4.0%)

lent offenses exceeds that of the general population, and quite dramatically so.

While these data are insightful in their own right, they become even more instructive when compared with the earlier work done in New York by Brill and Malzberg, who studied only male released patients (4). In looking at the statewide arrest rates of men released from New York State facilities in 1947, these authors found former male patients arrested less often than the general population for all categories of offenses. While this finding is often cited, what is equally important but usually overlooked is that the rates of subsequent arrest among those ex-patients with prior records were substantially different from those of patients with no arrests prior to hospitalization. Table 5 compares Brill and Malzberg's data in this area with those of the men in our two study groups.

Most striking in the data in table 5 are the substantial increases in the percentage of the state hospital male patients who had a police record prior to their admission. In Brill and Malzberg's study only 15% of the male patients had prior police records whereas our 1968 sample included 32% with prior records and the 1975 group had 40%. This substantial increase is important in interpreting patient arrest rates in that, as table 5 also shows, the percentage of those with prior arrests and those without prior arrests w
sequently arrested has remained practic
over the 30-year-period these data compr
contributed to the increases in overall ra
patients being arrested is that there are
in state hospitals who have records, and
which had a substantially higher probab
arrested after release in 1947 and contin
higher probability in 1968 and 1975. T
without records continue to be arrested

In summary, while the annual arrest
mental patients have risen steadily in the
there has remained an easily demarcated
viduals among former patients who acc
arrests. Because the size of this group
dramatically among persons admitted to
atric facilities, the overall crime rate of
tal patients has increased. It is this fa
York, that explains the finding that fo
were arrested more often than was the
lation. It is likely that a similar phenom
be operating throughout the rest of the
Herein lies the primary explanation for
research findings in this area before ar

DISCUSSION

A number of suggestions have been
plain the increasing arrest rate among
patients. Dominant among these sugge
that recent community treatment ideolo
grams have put more persons who are
into the community. Further, this ex
pointed to the overreactions to such pro
states that have "dumped" persons no
dependent living into high crime areas
developments may have contributed to
found over a 30-year period in New
more parsimonious explanation, which
cally supported from the data reported h
composition of state hospital populatio
in ways directly related to increasing the
postrelease arrest. Rather than focusing
of mental health systems, our data le

more at the systems' inputs, which have changed substantially.

In any group of individuals, those who have previously been arrested are much more apt to be arrested than those without prior arrest. This, of course, is not to say that one could accurately designate those who will be arrested by this single factor without inaccurately selecting many who would not be arrested. However, if one were to gather a group of men, of whom 40% had previously been arrested, from the general population, it is quite likely that the arrest rates found among the 1975 former patient group would be duplicated or exceeded. It may be surprising, but there are no data to accurately indicate what percentage of the general population has ever been arrested. While police agencies at both the state and federal level can provide data on the rates of arrest, they are unable to offer current figures on how many people have ever been arrested. Brill and Malzberg reported an estimate from a correctional source that placed this figure in the mid-1950s at about 5% of the general population. This estimate contrasts sharply with the 15% of Brill and Malzberg's sample of men who had previous arrests and even more so with the 32% and 40% in our two study groups previously arrested. These discrepancies in themselves point out why the general population and ex-patient rates vary so widely and why arrest rates of former patients have increased so substantially over the past 30 years.

Just why the number of patients with prior arrests has changed goes beyond the data we have gathered. The exchanges in responsibilities that may have occurred between the criminal justice and mental health systems over 30 years would require analyses beyond those possible here. Similarly, one could only guess at the ways in which the recent state of mental health litigation has contributed to these changes in patient characteristics. Whatever the causes, it is certain that there has been some shift in practice, if not in policy, so that persons who formerly would have been caught in the "revolving cell door" are now bouncing back and forth between state hospitals and jails as solutions are sought in mental health treatment for what are usually nuisance behaviors or property offenses. It is instructive to note how accurate for our data are the observations of Drs. Brill and Malzberg, who wrote nearly 25 years earlier:

> Arrests in the ex-mental hospital patients were very largely concentrated in a relatively small, rather well-demarcated group of persons with previous criminal records, and their anti-social behavior was clearly correlated with well-known factors which operate in the general population and was not correlated with the factors of mental illness except in a negative way. Available standards indicate that the rate of subsequent arrest among ex-mental patients with a record of criminal arrest prior to hospitalization was, if anything, lower than rates of rearrest in persons with similar records who had not been in a mental hospital.
>
> The conclusion drawn from this survey is that attack of mental illness with hospitalization does not tend to leave an inclination toward criminal activity greater than that which existed prior to the illness and that it does not produce such a tendency if it did not previously exist. . . . (4, pp. 12–13)

Many changes in mental health treatments and ideology may have occurred since 1947, but the fundamental explanation for the arrest rates of former mental patients posited by Brill and Malzberg remains true today.

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Animal-Human Correlates of Narcotic Dependence: A Brief Review

BY BARRY STIMMEL, M.D., AND STANLEY D. GLICK, PH.D., M.D.

Knowledge of opiate action in laboratory animals can be of great value in developing a rational clinical approach to the treatment of narcotic dependence. Physiological alterations experimentally produced by narcotics have been confirmed in human subjects, and classical laboratory effects of conditioning and operant behavior also have clinical counterparts. Realization of animal-human correlates of narcotic dependence allows a better understanding of the strengths and weaknesses of existing therapeutic modalities.

IN ATTEMPTING to provide a better understanding of the pathophysiology of human disorders, scientists are constantly searching for animal models of human diseases which they can study and more clearly define. Despite our similarities to mammals and even primates, such attempts in most instances are frustrating. One area that has been fruitful relates to the study of pharmacological and physiological effects of narcotic drugs in producing dependence, tolerance, and withdrawal.

Although the exact mechanisms underlying these phenomena are still unknown, recent exciting advances have been made with respect to the location of opiate receptors (1). Opiate receptor activity is found in the amygdala, periaqueductal gray matter of the midbrain, hypothalamus, caudate nucleus, and the medial thalamic area. Other areas of the thalamus and the cerebral cortex have also been shown to exhibit opiate binding to various degrees. In addition, brain extracts from naïve animals have been found to contain morphinelike substances which not only bind to the opiate receptor but also have opiatelike effects on isolated tissues. Such findings will undoubtedly stimulate further investigations to clarify the mechanism of analgesia, euphoria, tolerance, and dependence readily observed with narcotic agents. A great deal of information has already been elicited concerning the behavioral effects of narcotic agents on the organism. These observations can be of help in clinically unders addicts' compulsive craving for narc great difficulty in maintaining abstinenc

Unfortunately, this information has n disseminated and is known more by scie engaged in research in drug dependence professionals clinically active in the fie sulted in the proliferation of theories ab of narcotics on psychosocial behavio ance of advocates of a particular ther to the exclusion of all others.

The purpose of this paper is to review of animal behavior that are affected by n dence and, with the use of this knowled the advantages and disadvantages of c ties of therapy. It should be emphasize view is directed toward the health profe field of drug dependence. For this reas presented an exhaustive bibliography o detail experiments conducted at the lab It is also important to realize that focusi cological and physiological effects of dr way negates or minimizes the psych cioeconomic forces that undoubtedly p in initiating narcotic dependence and in preventing maintenance of the abstin addicts.

DEPENDENCE

Wikler (2) has used a conditioning th dependence to formulate an operatio framework for relating experimental a pects of narcotic dependence. Drug dep fined as "habitual, nonmedically indica ing and drug using behavior which is co maintenance upon pharmacological an not necessarily upon social reinforcem concept, one can measure the overall s dependence by its resistance to extinc degree of physical dependence being re verity of withdrawal symptoms induced ministration of the drug is discontinued

Narcotics readily produce dependenc animals. Although the addition of morp ing water results in a bitter-tasting liqu dence has developed rats choose morp and self-administer the drug, even in th

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sive stimuli (3, 4). Although it is frequently assumed that repeated administration of a narcotic for a prolonged period is a prerequisite of dependence, this is not the case. In naïve chronic spinal dogs, phenomena indicative of hind limb abstinence can be precipitated by nalorphine administration 1 hour after a single dose of morphine (5). Similarly, in man, unequivocal physical abstinence phenomena indicating dependence can be precipitated by nalorphine injection after administration of analgesic doses of morphine, methadone, or heroin four times a day for as few as 2–3 days (6).

Physical dependence need not be present for reinforcement to be obtained from opiate administration (7). Environmental factors or expected responses from injection of a specific agent may provide reinforcement, even when there is no opiate in the solution administered. Stolerman and Kumar (4) have demonstrated the presence of conditioned reinforcement in rats used to ingesting bitter morphine solutions. These animals, when offered a bitter-tasting quinine solution, consumed more than naïve rats because of the prior association of bitterness with morphine reinforcement. Similarly, clinical experience has shown that individuals accustomed to injecting heroin cut with quinine may experience a "high" when only quinine is injected and, indeed, may have been consuming such a poor quality of heroin that they may not show signs of physical dependence when challenged with a narcotic antagonist (8).

There is evidence that the acquisition of a drug "habit" may be facilitated in the presence of anxiety or stress. Rats will learn to self-administer amobarbital when a state of anxiety is provoked by the use of random electric foot shocks (9). Clinically, in addition to the fact that anxiety facilitates narcotic use, the cultural phenomena surrounding the injection of narcotics introduce the user into a defined social group, albeit a deviant one, which also serves as a positive reinforcement.

TOLERANCE

Narcotics also have the ability to produce tolerance on repeated injection. The development of tolerance also affects the self-administration of the drug, necessitating higher doses for a pleasurable effect to persist. It is not surprising that the response of laboratory animals does not differ from that of humans in this respect. Daily intravenous self-administration of morphine by rhesus monkeys will increase gradually over a course of 4–6 weeks and then stabilize, presumably indicating that tolerance has developed (10).

CHRONIC EFFECTS OF OPIATE USE

Physiological alterations produced by narcotics have been shown to persist long after administration of a narcotic has been discontinued and in the absence of obvious withdrawal symptoms. Altered responses of laboratory animals to painful stimuli have been found up to 12 months after production of morphine dependence (2, 11). Loss of tolerance to morphine analgesia in rats was incomplete up to 12 months after morphine withdrawal, with EEC responses to morphine challenge remaining different from those of naïve control animals (11). Clinically, alteration of the respiratory center of man to atmospheres containing carbon dioxide has likewise been shown to persist for prolonged periods of time following discontinuation of narcotic agents (12).

ABSTINENCE AND WITHDRAWAL

The physiological disturbance of the abstinent state can be well demonstrated in the laboratory animal. This is not only related to the actual physiologic and pharmacologic effects of ceasing narcotic administration but also can be the result of conditioning. A conditioned abstinence syndrome can be produced by the use of paired nonaversive stimuli given during the production of abstinence when the animal is dependent. Thus narcotic-free rats will undergo withdrawal when placed in an environment that has been associated with production of the abstinent state during previous physiological withdrawal (13). Monkeys that have been narcotic-free for up to 4 months will experience withdrawal in response to a visual or auditory stimulus previously associated with a withdrawal-inducing nalorphine injection (14). Wikler (6, 15) has been able to provide evidence for the conditioning ability of nalorphine-precipitated abstinence in man by producing abstinence when saline injections were substituted for nalorphine. This conditioning effect has long been known "on the street," where addicts become uncomfortable and actually develop signs and symptoms of withdrawal when, in the narcotic-free state, they enter an environment where they had previously experienced withdrawal.

It is also important to realize that symptoms and signs of withdrawal, although most prominent in the immediate postnarcotic dependent period, have also been found to persist for considerable periods of time. Narcotic-dependent rats exhibit signs of abstinence that may persist for 5 months after withdrawal of morphine (16). Similarly, monkeys have been shown to exhibit abstinence phenomena for up to 120 days after discontinuation of narcotics (17). Such animals, if previously trained to self-administer opiates, will relapse promptly after morphine withdrawal. Rats will consume a significantly larger volume of an opioid solution than control animals for up to 1 year postaddiction (16). This may be associated with increases in metabolic rate, norepinephrine excretion, and locomotor activity (18). Whether such a relapse results from permanent physiological alterations in the organism induced by initial morphine dependence, a protracted physiological abstinence state, a conditioning process,

or a combination of all three has not been clearly determined. Nonetheless, such effects do persist, have been documented in man for up to 30 weeks post-withdrawal, and must be considered in the therapy of heroin dependence (19).

THERAPEUTIC GOALS

After consideration of the knowledge gained from animal experiments, it becomes important to outline desirable objectives when dealing with the narcotic addict. It is not sufficient to merely withdraw the addict from narcotics either abruptly or with slow detoxification. One must address 1) reasons leading to narcotic use, 2) actual physiologic changes produced by chronic narcotic use as well as acute and chronic abstinence, 3) existing stimuli associated with narcotic reinforcement, 4) operant behavior surrounding self-administration, 5) existing stimuli associated with withdrawal, and finally 6) means of altering long-term behavior so as to maximize the possibility of an individual remaining narcotic-free. However, it should be emphasized that, if the physiologic changes produced by chronic narcotic use in an individual are sufficiently profound, maintenance of abstinence may not be realistic. In such cases therapeutic goals would be directed toward the first five factors, with alteration in long-term behavior implying the ability to lead a socially productive and personally fulfilling life without abstinence being a primary goal.

RESIDENCY HOUSES

Most persons respond to pain or anxiety in one of several ways: fear, rage, or isolation. Manifestations of the fear are flight or denial; of rage, acting out; and of isolation, internal withdrawal or "encapsulation." Heroin use facilitates the "encapsulation" response by allowing the addict to build a shell around himself that must be removed before the therapeutic process can begin. Residency houses attempt to explore the individual's feelings and get through the "impenetrable" barrier by group processes centralized in encounters, either pro- or introspective. Existing stimuli associated with drug reinforcement and withdrawal are removed through the encounter mechanism as well as by forced compliance with the "house rules." Deviation is not tolerated, infractions are dealt with, often severely (e.g., aversive stimuli). The operant behavior surrounding narcotic administration is attacked through incarceration (albeit voluntary) within the community setting, associated with close observation by not only staff but also other residents. Verbal therapy through encounter groups as well as individual contact serves to facilitate extinction of previously conditioned responses. Gradually the support offered by the staff as well as the punishment they deliver creates a role model through which the individual is able to alter behavior on a prolonged basis in the abs[illegible] sive stimuli. Education and training, ini[illegible] by assigning jobs of increasing difficul[illegible] house setting, can be expanded to profe[illegible] tional training and/or formal education.

Such therapy would be most success[illegible] persons whose physiologic dependence [illegible] has not been firmly established or w[illegible] traordinarily high degree of motivation [illegible] narcotic use. The presence of actual p[illegible] dence is easily identifiable in the abse[illegible] testing with a narcotic antagonist, even [illegible] of prolonged narcotic use exists. Blacidy [illegible] over 40% of applicants to a methadone [illegible] program with a positive history of he[illegible] oin in their urine showed no withdraw[illegible] following naloxone administration. Alth[illegible] psychologically dependent on the drug, [illegible] have not developed a true physical de[illegible] cause they have used an extremely poo[illegible] oin or have used the drug intermittently[illegible] hand, individuals who are not able to tol[illegible] environment or who have a severe de[illegible] narcotic agent may find great difficulty [illegible] their increasing anxiety in such settings. [illegible] plain the high "split" rate seen in re[illegible] houses, as well as the use of alcohol or t[illegible] lieu of narcotics. When the closed env[illegible] primary inhibiting factor, benefit may be [illegible] drug-free programs on an ambulatory ba[illegible] experience has indicated that ambulat[illegible] usually does little to remove the existin[illegible] ciated with drug use or the operant beha[illegible] ing self-administration. For this reason [illegible] are rarely effective in enabling an indi[illegible] tain the abstinent state.

NARCOTIC ANTAGONISTS

The use of narcotic antagonists (cyclo[illegible] one, or naltrexone) in heroin-dependen[illegible] have been detoxified from narcotics will[illegible] the euphoric effect of heroin should it be [illegible] Such agents, however, have the potent[illegible] inforcing the stimulus associated wit[illegible] administered too early in the abstinen[illegible] tion, experimentally, antagonists at lo[illegible] been found to have a reinforcing effect [illegible] opiate administration. Only at higher [illegible] tinction occur (20). The initial occurrenc[illegible] morphine-seeking activity may be therap[illegible] eficial if a daily blocking dose of an ant[illegible] ministered, as it will facilitate extinction[illegible] ty when the reinforcing effect of eupho[illegible] tained. Once this behavior has been e[illegible] may be possible to discontinue use of th[illegible] tagonist. If relapse is purely psychologi[illegible] repeated administration of an antagonis[illegible] in a marked diminution of heroin use and [illegible]

relapse. If, however, persistent physiological and biochemical alterations induced by narcotic drugs are prominent, once abstinent such a person might find great difficulty in maintaining the drug-free state, and administration of narcotic antagonists might intensify the physiological effects of chronic abstinence. In this regard, it is also important to realize that most narcotic antagonists also have an agonistic effect and are mild analgesics with the capacity to alter mood and cause some degree of euphoria and dependence. Although euphorigenic effects without sedation, associated with increased activity, have been considered to enhance rehabilitation efforts (21), the danger of dependence upon such antagonists is real and must be considered quite carefully (22). Cyclazocine and nalorphine can produce physical dependence similar to that seen with morphine. Discontinuation of these agents also results in a comparable abstinence syndrome, somewhat slower in onset and associated with drug-craving or drug-seeking behavior (23). Naloxone is an exception in that it has only antagonist effects. Unfortunately, the short duration of action of naloxone, associated with its relatively low potency and its high cost, has prevented its use as a long-term pharmacologic agent (24, 25). Naltrexone, the cyclopropylmethyl analogue of naloxone, is also reported to have minimal agonistic properties and is currently under study (26, 27). Further work is necessary before these agents can be recommended as standard alternative therapeutic modalities for heroin dependence.

The use of narcotic antagonists in combination with behavioral therapy has been advocated by Wikler (15). With this technique the addict who has been detoxified and maintained on an antagonist in an inpatient setting is presented with controlled conditioned stimuli to elicit the conditioned response, which is subsequently eliminated through supervised self-injection of the narcotic in doses insufficient to overcome the antagonist blockade. It is anticipated that subsequent to the repetition of this process, previously experienced stimuli will cease to evoke a conditioned response. Once the patient is discharged, therapy with the narcotic antagonist might be continued for at least a year to maintain extinction should a relapse to previous conditioned stimuli occur in the ambulatory setting.

METHADONE MAINTENANCE

The substitution therapy most often used in this country is methadone maintenance. The reasons for using methadone rather than other narcotic agents relate to its long duration of action, its effectiveness when administered orally, and its inability to cause euphoria or somnolence when given in appropriate doses to tolerant individuals. Thompson (28) has demonstrated experimentally the ability of methadone to suppress self-administration of morphine, thereby diminishing the probability of seeking further drug reinforcement. Methadone extinguishes the operant behavior associated with withdrawal. When administered in appropriate dosages so as to slowly raise the tolerance threshold, it will also extinguish morphine-seeking behavior by preventing euphoria following injection of street heroin. This last point is worthy of emphasis, since methadone, like any other narcotic, can cause euphoria and/or narcosis when administered to a nontolerant individual or in a dose that exceeds the individual's tolerance. Administration of methadone, therefore, allows the addict to remain comfortable, free of both the psychological anxieties and physiological realities associated with withdrawal. The protection against the "high" is similar to that achieved with narcotic antagonists, although, unlike the person on antagonists, there is no discomfort related to abstinence. Methadone also eliminates operant behavior surrounding self-administration, but only of the narcotic agents. If anxiety still exists, the individual will seek other mood-altering drugs. This explains the not infrequent association of polydrug abuse in methadone programs, most prominently drugs in the alcohol-barbiturate-tranquilizer group.

Methadone and other pharmacological modalities, however, do not address either the factors that lead to heroin injection or the alteration of behavior to effect long-term changes. For this reason pharmaceutical agents should be only one component in the rehabilitative effort. Such programs should be staffed with multidisciplinary professional personnel to provide the ex-addict with the psychological and vocational support necessary to allow the fullest realization of his or her potential. It should be emphasized that not everyone is a candidate for long-term methadone maintenance. For those whose addiction is of short duration or is intermittent rather than constant, another therapeutic modality may be of value.

CONCLUSIONS

It is quite important, regardless of what modality is recommended, to realize that many factors related to persistent heroin use are not imagined but are based on solid experimental evidence. It is extremely difficult for a heroin addict to give up the "habit." This difficulty is not necessarily associated with a weak personality; it is related to the existence and persistence of pharmacological, physiological, and conditioned behavioral changes. Each person must be individually evaluated to assess the prominence of these factors, with subsequent recommendations to appropriate modalities. When this is done, the chances of successful rehabilitation are maximized to the benefit of both the individual and the community.

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Causes for the Premature Interruption of Psychotherapy by Private Practice Patients

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Thirty patients who discontinued private psychotherapy prematurely were evaluated. Factors stemming from the dynamic interaction between the patient and the treatment process were the most common reasons for dropping out, followed by chronic character traits such as impulsivity and masochism. It is hoped that this assessment of variables in the premature termination of therapy will serve to alert the therapist to the probability that treatment will be aborted, suggest which mechanisms will cause interruption, and enable the therapist to use preventive measures sooner.

THERE IS AN established bibliography dealing with outcome in psychotherapy, predictability, and reasons for patients dropping out. However, the studies in this area are generally limited to the measurement of factors that are not difficult to define, objectify, and quantify. They include demographic variables such as age, social class, and intelligence, which can be readily and objectively measured, in addition to such factors as the patient's ego strength, diagnosis, and motivation and the therapist's empathy, which are also not very difficult to assess. What is lacking in this area of research is an effort to elucidate psychodynamic factors in the therapeutic process that lead to premature interruption and their interaction with other variables. Only with this more subtle information will the therapist be able to deal effectively with premature termination, both preventively as well as when the crisis develops. Without these data the therapist is equipped only with information as to which patients are likely to drop out of treatment and which are not.

This area has not been researched adequately because of the difficulty in identifying psychodynamic elements on a cause-and-effect basis from a myriad of alternatives. This requires a degree of inference that makes the resulting judgments vulnerable. Another recognized difficulty is defining the psychodynamic variable itself (such as transference) so that several experienced judges will share a commonality of meaning.

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LITERATURE REVIEW

Brandt (1) reviewed 25 studies of adult patients who were in individual long-term psychotherapy at outpatient clinics and terminated clearly without the therapist's concurrence. "Acceptors" of treatment had a high level of education, annual income, and motivation and were more likely than the "rejectors" to have been referred by physicians, courts, and themselves rather than by social agencies. McNair and associates (2) found that remaining in therapy was related to the degree of interest the therapist expressed in the type of problem presented by the patient. Borghi (3) observed that termination of therapy was a result of incongruence between expectations of the patient and the therapist. Shapiro (4) studied 24 child and adolescent clinic patients and found that premature termination may be a behavioral reaction to the negative feelings of therapists and that families seem to evoke less negative countertransference than do individual patients. Weber and associates (5) observed that unfavorable outcome was related to severity of pathology, such defense mechanisms as denial and projection, fragile personality organization, a proclivity for strongly dependent transference relationships, and a tendency to elicit negative countertransference.

In a comprehensive review Luborsky and associates (6) examined 166 "valid" quantitative studies of factors that influenced the outcome of individual psychotherapy for selected adult patients from 1946 through 1969. Among patient factors, adequacy of general personality functioning, motivation, expectation of change, payment of fee, intelligence, anxiety, and affect all correlated positively with outcome and nontermination, as did self-awareness, interest in human relations, achievement, and likability. Ethnocentrism, defensiveness, and somatic concern were negatively related to outcome. Among therapist factors, level of experience, skill, and interest as well as empathy, warmth, and genuineness were positively correlated, as was similarity between the therapist and patient in social class, interests, values, and orientation to interpersonal relations. Seeman (7) noted that terminators

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tended to drop out of therapy with the advent of transference elements similar to those that were prominent and troublesome in adolescence and that led to avoidance instead of intrafamily confrontation and resolution.

Weiner (8) attributed psychotherapeutic impasse primarily to incongruence of the therapist's and patient's goals, inappropriateness of the therapy prescribed, lack of rapport, and an antitherapeutic alliance. Kohut (9) discussed analysands who quit suddenly because of a narcissistic insult or threatened deprivation. Cooperman (10) emphasized the need of the patient dropout to defeat the therapist by thwarting the treatment or breaking it off. Rosenfeld (11) underscored the interruption of therapy based on a negative therapeutic reaction. Loewald (12) observed that the more intractable negative therapeutic reaction was refractory to interpretation because of guilt and the need for punishment. Stone (13) identified the causes for premature interruption in the therapy of wealthy individuals as extreme narcissism and manipulation by the patient and envy, contempt, and corruptibility through overcharging by the therapist.

METHOD

We undertook to study a series of 30 prematurely terminating private outpatients from the standpoint of psychodynamic as well as fixed variables. Our sample was different from that in most studies of patient dropouts, which have dealt with clinic patients from a lower socioeconomic group.

We met on alternate weeks. We were similar in that we ranged in age from 38 to 46, had been in practice for 12 to 17 years, were university trained, and spent most of our workweek in private practice in Manhattan doing primarily psychoanalytically oriented psychotherapy of a supportive-expressive type. One case was presented at each meeting, with an emphasis on data concerning the premature interruption of therapy. The time at each session was divided approximately as follows: 1 hour for a general presentation of the case and 30 minutes for a discussion of factors specifically related to the interruption of therapy. Each of us, using an inventory of causative factors, then rated the case independently in order to assess rater reliability. This was followed by another 30 minutes for general discussion and elucidation of the data. Finally, there was a composite rating of factors based on the four individual ratings.

There was no preselection of patients in that each of us presented all of the cases qualifying as premature interruption that occurred in our practice from a point 2 years before the beginning of the project. The criteria for premature termination of therapy were as follows: 1) the patient had weekly sessions for a minimum of 3 months and 12 treatment hours, 2) the ending was relatively abrupt, 3) termination was unilateral and without the therapist's concurrence, and 4) the main goals remained unaccomplished. Each of us ha[d] [to] cases. All patients were treated contin[uously] schedule of 1 or 2 sessions per week. Th[e] duration of treatment was 9 months and [the] ber of sessions 41. Most of the patients [had] out of therapy within 6 months of the ti[me] were presented, and 67% of the subjects [were] ment during the project; hence we were [alert] of premature termination and the fact [that] Cases were not presented until 4 month[s] nation to ensure that they were true ter[mi]nations not just "recesses."

In constructing the inventory of reason[s] leaving therapy, we attempted to be over[in]clusive listed variables that could prove valid as [well] that we thought would not. Although a [5]-point was used to rate the degree to which a ca[use] was involved in the termination of thera[py], the number of cases falling into each point [was] sary to regard the variable as existent [or] rather than as quantitatively assessed. [Cate]gory definitions were continuously discusse[d] would retain a reasonably uniform und[er]standing the terms and concepts.[1] In addition to [the] ry items, several other factors were d[e]emed consensus to lead to premature terminat[ion]. [They] sisted of social position (using the two-[fac]tor [14]), ego strength (using six separate [fac]tors and diagnostic category. Diagnoses follo[wed] (15) except for the inclusion of the cate[gory] derline" as defined by Gunderson and [Kolb].

Reliability, as measured by instances [of] ment among the 4 of us, was computed [by] methods on a randomly selected sampl[e] The first method was percent agreement [for] each item (see table 1). Seventy-five-[per]cent ment meant that we agreed on the scoring [for] out of every 4 cases. The overall agreem[ent] The second method of computing reliabi[lity] basis of percent agreement on the 22 [items] case. The average amount of agreeme[nt] which meant that we were in total agre[ement] 1 rating level on 18 of every 22 items. [The] agreement remained uniform throughou[t].

RESULTS

The principal findings in this study [are] prevalence of causes for patients leavi[ng] table 2). Most of the causes for droppi[ng] ment could be divided into four major [ca]tegories. largest category, "reactive to treatme[nt]," 87% of the patients and involved factor[s] dynamic interaction of an evolving th[era]py with the patient's anxieties and conflict[s]. this category were more treatment-evo[lved] or

[1]A list of selected category definitions is availabl[e] D. Le[vin]son on request.

TABLE 1
Percent Agreement Among Four Psychiatrists on Rating of Causes for Patients Leaving Therapy*

Cause	Percent Agreement
Fear of loss of defenses	75
Fear of dependence	67
Fear of aggression or sexuality	67
Transference	42
Decompensation	75
Masochism	83
Poor motivation	100
Negativism	75
Flight into health	92
Gratification of frustrated drives	75
Need to fail	75
Paranoia	92
Unrealistic expectations	67
Fear of mental illness	92
Iatrogenic factors	75
Countertransference	67
Interpersonal factors	75
Reality factors	58
Absence of therapist	92
Cultural factors	100
Physical illness	92
Drugs	92

*Rating within 1 level on the 5-point scale.

TABLE 2
Prevalence of Causes for Patients Leaving Therapy

	Patients (N=30)	
Causes	Number	Percent
Reactive to treatment	26	87
Fear of loss of defenses	14	47
Fear of dependence	12	40
Fear of aggression or sexuality	11	37
Transference	18	60
Decompensation	1	3
Intrinsic factors	17	57
Masochism	8	27
Poor motivation	3	10
Negativism	6	20
Flight into health	2	7
Gratification of frustrated drives	2	7
Need to fail	7	23
Paranoia	2	7
Unrealistic expectations	1	3
Fear of mental illness	1	3
Therapist factors	11	37
Iatrogenic factors	2	7
Countertransference	10	33
Reality factors	12	40
Interpersonal factors	7	23
Reality factors	1	3
Absence of therapist	6	20
Cultural factors	0	
Physical illness	0	
Drugs	0	

items. The second major category, "intrinsic factors," which involved 57% of the dropouts, was based on chronic personality characteristics of the patient that preceded the therapy process. The third category, "therapist factors" (37%), consisted of counter-

TABLE 3
Diagnoses of Patients Leaving Therapy

	Patients (N=30)	
Diagnosis	Number	Percent
Neurosis	0	
Personality disorder	16	53
Obsessive-compulsive	4	13
Hysterical	7	23
Dependent	2	7
Passive aggressive	2	7
Schizoid	1	3
Borderline personality	9	30
Schizophrenia	5	17
Chronic undifferentiated	2	7
Affective type	1	3
Paranoid	1	3
Undefined	1	3

transference and iatrogenicity. The last category, "reality factors" (40%), included interpersonal causes such as family or spouse interference, reality factors such as moving away and loss of money, and therapist absence when it was not iatrogenically based. In regard to this last factor, all absences were within reasonable expectable limits in respect to timing and duration.

In studying table 2, it is important to keep in mind that premature interruption of therapy appeared to be a multidetermined action with a mean of 3 causative factors per case in this group of patients. Transference factors, fear of loss of defenses, fear of dependence, and fear of aggression or sexuality, in that order, were the most common individual causes for dropping out. Transference was listed as one cause of the interruption in 60% of all cases. The most frequent nontreatment-reactive factor was countertransference, which appeared to be a cause for dropping out in one-third of the cases. Other causes listed in at least 20% of the cases were interference from family or spouse, masochism, negativism, fear of success (or need to fail), and complications related to therapist absences. Interestingly, reality factors, which are often offered to rationalize leaving therapy, caused premature interruption in only 1 case.

Table 3 presents diagnostic data on the 30 patients. More than half were diagnosed as having a personality disorder, 30% as borderline personality, and 17% as schizophrenic. None of the 30 dropouts was diagnosed as having a neurosis.

We also analyzed the epidemiological and treatment characteristics of the total dropout group, excluding categories that did not show significance or trends, and the breakdown by causes for leaving therapy.[2] The following can be noted:

Transference, which was the most common cause for leaving therapy, tended to occur among those who were married, Catholic, had more frequent therapy

[2]A table presenting these data is available from Dr. Levinson on request.

sessions, were not schizophrenic, and who approached 50% of their treatment goals. (Goals in all cases included symptom relief as well as restructuring.) Transference was particularly related to leaving therapy when the diagnosis was personality disorder.

More men than women left therapy because of fear of loss of defenses or fear of dependence. However, men who left for fear of loss of defenses were more likely to be married, whereas those who left for fear of dependence tended to be divorced.

There were fewer Jews and more Protestants and Catholics among those who left for fear of dependence. Those who left for this reason tended to be diagnosed as borderline; none were diagnosed as schizophrenic. They also tended to achieve 50% of their goals.

Of the 11 subjects who left for fear of aggression or sexuality, 9 also left because of transference. Therefore, a separate analysis was not done for the category of fear of aggression. However, chi-square analysis indicated that women, Jews, and patients with less ego strength were less likely to drop out for this reason.

Those who left due to intrinsic factors tended to be slightly older and married or divorced, very few were single. More of these patients were diagnosed as borderline and schizophrenic than those who did not leave due to intrinsic factors.

Of those who left because of therapist factors, more tended to be single and few divorced. More were schizophrenic, compared to the total group, and they tended to leave therapy gradually; few left semi-abruptly. These patients tended to approach 50% goal achievement.

The fourth category, reality factors, was not included in the analysis because it played a relatively small role in termination and did not show any particular trends.

Those subjects with greater ego strength were more likely to have a personality disorder than a more severe illness and to have more goal achievement. Greater ego strength was associated with dropping out due to treatment-reactive causes, mainly fear of loss of defenses and fear of aggression or sexuality. This is logical in view of the "stronger" patient having fewer chronic, intrinsic personality traits, inciting less negative therapist response, and constructing a life featuring less destructive family influence and less social and economic instability and impermanence.

Another finding was that the reasons for leaving therapy which involved therapist-patient interaction (transference, dependence, therapist factors) led to achievement of more of the therapy goals than did other reasons for terminating. It appears that involvement with the therapist, even in an aborted therapy, is productive to the well-being of the patient. Perhaps therapists ought not to consider patient termination as much of a failure when the cause of termination is related to their interaction with the patient.

The following vignette concerns termination of therapy due to therapist-patient interaction.

A 29-year-old actor prematurely terminate
ter 8 months of once-per-week sessions. Alth
personality disorder, his main symptom w
anxiety, which was severely restricting his c
an only child; his mother had been very sc
father jealous and threatening. The son rep
and competitiveness toward his father, was v
and was a genital exhibitionist in childhoo
stage he consciously dreaded that a good per
make the women in the audience fall in love

As the treatment progressed the patient
tudes of resentment and submission towa
With the formation of the transference ca
urges, which were attributed to the doctor
supported by explicit dream material. In on
tient intended to kill his teacher, but the o
of the gun and gently dissuaded him.

Premature termination was consensuall
tile and homosexual factors in the transfere
mitting aggression or sexuality in the sessio
implied and provided a second factor in the
other cause was the fear of continuing h
one being repression of homosexual urges
ered but not selected as important were the
lack of money. The latter turned out to be
Decompensation had not yet set in to contri
ruption.

CONCLUSIONS

A vivid profile of the dropout patien
derived because of the variability in s
overlap and multidetermination of cause
stacles were presented by the small num
each cause category and the absence o
ent control group, although matchi
dropped out for a specific reason w
dropped out for all other reasons provi
group comparison. Still, this study re
to struggle with a difficult methodolog
order to alert the therapist to the proba
ment will be aborted, to suggest whic
will cause interruption, and to enable
use preventive measures sooner.

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Sudden Infant Death Syndrome: Impact on Families and a Direction for Change

BY STANLEY E. WEINSTEIN, M.S.W.

Until recently, there were no standardized approaches to the diagnosis of sudden infant death syndrome (SIDS), the leading cause of death in the age group of one week to one year in the United States. This has led to confusion among professionals and hardships for parents and families. The Sudden Infant Death Syndrome Act of 1974 provided funding for the dissemination of information, counseling, and education. The author reviews the scope of the SIDS problem, the ways in which it affects families, and mechanisms by which community resources can be mobilized to assist families.

SUDDEN INFANT DEATH SYNDROME (SIDS), commonly referred to as "crib death," is the leading cause of death in infants between the ages of 1 week and 1 year in the United States. SIDS is the unexpected death of an apparently healthy infant for which thorough postmortem examination fails to find an adequate cause. The syndrome occurs more often in the colder seasons and usually when the infant is sleeping. Accurate statistics are not available for the incidence of SIDS in the United States because, until recently, autopsies were performed in only 50% of the cases and many communities did not list SIDS as an approved category on death certificates. However, it is estimated that between 8,000 and 10,000 infants die each year of SIDS. Diagnostic procedures and official causes of death vary from community to community.

The ambiguous nature of SIDS makes many physicians, clinical investigators, and pathologists uncomfortable. Theories of causation have included enlarged thymus gland, allergy to cow's milk, suffocation from bedding, infection, and immature heart. Some important areas of current research include studies relating to prolonged sleep apnea (1), chronic oxygen deficiency (2), and enzyme abnormalities: [illegible] have yielded promising results. Other [illegible] of the SIDS pattern tend to include [illegible] mothers under 20, preceding colds, [illegible] cidence at 2–4 months of age (3). Upp[illegible] infections have been associated with SI[illegible] infants who die of SIDS have suffered fre[illegible] tions. Only recently have investigators l[illegible] at SIDS as the end result of an interac[illegible] factors, which may not be the same in a[illegible]

Bergman (4) reported that 14% of the [illegible] sample said they had not been told wh[illegible] died, 50% felt that they were not give[illegible] explanation of the cause of death, and 8[illegible] the death was caused by a disease tha[illegible] from the cause listed on the death certif[illegible] of the 74 primary counties reported in th[illegible] considered to have an organized appro[illegible] with problems related to SIDS.

Bergman attempted to assess how co[illegible] knowledged the problem of SIDS and the[illegible] offered. One variable examined was "[illegible] sources." Bergman found that only one [illegible] association out of 48 had an organized [illegible] local health departments were involved [illegible] grams, and no state health department h[illegible] gram. The community resources Berg[illegible] were physicians (usually pediatricians) [illegible] edgeable public health nurses, occasiona[illegible] gist, and, at best, a chapter of a parent [illegible] the International Guild for Infant Surv[illegible] the National SIDS Foundation. In ma[illegible] were no community resources for th[illegible] SIDS. The services offered by the few [illegible] sources that did exist were the provis[illegible] tion and/or counseling. Bergman concl[illegible] lies which did not have a private phys[illegible] apt to receive the worst care and attent[illegible] poorer families in our society have a h[illegible] of SIDS and the least access to inform[illegible] seling available through community res[illegible]

Information about SIDS is necessary f[illegible] involved and for the general public. The[illegible] to have all of the available information [illegible] order to understand the disease as much [illegible] This may have been less of a concern in [illegible] other diseases (e.g., tuberculosis, sm[illegible] monia) were claiming the lives of more c[illegible] however, SIDS is the leading cause of in[illegible]

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accounting for almost one-third of infant deaths after 1 week of age (3).

THE FAMILY'S RESPONSE TO SIDS

Families experiencing a SIDS death generally face multiple problems. The situation may have been worse in the past, but it is still bad today. The parents are often young and may be experiencing the death of a relative for the first time. It is often the first major stress on their marriage. They had waited nine months for the birth and were just sampling the joys and adjustment of parenthood when their infant child died. They ask the logical question—"Why did my baby die?" They look to their physician for answers, but he does not have them. They want to make sense out of nonsense and put the pieces together in a way that will enable them to see a cause and effect. If there is no autopsy, they will wonder about the cause even if it is listed as SIDS, and they will have no knowledge about possible genetic defects. The autopsy is a procedure that says to parents, "We have looked and found no other known cause of death." The cause of death may be reported as pneumonia, particularly if there is no autopsy or if a coroner does not recognize SIDS as a cause of death. This may imply neglect on the part of the parents, and even if others do not say this, the parents certainly consider it themselves. If the parents have a lifestyle that is at variance with that of middle-class America, they may even be accused of child abuse. A number of parents have been charged with child abuse and imprisoned, even while their infant was being buried. It is often not realized that for the age range of one week to one year many more deaths are caused by SIDS than by child abuse. An autopsy can rule out abuse. Parents and families look for a reason—a germ, a defect, or some logical cause. The atmosphere of not knowing the cause generally leads parents to dwell on their own doubts or doubts suggested by others and to blame themselves for doing—or failing to do—something that led to the death. "If I had stayed home, I would have heard him." "If the window wasn't open." "If I had taken him to the doctor sooner." "If I hadn't left him with a babysitter." These statements may reflect the parent's feeling that "If I were a better parent" or "If I hadn't sinned," the child would be alive.

A common denominator of SIDS parents is guilt. The guilt, the grief, and the lack of knowledge together constitute major emotional stress for the parents both as individuals and as a couple. Guilt can exacerbate previous psychiatric conditions and marital problems. The grief and mourning are doubly hard because the parents had no preparation—the infants were viewed as thriving and healthy, not sickly. If the family has other children, what do they tell them? If the sibling shared a bedroom, do the parents wonder if the other child somehow caused the death? Could normal sibling jealousy or "death wishes" confuse the sibling to the point of believing he or she killed the infant (magical thinking)? What about future pregnancies? Could this happen again? How soon should the couple try to have another child and what can they do to prevent a recurrence of this tragedy? There are many concerns and many questions, but where do these families turn? The Bergman study (4) noted that physicians and parent groups were the two main community resources.

In the past the marital pair, the family, and the kinship group were considered the best support systems. The family is still the major support, although in our complex society the family is unable to handle all problems. As our industrialized society moved away from total reliance on the extended family to cope with family stress, formal community care-giving agencies were developed to provide these resources. The death of an infant, with the grief and mourning that follows, generates a crisis for all individuals within a family. It is important to note that this crisis may temporarily induce behavior that appears similar to mental illness, but "a state of crisis is not an illness" (6) and should not be seen as such. "Crises . . . represent mental health turning points or way stations. The balance may come down on one side or the other; there is the opportunity for a healthier development and the danger of a move toward pathology" (7). Some families grow closer as a result of dealing with crisis and others develop major problems. Studies show that before a state of illness, physical or mental, there is often a crisis that upsets a person's or family's previous level of functioning (8). Other studies (9–11) show the later effects of a child's death on siblings and parents. Thus a severe problem caused by the sudden infant death syndrome can affect the health of all family members. Community resources not only serve to help at a time of stress but can also serve a preventive function.

THE SUDDEN INFANT DEATH SYNDROME ACT OF 1974

Parent groups were the first community resource organized to deal with problems related to SIDS. The first such organizations were developed during the early 1960s by SIDS parents to provide information to newly bereaved families, educate the general public, support research, and facilitate change in the way communities respond to the problem. They also began to counsel SIDS families on a number of issues, including their guilt, their other children, the children they may have in the future, and their grief reactions. They attempted to provide these services because there was no other organized community resource available, and they knew that the circumstances surrounding this death were unique, generating many unique problems and requiring a unique kind of counseling. However, community response was slow to change and more families needed service than these groups could accommodate. These groups, along with some interested professionals, took their concerns

about SIDS problems to the United States Congress for hearings in 1972 and 1973. The Bergman study (4) was presented as evidence, and the result of the hearings was the passage of the Sudden Infant Death Syndrome Act of 1974. The act directed the Department of Health, Education, and Welfare to fund a number of SIDS projects around the country through the Bureau of Community Health Services, Maternal and Child Health Services.

These SIDS projects are specialized community resources set up to provide counseling services to families who have lost a child through SIDS, to educate the general public, and to mobilize and educate special groups of people who come in contact with these families. These special groups are defined as police, physicians, funeral directors, emergency room personnel, ambulance drivers, clergymen, and pathologists (12). These are not just special groups; they are also professional caregivers. Thus it is incorrect to say that there were no resources in the communities Bergman studied; rather, there were community resources that were not being used as support systems for families who experienced SIDS. Viewing community resources this way, one can also consider a number of others such as the medical examiner, the family service agency, the school counselor, the psychiatric clinic, and parent groups. All of these community resources are important, and for a given family in a community one cannot predict which resources will be received in the easiest or most meaningful way or will offer the best help. Assuming all community resources were good and available to all families, factors such as the culture of the family and history of the family's association with the particular resource, plus the age and sex and socioeconomic level of the family members, might determine which resources would be utilized.

COMMUNITY RESOURCES

How does one identify, educate, and support community resource systems? One starts by evaluating a particular community to see what resources exist at both state and local levels and in the public and private sectors. First, it is necessary to determine whether services should be built into existing delivery systems or whether new systems should be developed. To elicit effectively the support of other community agencies and professional caretaking groups, it is necessary to understand their view of their role and the way in which they perceive SIDS and the families of SIDS victims. One way to do this is to appoint professional agency representatives as consultants to the SIDS educational and counseling program. For example, in order to change the type of service SIDS families receive in hospital emergency rooms, it would be necessary to develop an educational program tailored to this goal. However, one must first understand the current service and the views of the emergency room staff toward change. An advisory group of representatives from various community hospitals, large and [illegible] and private, would be of great assistance.

Another question that needs answering is who should do the educating. Should the [illegible] DHEW-funded projects do the educating or someone else? Community resources should be examined so that one can understand how [illegible] training or inservice educational programs. The DHEW project staff can do the educating themselves or work toward using existing educational programs. Often it is better to take the latter course through consultation with and education of existing teachers instead of providing outside information is more likely to be accepted if presented by someone from within the system than a perceived outsider with special skills. For example, an educational program run by police academy instructors with SIDS project staff as consultants is better received than a program provided by the SIDS project staff.

ASSISTING A SIDS FAMILY THROUGH THE USE OF COMMUNITY RESOURCES

Listed below are guidelines that are [illegible] portant in dealing with a sudden infant death. The relevance of each of these to particular agencies or individuals will depend to some extent on the specific case and their relationship to the family.

1. Life-saving measures should be performed by those first on the scene (i.e., ambulance attendants or police officers), if for no other reason than to assure the family that every effort was made to save the infant. Parents often look to ambulance attendants or personnel as the people most likely to be able to save their infant. A parent's feeling of guilt may be increased when the professionals do not try.

2. There should be provisions for [illegible] ents to the hospital and for seeing that their infants are looked after. Once at the hospital, parents should be afforded privacy and access to a telephone in order to contact relatives, a clergyman, or their physician, and an emergency room staff member should be assigned as a link between the staff and the parents while efforts are made to revive the infant. Before informed of the death, parents should be given an opportunity to see the infant again should they desire, and the infant should be clothed or covered appropriately.

3. The need for an autopsy within [illegible] be emphasized and results of the autopsy should be shared immediately with the family. The autopsy examination in all communities should be performed so that there is agreement among pathologists on the use of the discrete category of SIDS.

4. Those dealing with the family at these stages should maintain a nonjudgmental attitude, doing their utmost to avoid implications of culpability in the infant's death. Often, [illegible]

must be investigated by the medical examiner's office or the police. In such cases, only factual data should be collected. Possible concerns about suspected child abuse can be clarified by the autopsy. If SIDS is suspected as a cause of death, the family should be referred to available literature and provided with suggestions for counseling.

5. Those who might be sought by the family for counseling, e.g., physicians, clergymen, public health nurses, psychiatric clinics, health departments, community agencies, or funeral directors, should acquaint themselves with the problems faced by such families and should be aware of other community resources to which they can refer the family.

6. Above all, those dealing with death should be concerned with the needs of the living—the family members whose distress could result in prolonged emotional problems, concerns about having additional children, or difficulties with further pregnancies if they are not given appropriate support in their time of crisis (12, 13). These families, including extended family members and siblings, often require specific guidance in such areas as making funeral arrangements. They may also need help with future crisis points, including the anniversary of the birth and death of the infant, the birth of other children, and Mother's Day and Father's Day.

Those assisting SIDS families should also be aware of the informal network of friends, relatives, employees, etc., who make up a large and often significant source of support for family members (14). These informal resources are usually available to families in crisis, and often their existence minimizes the need for long-term support from more formal community resources.

Caplan (7) has stated that "we can never aim at removing all problems from the world . . . we can, however, assist community leaders to arrange facilities so that people who are facing inevitable stress situations may be helped to solve their problems in a healthy way." The families experiencing the stress of sudden infant death syndrome deserve the care, concern, and assistance available through resources in their community.

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Father-Son Incest: Underreported Psychiatric Problem?

BY KATHARINE N. DIXON, M.D., L. EUGENE ARNOLD, M.D., AND KENNETH CALESTRO, M.

Six families are described in which 10 sons were involved incestuously with a natural father (N=4) or step-father (N=2). Father-son incest as a part of the spectrum of child abuse appears to be a more frequent clinical entity than was thought previously.

THERE HAS BEEN increasing public and professional awareness over the past decade of parental sexual exploitation of children as a form of child abuse. The psychiatric literature contains a number of studies describing clinical features and suggesting dynamic formulations associated with heterosexual parent-child incest (1), but there is a significant lack of discussion dealing with homosexual parent-child incest. It is unclear whether this conspicuous dearth of information reflects a very low incidence of homosexual incest, as suggested by some authors (2–4); a tendency to label this behavior pattern as simply homosexual rather than incestuous, as Cory (5) has implied; clinicians' failure to recognize the problem; or a combination of all three factors.

During a period of 4 years, cases involving six families with homosexual incest have come to our attention. We were not looking for incest but, rather, were impressed by the accidental finding of such a large number of homosexual incest cases in our population in view of the lack of attention the phenomenon has received to date in the literature. All of our cases involved father-son incest, and we are aware of no cases of mother-daughter or mother-son incest in the same clinical population.

CASE REPORTS

Case 1

Larry A, 5½ years old and the oldest of two children, was seen in the clinic over a period of 2 years for problems of hyperactivity, aggressive behavior, encopresis, and poor peer relationships. His IQ scores were in the b[illegible] tal retardation range. His mother, a dependen[illegible] woman, had been married and divorced twice [illegible] from each marriage.

After Larry had been in treatment for a yea[illegible] reported spontaneously that his natural father [illegible] put his ding-dong where I go pooh-pooh [illegible] The natural father was known to have been [illegible] sive when Larry was an infant. He reportedly [illegible] blanket and threw him into the crib from a [illegible] episode of sexual molestation probably occu[illegible] father was caring for Larry during Mrs. A's h[illegible] Larry was 5 years old at the time, and Mrs. [illegible] obtained a divorce from the father. The fathe[illegible] of Larry appeared to be more upsetting and di[illegible] mother than to Larry.

Case 2

Doug B, a 6-year-old boy who was the olde[illegible] dren, was hospitalized for evaluation of possi[illegible] by the father. Since Doug had begun talkin[illegible] complained to his mother that the father wa[illegible] genitals and putting his mouth on Doug's [illegible] these incidents were witnessed by the mothe[illegible] and bite marks were sometimes found around [illegible] The mother reported finding bruises on the ch[illegible] claimed resulted from his father striking him. [illegible] in a double bed with his father since infa[illegible] mother slept in a single bed in the same roo[illegible] younger children was molested by the fathe[illegible]

In a clinical interview, Doug was observed [illegible] overactive and became particularly agitate[illegible] tioned about his father. His remarks about th[illegible] ed "He stinks. He's drunk. He hits me. I'd [illegible] and burn down his bar." It was reported that [illegible] feces in a can and often urinated on the living r[illegible]

Mrs. B filed for divorce because of Mr. B's [illegible] ual abuse of the child. Mrs. B had a history [illegible] and psychiatric hospitalization, and custody [illegible] awarded to the father during the divorce proc[illegible] was not seen for follow-up or treatment.

Case 3

Steve C, 11 years old and the oldest of fou[illegible] brought to the outpatient clinic by his mo[illegible] learned that her husband, Steve's stepfathe[illegible] gaging Steve in sexual activity over the pre[illegible] The three daughters, natural children of this [illegible] been sexually abused. Steve's natural fathe[illegible] when Steve was 18 months old, but Steve was [illegible] Mr. C was his stepfather.

Mr. C, an alcoholic, was described as "ove[illegible]" by wife. She said he showed pornographic films [illegible] ions and locked the children out of the hous[illegible]

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intercourse with the babysitter. Mrs. C displayed some hysterical features and claimed that her husband often had intercourse with her while she was asleep. Steve had apparently watched at least one of these episodes on his stepfather's invitation.

During the two-year involvement with his stepfather, Steve had a series of accidents which included two near-drownings, several falls, and being struck by an automobile.

Mrs. C divorced her husband, but Steve did not continue his outpatient contacts with his therapist. He was next seen 3½ years later when he was admitted to the Adolescent Inpatient Service for refusing to attend school, conflict with his mother, defiance, running away, drug abuse, and at least one drug overdose. He was an anxious, insecure, and impulsive adolescent who was concerned about his own sexuality. His intelligence was in the low normal range. In a therapeutic program, Steve made positive gains with his many self-defeating, self-destructive, and maladaptive behaviors and was discharged home to his mother.

Case 4

Joe D, age 13, was the oldest of three children. He was referred for psychiatric treatment by the county children's services agency after discovery of his bisexual incestuous activity in his home. Police raided the family's trailer after receiving information suggesting that the patient's stepfather was moonshining and selling alcohol to minors. During the course of their investigation, they also discovered approximately 300 pornographic photographs depicting various sexual activities between Joe and his 11-year-old sister and his stepfather, and between the stepfather and Joe's sister. Subsequent physical examination of Joe's 3-year-old brother supported the children's later reports that he also had been repeatedly sodomized by Mr. D, who was this child's biological father.

The psychiatric evaluator found Joe to be a plump, immature, effeminate-appearing adolescent. He refused to discuss the home situation other than to say, "My father is rotten." His intelligence was above average (full scale IQ of 118 on the Wechsler Intelligence Scale for Children), and there was no evidence of a psychotic disorder or of significant depression. Results of psychological testing were essentially within normal limits, although there were indications that Joe was passive, submissive, cynical, and confused concerning issues of sexuality, rejecting the stereotypic masculine role. He also expressed overt hostility toward the father figure. Neither of Joe's siblings was referred for psychiatric evaluation. Short-term psychiatric admission was requested by and for Joe as a form of asylum from the notoriety this case had received in his small rural home community. Although the intrafamilial sexual activity had existed for at least 1 year, the patient was performing satisfactorily at school and no other problems were reported at the time of referral.

The patient's mother, who was in her mid-30s at the time of his admission, had suffered a stroke when Joe was 2 years old. Following this episode she showed significant personality and behavioral changes that interfered with her ability to care for herself and her young children. She was deserted by her first husband soon after her stroke and married Mr. D when Joe was 10 years old. She was pregnant with her youngest child at that time. She apparently was not a direct participant in the family's sexual activities with the children, although she was present during the photographing. The mother stated that she was fearful of physical violence from her husband and claimed she could do nothing to stop him. The stepfather, who was in his mid-40s, was described as violently explosive and had a record of convictions for manslaughter, bootlegging, and sale of pornography.

Mrs. D went into hiding for 3 months after the police discovered the family activities, later stating that she feared reprisals from her husband owing to her preliminary testimony. She subsequently refused to testify against Mr. D, as did the children. Their refusal, as well as the fact that the photographs could not be used because they had been seized without a warrant, led to the assault charges against Mr. D being dropped. He was convicted of selling liquor to minors and served a short sentence.

A 2-year follow-up revealed that Mr. and Mrs. D had separated and Mrs. D and her children had moved out of state. Joe's adjustment was reported as good, with no difficulties at school, home, or on his part-time job.

Case 5

David E, a 15-year-old boy, was originally seen for a psychiatric consultation on the surgical service, where he had been admitted for evaluation of headaches and neck pain. David had complained of various somatic problems for the previous 6 months, including chest and abdominal pain. For 3 months he had become progressively more withdrawn and depressed, with anorexia, insomnia, and self-mutilation. David and his mother led the consultant to believe that the depressive symptoms were temporally related to a change from special classes to a regular classroom, where he was unable to keep up with the work.

After several nonproductive sessions in which David remained withdrawn and uncommunicative, emergency psychiatric hospitalization was required for a crisis in which David was cutting his arms and legs and threatening harm to himself and his father. After his admission, with urging from his mother, David divulged the family secret that his father had been sexually abusing all six children in the family (two daughters, ages 13 and 14, and four sons, ages 8, 10, 12, and 15) over a period of 10 years. The youngest son had been treated for rectal incontinence at a pediatric clinic, but apparently no inquiries had been made to determine the cause. The mother claimed she had overt knowledge of this activity for only 1½ years but had taken no action to protect the children. The father's relationship with the children included a clear sadistic component, and he often beat them in combination with his sexual abuse. When the family noted the father to be in a "bad mood," the oldest daughter or son would "offer" herself or himself to protect the younger children.

All of the children in this family had significant school problems and several of them, including David, were in classes for the educable mentally retarded. David's achievement scores as a 9th grade student were on second, third and fourth grade levels. David's IQ, reported as 64 on a Stanford Binet at age 11, rose to 88 after his hospital admission and sharing the family secret.

The mother was able to follow through with divorce proceedings but then allowed the father to return home to live. None of the family appeared in court to testify against the father on the incest charges, which were consequently dropped.

At follow-up 3 years after the initial contact, the father had moved out of the home and remarried, though he continued to live across the street from the family home. He remained involved with the children at least to the extent of intruding into the relationships between his daughters and their boyfriends. All of the children continued to have school and

emotional difficulties. The oldest daughter sought counseling, and David called his former therapist once asking for advice about his impotence with his girlfriend. He was, however, unwilling to become involved in further treatment. None of the children received further help because the mother did not want to "open up old wounds."

Case 6

Richard F, 16 years old, was the second of five children. He requested psychiatric admission for "nervousness," which he attributed to several years of sexual abuse by his father. He had not attended school for the previous year and had spent his time "sitting around the house." In addition to running away frequently, he used alcohol and marijuana excessively. A history of "accidents" since age 12 included a gunshot wound in the chest deliberately inflicted by a friend, burns from "lying on a register," a fall from a roof, and assorted hand and knee injuries. There were numerous other clinical indicators of anxiety and depression.

Richard's parents had an 18-year history of marital and sexual problems. Mrs. F was aware when she married Mr. F that he had been involved homosexually with his brother and her brother. She suspected his sexual involvement with Richard from the time Richard was 10 but never attempted to confirm her suspicions. On a number of occasions she awakened in the middle of the night and heard Mr. F in the son's room, but accepted at face value her husband's explanation that he was "just checking Richard." Father and son were involved homosexually almost nightly from the time Richard was 14, and only when Richard openly confronted his mother 2 years later did she take any action. The charges she filed with the police against her husband were almost immediately withdrawn "at the children's insistence," she claimed.

Mr. F, a fifth grade drop-out, had been unemployed for several years, disabled due to "nerves." He drank excessively and was physically abusive with all his children. He whipped one son with a belt for 20 minutes "because he wouldn't cry." He openly admitted the sexual relationship with his son, blaming it on the poor sexual relationship with his wife.

Mrs. F, an anxious, suspicious, hostile woman, stated that she was unable to leave her husband because she could not support the family herself. Her history indicated that she was able to do little other than complain about the many terrible things her husband had done. She appeared overwhelmed by family and marital conflict but was unable to use the support of a social agency to help her cope with her problems.

Although there were four sons and a daughter in the family, only Richard had been singled out for his father's advances. An older son had a history of impulsiveness, excessive alcohol use, and drug abuse. At age 19, he had been married and on welfare for 2 years. One year after the initial contact, a younger son was seen on an emergency basis in the psychiatry outpatient clinic because of threatened violence and increasing conflict with the father. Fearful of being forced into a homosexual relationship, as Richard had been at his age, he was having some difficulty sleeping and was feeling homicidal toward his father, whom he accused of doing "awful" things.

No significant changes occurred within the family as a result of the family's and sons' psychiatric contacts, and after Richard's second admission he was discharged directly to the Job Corps.

DISCUSSION AND CONCLUSIONS

It is generally agreed that father-daugh... involves more than two people and is an e... intrafamilial conflict and pathology. Som... ingredients conducive to father-daughter ... poor marital relationship, an overburd... adequate mother who has usually abdicat... role, and a father with a poor sexual ad...

Although firm generalizable conclus... factors associated with father-son ince... drawn from these six families, there ar... table patterns that warrant attention ... identified patients were the oldest of mul... However, younger siblings were also ... fathers generally had histories of alcoh... athy, or both. Four fathers were known ... and physically abusive with the children ... had a history of poor judgment and imp... None was known to be homosexually ... other than immediate family or close rel...

There seemed to be a high degree of ... the part of the mothers. In several c... known about the sexual activity, overtly ... for several years before community reco... problem or direct confrontation by the ... them to take some action that could ... vention. They often appeared overwhe... able to protect their children, even with ... some cases.

In father-daughter incest, the daughte... anger and resentment toward both paren... as perpetrator and the mother as nonpro... cases, the sons expressed intense nega... even homicidal wishes, toward the fathe... of the families did the son describe the ... nonprotector.

Homicidal and/or suicidal ideation ... four of the six identified patients. Three ... fied patients had a history of self-dest... some form, ranging from suicidal gestur... tilation to reckless use of drug combina... tiple "accidents." None of the 10 be... families was psychotic.

All six families were white, even tho... population includes a substantial prop... races (approximately 20% black). How... skew could occur by chance in a series ... of the families were in the lower three ... socioeconomic classes, and five of the ... were urban. The clinic serves primarily ... and the socioeconomic distribution of ... normal.

There did not seem to be a consistent ... ther-daughter sexual abuse in the prese... sexual incest. Five families had both m... children. In two of these families, both ... male children were sexually used by the ... three families the sons were the only ta... ther's sexual abuse.

Following public disclosure of the family secret, at least one family member was extruded from the family system, usually the father or son. In one case, the mother was essentially extruded when custody was awarded to the father in a contested divorce. In one case, the disclosure was made after the father had left the home.

In general, there seem to be more parallels between father-son and father-daughter incest than between father-son and mother-son incest. This would suggest that the sex of the parent is a more important variable than the sex of the child.

The clinical experience from which these cases were drawn consisted of approximately 1500 cases of male child and adolescent patients. Thus, these cases represent 0.4% of our male child psychiatry patients. Since our patient population is not atypical in other regards for a child psychiatry service, it seems reasonable to expect that the frequency of father-son incest in the overall child psychiatric clinic population is higher than has been assumed.

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BRIEF COMMUNICATIONS

Effect of Haloperidol and Apomorphine Treatment on Dopamine Receptors in Pituitary and Striatum

BY WILLIAM C. FRIEND, M.SC., GREGORY M. BROWN, M.D., PH.D., GEORGE JAWAHIR, TYRONE LEE, PH.D., AND PHILIP SEEMAN, M.D., PH.D.

Prompted by an interest in the similarity of brain and tuberoinfundibular systems, the authors studied butaclamol-specific neuroleptic and apomorphine binding in pituitary and striatum after chronic haloperidol and acute apomorphine treatment. Striatal binding increases but pituitary binding decreases in haloperidol-treated rats. Pituitary binding changes rapidly in response to apomorphine exposure and striatal binding does not. These findings suggest that factors influencing binding differ in these two tissues.

CONSIDERABLE INDIRECT EVIDENCE indicates that neuroleptic drugs act by blocking dopamine receptors. For example, the molecular structure of chlorpromazine, in its preferred conformation, can be partially superimposed on the dopamine molecule and so might act by fitting into the dopamine receptor (1). Neuroleptics produce the parkinsonian symptoms of rigidity, tremor, and akinesia (2), which can be partially reversed by L-dopa treatment (3, 4). Neuroleptics block the effects of apomorphine (5) and amphetamine (6), accelerate dopamine turnover (6, 7), increase the firing rate of dopamine neurons (8), and increase homovanillic acid accumulation (9). Further, t[illegible] a[illegible] fective inhibitors of dopamine-sensitive a[illegible] clase (10, 11). The recent finding that [illegible] compete with dopamine for stereospecific [illegible] in membrane fractions in vitro and that th[illegible] compete can be correlated with their cli[illegible] to control schizophrenia provides direct [illegible] the dopamine receptor-blocking action of [illegible] (12–14).

During the past decade, it has become [illegible] dopamine plays a major inhibitory role in [illegible] amic control of prolactin secretion. Dop[illegible] act by stimulating the release of some as[illegible] tified hypothalamic prolactin-inhibiting f[illegible] then travels to the pituitary via the porta[illegible] ternatively, dopamine itself could act di[illegible] pituitary as a prolactin-inhibiting factor. F[illegible] cal and physiological evidence exist to sup[illegible] ond hypothesis. Tuberoinfundibular do[illegible] rons terminate in the median eminence be[illegible] tal vessels, and although these neurons ar[illegible] nerve terminals which possibly contain [illegible] regulatory factors (15), the basal hypoth[illegible] tains few, if any, dopamine receptors tha[illegible] volved in triggering the release of a non[illegible] lactin-inhibiting factor. However, pituitary [illegible] contain dopamine receptors (16). Ph[illegible] dopamine directly inhibits prolactin secret[illegible] tuitary glands incubated in vitro (17, 18): [illegible] extracts also inhibit prolactin secretion [illegible] their inhibitory effect can be totally acco[illegible] the presence of dopamine and norepine[illegible] extract (19). Finally, neuroleptic treatm[illegible] an increased prolactin secretion, proba[illegible] blockade of pituitary dopamine receptors [illegible] hibition of prolactin secretion by dopa[illegible] does not occur if haloperidol is added to[illegible] (20).

The tuberoinfundibular-pituitary dopam[illegible] syste[illegible]

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might serve as a convenient model of brain dopamine systems such as the nigrostriatal and mesolimbic systems. For example, the measurement of pituitary hormones, which can now be done with relative ease, might provide an index of the extent of central dopamine blockade produced by neuroleptics. As a test of whether the tuberoinfundibular-pituitary system could be used in this way, it seemed appropriate to compare the response of dopamine receptors in pituitary and striatum to chronic neuroleptic blockade and short-term apomorphine exposure.

METHOD

Male rats were maintained on a graduated dosage of haloperidol, beginning with 0.32 mg/kg twice daily for 4 days. This was followed with 0.64 mg/kg twice daily for 4 days, and 1.25 mg/kg twice daily for 4 days. Finally, the animals received 2.5 mg/kg twice daily for 8 days. A second (control) group was maintained on the vehicle of tartaric acid in water. All injections were intraperitoneal and the total drugging period was 20 days. On the 7th day after the last injection, animals were injected with 0.08 mg/kg of apomorphine, 0.32 mg/kg of apomorphine, or a vehicle of 0.1% sodium meta-bisulfite (an antioxidant) in water. They were killed by decapitation 30 minutes later, and pituitaries were immediately removed and frozen on dry ice. Brains were also removed and cooled on ice. Later, at the completion of the procedure (about 2 hours), striata were removed and frozen at −20° C.

The dopamine receptor assay was carried out according to the method of Seeman and associates (12, 21). This assay measures the binding of tritiated haloperidol or apomorphine to membranes and uses the positive (active) enantiomer of butaclamol to define nonspecific binding. The specific component of binding can be calculated as that amount of tritiated ligand bound in the absence of (+)-butaclamol minus that bound in the presence of (+)-butaclamol. For homogenization and assay, striatal and pituitary tissue were pooled within groups. The results are stated as femtomoles per milligram of protein. For statistical analysis, we used a two-way analysis of variance and Student's t test.

RESULTS

The results of [^{3}H]haloperidol binding are shown in table 1. Chronic haloperidol administration caused an 18.3% increase in [^{3}H]haloperidol binding in striatum ($p=.003$). However, a 54.8% decrease in binding occurred in pituitary ($p<.001$). Exposure to apomorphine 30 minutes prior to decapitation did not significantly alter binding in striata of either normal or haloperidol-pretreated rats. However, in pituitary an interaction effect appeared; that is, the influence of apomorphine on receptor number was altered significantly by pretreatment with haloperidol. Apparently in pituitary, short-term apomorphine exposure causes a dose-dependent decrease in binding in normal rats but a dose-dependent increase in rats chronically maintained on haloperidol ($p=.004$).

TABLE 1
Specific [^{3}H]Haloperidol Binding in Rat Pituitary and Striatum

Experimental Conditions	[^{3}H]Haloperidol Binding (fmol/mg protein)			
	Rat Striatal Tissue*		Rat Pituitary Tissue**	
	Mean	SE	Mean	SE
No haloperidol				
No apomorphine	217.95	17.75	47.41	0.54
Apomorphine				
0.08 mg/kg	236.66	9.57	30.69	11.43
0.32 mg/kg	256.81	13.97	24.92	2.48
Haloperidol				
No apomorphine	292.39	6.97	21.42	0.61
Apomorphine				
0.08 mg/kg	280.01	16.69	38.16	6.31
0.32 mg/kg	268.86	17.91	46.94	4.42

*Each experimental group represents 9–12 determinations made on approximately 7 animals.
**Each experimental group represents 3 determinations made on approximately 14 animals.

Animals treated chronically with haloperidol showed a 20.6% increase in butaclamol-specific [^{3}H]apomorphine binding in striatum (14.99±0.98 versus 18.08±1.13 fmol/mg of protein, $p<.05$). However, binding decreased in pituitary by 42% (10.68±0.75 versus 6.19±0.15 fmol/mg of protein, $p<.05$). In striatum both experimental groups represent 23 determinations each from 13 animals. In pituitary both experimental groups represent 2 determinations each from 9 animals.

DISCUSSION

The finding that chronic haloperidol treatment will produce an increase in specific [^{3}H]haloperidol binding in striatum is not new (22). Burt and associates (23) have reported that not only haloperidol but other antischizophrenic drugs such as fluphenazine and reserpine can increase striatal [^{3}H]haloperidol binding, apparently by an increase in receptor number rather than an increase in receptor-ligand affinity. The increase in specific striatal binding with chronic haloperidol treatment provides an explanation of the supersensitivity to apomorphine observable in rats following chronic neuroleptic exposure (24) and the development of striatal dopaminergic hyperactivity, seen clinically as choreiform movements, during chronic neuroleptic therapy. However, the receptor regulatory factors in pituitary tissue seem to be different from those in striatal tissue, since the response of [^{3}H]haloperidol binding in pituitary tissue to haloperidol exposure is opposite from that in striatal tissue. In the present study, we did not determine whether the observed changes in binding are a result of changes in receptor

number or avidity. Differences between the tuberoinfundibular-pituitary dopamine system and the striatal system have been observed previously. Schmidt and Hill (25) were able to detect dopamine-sensitive adenylate cyclase in striatal but not pituitary tissue. Also, haloperidol increases dopamine turnover in the corpus striatum but not in the median eminence, indicating that regulatory mechanisms in the dopaminergic neurons differ in the nigrostriatal and tuberoinfundibular systems (26).

Creese and associates (27) have suggested that the dopamine receptor exists in either an agonist or antagonist state. Agonists of dopamine would display a higher affinity for the agonist state receptors, and antagonists of dopamine would display a higher affinity for antagonist state receptors. For this reason we were interested to see if the receptor-binding changes measurable with [^{3}H]haloperidol were a reflection of an entire pool of dopamine receptors or just those in the antagonist state. Since we observed similar changes using either the dopamine antagonist, [^{3}H]haloperidol, or the dopamine agonist, [^{3}H]apomorphine, we concluded that the alteration in binding after long-term haloperidol administration involves both agonist and antagonist state receptors.

The prompt changes in pituitary [^{3}H]haloperidol binding observed with brief exposure to apomorphine indicates that pituitary tissue is capable of rapidly modifying either the number or affinity of dopamine receptors. Furthermore, the history of the pituitary is critical in determining the direction in which the receptors will be modified in response to a stimulus. In contrast, receptors in the striatum do not seem to be acutely modifiable by apomorphine.

It appears that pituitary dopamine receptors differ from striatal dopamine receptors, and therefore the tuberoinfundibular-pituitary dopamine system may be a poor model for the nigrostriatal system. Whether there are major differences between the mesolimbic dopamine receptor, which is probably involved in schizophrenia (28), and the tuberoinfundibular-pituitary dopamine receptor remains to be determined.

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Psychiatric and Neurologic Sequelae of Infectious Mononucleosis

BY NELSON HENDLER, M.D., M.S., AND WILLIAM LEAHY, M.D.

Infectious mononucleosis is usually thought to be a benign disease with occasional neurologic sequelae. Depression, incoordination, a reduction in intellectual ability, and altered EEG patterns were found in two patients; one recovered and the other seemed to have permanent residual effects. The possibility of tranylcypromine as a treatment for the depression and appropriate counselling of patient and family are discussed.

INFECTIOUS MONONUCLEOSIS (IMN), considered to be a disease of adolescence, presents as a febrile illness associated with lymphadenopathy, hepatosplenomegaly, pharyngitis, malaise, and myriad other complaints. The yearly incidence for this normally benign disease is 38 per 100,000, and 1 per 3,000 of these patients dies, usually as a result of neurologic complications (1). Nonfatal central nervous system involvement was first reported in 1931 (2), and reviews since then have reported a wide spectrum of neurologic dysfunction including meningoencephalitis (3, 4), encephalitis (5–7), the Guillain-Barre syndrome (8, 9), and mononeuritis (10). Schnell and associates (11) found one case in which neurologic findings were the only presenting symptom of IMN, and Bonforte (12) has reported convulsions as a presenting sign. Occasionally, personality changes such as irritability, emotionality, asocial behavior, and destructiveness have been described (13). Rapid and complete recovery from such involvement has been the rule; however, Cadie and associates (14) found that 13 of 20 women suffered depression and anxiety for as long as 1 year after IMN. We found prolonged neurologic and psychiatric consequences in the following two cases, one with confirmed and the other with presumptive sequelae of IMN.

CASE REPORTS

Case 1. John, a 14-year-old male, reported an inability to concentrate, a loss of athletic ability, a feeling of depression, irritability, and suicidal ideation for 5 months, ever since an attack of IMN, documented by a positive mono spot test, lymphadenopathy, splenomegaly and tenderness, general malaise, leukopenia, and fatigue. Before contracting IMN, he was well adjusted socially, and his only major medical illness was thyroiditis 4 years earlier.

John's father is a prominent physician, and his mother is a housewife and part-time nurse. His mother had hypothyroidism, and a maternal aunt had a 1-year depressive episode. His developmental history was normal, with an absence of both febrile convulsions and dyslexia. John's mental status examination showed that he had no difficulty with concentration, memory, or recall. The neurologic examination was normal. An EEG revealed diffuse slow activity (2–7 cps) and diffuse low voltage, fast activity (15–25 cps). At the start of therapy, his verbal IQ was 145, but his performance IQ was 112.

Initially, John was placed on imipramine, 150 mg h.s., and sodium liothyronine (Cytomel), 25 mg every morning for 1 month; no improvement was noted. Imipramine was discontinued, and he was then placed on tranylcypromine, 20 mg b.i.d., which improved his concentration and eradicated his depression. After 2 months, John wanted to discontinue medication. Within 1 week, he was again depressed, suicidal, and had difficulty concentrating. The medication was reinstituted, and within 2 weeks John felt better. He was maintained on tranylcypromine, 15 mg b.i.d., for an additional 2 months.

In therapy, John talked about the guilt associated with his poor school performance, his anger about the disability created by the IMN, and his frustration at the inability to control his concentration and motor skills. Repeated psychological testing 9 months after the initial visit resulted in a verbal IQ of 147 and a performance IQ of 131. This marked improvement in perceptual motor tasks, paralleled by a change in school performance from a D level to his previous honor role status, was accompanied by a normal EEG. There were no residual signs of depression, and his motor skills at the end of treatment had returned to his premorbid level.

Case 2. Jane, a 16-year-old single high school student, presented with the chief complaint of "I want to die and life is not worth living." She was admitted to an inpatient psychiatric unit after a suicide gesture. Her psychiatric disturbance began when Jane contracted mononucleosis at age 9. She was out of school for 5 months and failed a grade after she returned. The IMN precipitated reduced concentration, ability to perform calculations, and general difficulty with school work. She also reported that her fine motor coordination was greatly impaired after the disease.

Jane attributed her difficulty at home to the fact that both her mother, a dance teacher, and her father, a prominent lawyer, were pressuring her to do well in school. She felt guilty because she could not live up to their expectations. Her psychiatric and developmental histories were unremark-

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able, and she had been a well-adjusted child until the onset of IMN.

The pediatrician who diagnosed IMN documented the disease by obtaining a heterophile titer, which was 1/320 initially, progressed to 1/1792 10 days later, and was accompanied by lymphadenopathy, hepatosplenomegaly and tenderness, SGOT of 148 IU, and lymphocytosis. There is no family history of thyroid disease, diabetes, alcoholism, or mental illness. On physical examination, there was some questionable asymmetry of pupil size, the left side being slightly (1 mm) larger than the right, minimal dysdiadochokinesis, minimal ataxia, and some difficulty with tandem walking. She had a fine, bilateral intention tremor, and mildly increased deep tendon reflexes on the left. EEG showed a great deal of diffuse low voltage activity (15–25 cps) and considerable amounts of diffuse low to medium voltage activity (2–7 cps). Echo results were suggestive of an enlarged right lateral ventricle, and cerebral mantel thickness measured 2.6 cm on the right and 3.4 cm on the left. The mental status examination revealed that her concentration, memory, and recall were intact. She admitted to feeling depressed but denied any vegetative signs of depression. She had a verbal IQ of 104 and a performance IQ of 104. Family therapy centered around convincing her parents that their expectations for Jane were somewhat unrealistic in light of her intellectual and motor functioning, which probably resulted from the IMN and not from rebellion. Once the parents recognized that their hopes for Jane might have been in excess of her abilities and eased their pressure on her, Jane reported feeling more relaxed and less depressed. Three years later she finished high school and is now living comfortably with her husband.

DISCUSSION

There are some similarities between these two cases that are worthy of recognition. Both children had good premorbid adjustment, good grades in school, and supportive family situations. Both patients felt that their psychiatric difficulties began after they contracted IMN, and both reported difficulty with concentration, fine motor activity, and with understanding what had wrought the changes in their mental functioning, physical skills, and personality. Both had reported depression after their bouts with IMN. Both suffered dramatic changes in their school work, which compounded their depression. Both came from upper-middle-class, achieving families and regarded impaired intellectual functioning as disastrous. Both had minimal, if any, significant localizing neurologic signs, and both had 2–7 cps diffuse low to medium voltage activity and 15–25 cps diffuse low voltage fast activity on their EEG. Both had reduced IQ scores, although one score was transient and the other could have been due to other causes.

Viral diseases (such as cytomegalovirus) may produce a wide spectrum of psychiatric disturbances, varying in both intensity and duration (15). Anxiety and guilt reactions are most commonly appreciated (13); more devastating stages range from confusion, delirium, and affective psychoses to the more profound "toxic organic" psychoses associated with severe mental disturbance and coma (16). Lipow[illegible] 17) m. Imboden (18) have advanced the notion [illegible] psychological variables determine the length of re[illegible] course of IMN. Slow recovery has been associate[illegible] with g scores of ego strength (19), and since the [illegible] closed environment is also changed by an infect[illegible] psychosc "there is a tendency for the observed symp[illegible] to be strongly tied to the premorbid perso[illegible] ture upon which the disease impinges" ([illegible] 20 noted that some conduct disorders occu[illegible] are similar to minimal brain dysfunction, and [illegible] 21 and Cadie and associates (14) reported [illegible] experienced anxiety and depression after [illegible] and associates (14) also noted an increas[illegible] symptoms after IMN. This increase in [illegible] pression, and somatic concern is not unlik[illegible] seen in most people with a chronic illne[illegible] 2[illegible].

Neurologic sequelae of IMN have been [illegible] sively described. EEG abnormalities ha[illegible] ported in acute cases of IMN (11) and s[illegible] range (4–7 cps) activity in either paroxys[illegible] continuous patterns. However, several of [illegible] ported had diffuse activity ranging from 1-[illegible] the early phase of the illness (days 1 to 2[illegible] our patients, theta activity (2–7 cps) wa[illegible] both patients also showed diffuse low vo[illegible] (15–25 cps), as well as unstable posterior [illegible] um voltage (8–10 cps) alpha rhythm; how[illegible] the EEGs were obtained after the acute p[illegible] Localizing, unilateral neurologic findings [illegible] reported, manifesting as weakness a[illegible] clumsiness in the use of hand and foot (2[illegible] eral Gordon and Chaddock reflexes (24) [illegible] of ataxia, slurred speech, decreased abili[illegible] trate (25), and disorientation (7) have also [illegible] ed. Silverstein and associates (26) report[illegible] neurologic complications are the first a[illegible] signs of IMN, some of which, like metam[illegible] "Alice-in-Wonderland" syndrome, may [illegible] as drug-induced states or psychiatric d[illegible]

Some authors (3, 26) have report[illegible] volvement as a heralding feature of the [illegible] others (10) find that CNS involvement [illegible] the acute illness and note that the neuro[illegible] ten may predominate or obscure the usu[illegible] systemic symptoms. The incidence of ne[illegible] function in IMN varies, but most author[illegible] mately a 1% rate. One-year follow-up in [illegible] hensive series (1, 26, 28) showed rather r[illegible] plete recovery from IMN. However, the [illegible] have presented reveal that persistent ne[illegible] function, as well as mental aberrations, m[illegible] though a reactive depression in respons[illegible] likely consideration, an organically [illegible] ponent, secondary to CNS involvement, [illegible] bility. Intellectual, depressive, and mot[illegible] ities should not be dismissed on the bas[illegible] reactive and/or volitional component (co[illegible] conscious).

Understanding that family as well a[illegible]

pectations may need to be altered as a result of CNS involvement secondary to IMN may significantly help the patient to accept difficulties that he or she experiences. Unrealistic expectations, or assigning psychological interpretations to a physiological response, can create undue stress on the patient. Reassurance of patient and family are necessary to ensure a successful treatment for the depression (no matter what its etiology) associated with IMN. The monoamine oxidase inhibitor tranylcypromine may also be useful in the treatment of post-IMN depression, in part because of its amphetamine-like structure, although its mechanism of action is due to presynaptic accumulation of norepinephrine. This suggests that the CNS involvement after IMN is similar to minimal brain dysfunction, since both conditions respond to amphetamine-like substances, are associated with difficulty in school, and may be secondary to viral diseases that affect the CNS.

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A National Survey of Resident Assessment Methods

BY WARREN PROCCI, M.D., CLAUDE FRIEDMANN, M.D., GILLA PRIZANT, M.D., AND SHERWYN M. WOODS, M.D.

The authors distributed a questionnaire regarding the assessment of psychiatric residents to 130 directors of residency training programs. Formal examinations were required by 37% of the programs and were being considered by an additional 49%, suggesting that formal examinations may be an integral part of residency training programs within five years. The authors believe that this trend toward utilization of objective measures reflects a growing national concern with accountability in medicine.

ONE OF THE notable developments in medicine during the 1970s is the emphasis on accountability. Psychiatry is not exempt from it (1–3), witness the recent decision by the American Psychiatric Association (4) to require evidence of continuing education for maintenance of membership. Similarly, many states now require such education for maintenance of licensure. The American Board of Psychiatry and Neurology, Inc., (ABPN) is currently studying the feasibility of recertification procedures for psychiatrists, and the Group for the Advancement of Psychiatry (GAP) (5) has suggested that Part I of the ABPN examination be given before graduation from residency programs.

It is organized psychiatry's responsibility to insure quality control, not only of practitioners, but also of residents in training. Although such formal self-scrutiny has not previously been characteristic of the medical profession, it has now become a necessity (6). It has been pointed out (7) that the training of competent psychiatric clinicians is both a matter of public concern and the primary responsibility of the psychiatric educational community. This goal is achieved in part by the assessment of the educational quality of residency programs, which is essential because it helps to assure requisite educational opportunities for becoming a qualified specialist. However, the presence of adequate educational opportunities in no way ensures that individual residents will use them. An important question then is to determine the means by which programs decide whether residents have acquired necessary skills. We undertook this study to [illegible] following two questions: 1) What me[illegible] programs currently use to determine a[illegible] formance? and 2) What changes, if any[illegible] anticipate making in the next five years?

METHOD

A 12-item questionnaire was distribute[illegible] bers attending the 1976 Annual Meeti[illegible] can Association of Directors of Psychi[illegible] Training (AADPRT).[1] A follow-up m[illegible] responders and members not in atten[illegible] ducted two months later.

Each program director was asked to [illegible] lowing: program name, geographic loca[illegible] cility, number of residents, number of f[illegible] ment methods currently used, weight [illegible] method, any exams used and their n[illegible] sequences, persons involved in plann[illegible] methods, and any contemplated change[illegible]

RESULTS

Responses were obtained from 1[illegible] 176 residency programs listed in the A[illegible] bership. Response rates did not differ a[illegible] geographic areas (South, West, North[illegible] east, Mid-Atlantic). Not all respon[illegible] each item in the questionnaire, so the [illegible] item does not necessarily equal 130. T[illegible] residents in the responding programs r[illegible] the case of several programs in planni[illegible] The number of full-time faculty in [illegible] ranged from 1 in a planned program t[illegible] in several large programs. Ninety-one [illegible] identified as primarily university progr[illegible] 39 programs, which were affiliated w[illegible] vate, and military hospitals, were consi[illegible] as nonuniversity programs.

Table 1 lists the evaluative methods [illegible] chiatric residency programs, the numbe[illegible] age of programs that used each, and th[illegible] ranks assigned by the training directors [illegible] ods they considered most important. Ta[illegible]

Dr. Procci is Assistant Professor, Dr. Friedmann is a resident, Dr. Prizant is an intern, and Dr. Woods is Professor and Director of Graduate Education, Department of Psychiatry, University of Southern California School of Medicine, LAC/USC Medical Center, 1934 Hospital Pl., Los Angeles, Calif. 90033. Address reprint requests to Dr. Procci.

[1]A copy of the questionnaire is available f[illegible] request.

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TABLE 1
Evaluative Methods in Psychiatric Residency Training Programs

Evaluative Method	Residency Programs (N=130) Number	Percent	Mean Importance Ranking*
Written evaluation			
By clinical supervisor	119	92	4.22
By unit chief	99	76	3.64
By seminar leaders	56	43	2.62
Self-evaluation by resident	37	28	2.75
Written exam			
Year-end	27	21	2.83
At end of individual courses, seminars, rotations	24	18	2.41
Oral exam			
Year-end	18	14	2.75
At end of individual courses, seminars, rotations	13	10	2.76

*Derived from training directors' ranking of the 5 evaluative methods they considered most important; 5=most important.

TABLE 2
Type and Use of Examinations in Psychiatric Residency Programs

Characteristic	Residency Programs (N=130) Number	Percent
Type of examination		
Involuntary, nonanonymous	33	25
Involuntary, anonymous	15	12
Voluntary, nonanonymous	10	8
Voluntary, anonymous	18	14
Use of exam		
Evaluation of resident progress	43	33
Consequences of failure		
Remedial work	43	33
Drop from program	12	9
Repeat of year	8	6

that 48 programs (37%) required formal examinations; it also shows how many programs allowed anonymity on exams and how they used exam results.

Some definite future change in the assessment process was contemplated by 101 of the programs (78%); the changes planned are summarized in table 3. Since some programs had multifocused designs, the total number of items in the table exceeds the number of respondents.

University and nonuniversity programs were compared on the following items: techniques of assessment used, use of techniques to evaluate progress, changes planned in assessment techniques, personnel involved in planning resident assessment, whether exams were voluntary, whether anonymity was allowed on examinations, and the use of a variety of examination modalities (e.g., direct observation of resident interviews, APA Self-Assessment Examination, etc.). There were no significant differences between university and nonuniversity programs.

Similar analyses were conducted for the various geographic regions and for program size. No consistently significant patterns were noted.

TABLE 3
Changes in Assessment Contemplated by Psychiatric Residency Programs

Proposed Assessment Method	Number of Programs
Written evaluation by supervisor	10
Self-evaluation by resident	7
Examination	
Oral	12
Written	18
APA Self-Assessment	22
Mini-boards	6
Unspecified	6
Subtotal	64
Use of videotaping	19
Unsure	14
Other	26

DISCUSSION

This study indicates that assessment practices in psychiatric residencies are relatively uniform across program type, size, and location. Written evaluations by clinical supervisors and unit chiefs, relatively subjective forms of evaluation, are emphasized currently. Formal written or oral examinations, which are considered more objective measures, were used much less frequently, but many programs were planning to implement such techniques. If these plans actualize, we predict that within the near future such exams will be an integral part of the assessment procedure in virtually every program. We can speculate that this trend toward increased utilization of objective measures is at least in part a response to the growing concern with accountability (5, 8).

A quarter of all programs responding (N=33) gave involuntary, nonanonymous examinations that were used in evaluating resident progress. The consequence of poor performance on such examinations was most commonly remedial work, but in some programs poor examination performance resulted in repetition of a training year or even termination from residency. Since the number of programs using examinations is likely to increase in the near future, an increasing number of residents may find such accountability awaiting them during their years in training.

It is interesting to note that 25% (N=33) of the programs polled indicated that they were currently using or planning to use the APA Self-Assessment Examination as an evaluative method. This indicates that with the encouragement of a professional organization new and innovative assessment methods can be made available and acceptable to a large portion of its membership. Such leadership can provide impetus for the profession as a whole in the "interwoven issues of lifetime education, government supervision, and the constant updating of knowledge" (9).

With any such shift in emphasis, there is always a possibility of overemphasis, especially if the presumably quantifiable and objective results of these examinations are seen as a path of least resistance for as-

suaging the need for accountability. At best, a formal written examination measures a certain cognitive data base but in no way measures a variety of other skills, such as the ability to conduct an interview, elicit pertinent data, and formulate a diagnosis based on those data. These skills are obviously also important for a psychiatric trainee to possess. Similarly, other forms of examination have limitations (10).

Another caveat of giving examinations is that overemphasis on apparently objective measures, especially if other methods are ignored, may result in a uniformity of product and a stifling of creativity. Also, it is very difficult to correlate examination performance with performance in actual clinical practice (11–13). Therefore, we would urge programs to consider the use of a wide variety of assessment methods that would emphasize a more global approach to the evaluation of resident skills (14).

The importance of clarifying educational goals for residents early in their training has been stressed (11, 15). Examinations without clarification of objectives may well lead to their being viewed as threatening rather than as a measure of mastery. Although the establishment of goals and objectives is a worthwhile endeavor that has long been neglected, this process does have limitations. Difficulties may arise if the resident views such goals as comprehensive rather than as guidelines. Of course, it would be unrealistic to assume that the educational process is complete when specified objectives are attained.

Change is often met by resistance, and the degree of resistance is directly related to the distance between those experiencing the change and those establishing it. A majority of the programs polled involved residents in assessment planning (N=85) but a sizeable number did not. Smooth implementation of new evaluative methods in residency training programs is likely to occur if there is meaningful participatio[illegible] by the residents involved. We hope that [illegible] pation during residency will provide the [illegible] tive involvement in professional accoun[illegible] one's entire career. Lest psychiatry beco[illegible] with government and lay regulatory [illegible] default (7, 8), such an active role is oblig[illegible]

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The Children's Psychiatric Hospital Unit in the Community: I. Concept and Development

BY BARTON J. BLINDER, M.D., WILLIAM M. YOUNG, M.D., KENNETH R. FINEMAN, PH.D., AND STEPHEN J. MILLER, M.A.

The authors describe a children's mental health unit (CMHU) in a general hospital and cite the need for short- and intermediate-term children's psychiatric facilities within the community. They point out the special need for psychiatric inpatient units for the 3–13 age group and note that in their CMHU the average length of stay and patient turnover ratio compare favorably with adolescent units, which they attribute to the emphasis on diagnosis and treatment rather than long-term rehabilitation.

RECENT STUDIES (1–5) point to the increasing need for short- and intermediate-term children's psychiatric hospital units in the community. Von Mering and Carpenter, as cited in the 1970 report of the Joint Commission on Mental Health of Children (2), emphasized the need for timely, specialized community child mental health services that include centers for the treatment of autistic children, therapeutic nurseries for the evaluation of complex developmental problems, and centers for more comprehensive diagnosis and treatment of atypical ego development in children.

The lack of comprehensive child mental health services leads to delayed detection of more serious deviations in child development, which results in complex adverse consequences for the emerging individual and for society. Often families experience ambiguity in the diagnostic efforts of unintegrated services, which interferes with effective intervention (5).

Portions of this paper were presented at the 26th annual meeting of the American Association of Psychiatric Services for Children, New York, N.Y., Nov. 20–24, 1974, and at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

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Despite efforts by individual agencies and clinicians, few children identified as needing mental health services receive them. Von Mering and Carpenter (2) estimate that, of the 93 million young people (under age 24) in the United States, 1 to 3 million are seriously disturbed and identified as mentally ill or impaired. An earlier study (6) of inpatient treatment reported that facilities exist for approximately 7% of youths who require treatment.

THE CHILDREN'S MENTAL HEALTH UNIT

Location

The purpose of this article is to define a children's psychiatric inpatient unit in a community general hospital that provides easily accessible diagnosis and treatment of children without resorting to a more formal psychiatric institution. Our model is the Children's Mental Health Unit (CMHU) at Huntington Intercommunity Hospital in Huntington Beach, Calif., founded in 1973. The extreme pressure of population growth, the high youth-dependency ratio, the lack of coordinated diagnostic and therapeutic services for children, and the reasonable availability of funding sources render this area a laboratory for innovations.

Staff

The attending psychiatric staff at CMHU consists of 5 child psychiatrists who provide the administrative direction and medical supervision of all admissions to the unit. The CMHU team is comprised of a child psychiatrist, consultant pediatric neurologist, pediatrician, clinical and educational psychologist, special education staff, art therapist, consultant speech and language pathologist, psychiatric social worker, and nursing staff. Members of the multidisciplinary team collaborate in formulating a treatment plan appropriate to the physical and emotional needs of each child.

Indications for Admission

The CMHU offers a range of treatment programs. For example, a short-term stay of 1 week might be appropriate when a family disaster results in a posttraumatic reaction in the child. Crisis intervention may be required for child and adolescent suicide attempts or episodes of antisocial acting-out. A longer period of hospitalization (3–6 weeks) might be appropriate for

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children who require careful diagnostic evaluation and some longitudinal observation; this category includes borderline psychotic children, children with seizure disorders and secondary behavioral difficulties, and children with cerebral dysfunction that requires careful delineation and control. Longer term admission for 3 months might be appropriate for stabilization and more intensive treatment of the acutely psychotic child. Reevaluation and assessment of the chronically psychotic or autistic child might be required to establish maximum educational milieu intervention in addition to efforts for appropriate placement. Reconstructive milieu treatment would be useful in helping to resolve a period of acute, severe preadolescent turmoil or in interrupting a deteriorating child-parent relationship.

These situations are a sample of disorders for which an inpatient unit for children within a community general hospital can serve usefully and appropriately. Other conditions that represent potential admissions are early detection of developmental deviation and developmental delay in the first 5 years of life (these admissions might be coordinated with concomitant day care therapeutic nursery programs); complex juvenile diabetes; and such entities as juvenile colitis, rheumatoid arthritis, asthma, obesity, and anorexia, or more rare entities, such as compulsive tic and dysautonomia. If the CMHU were located in a large children's hospital, the incidence of psychophysiological disorders would probably be higher.

Characteristics and Disposition of Patients

Of 117 patients admitted to the Huntington CMHU between April 1973 and April 1975, 91 (78%) were boys and 26 (22%) were girls. In addition, 7 boys were readmitted. The average length of stay was 51 days (range 4–225 days), and the mean age was 9.7 years (see table 1). Patients with discharge diagnoses of personality disorder, developmental disorder, and reactive or crisis disorder accounted for 65% of the admissions[1] (see table 2). The relatively small number of psychotic disorders (10%) may be representative of the incidence of psychosis among referred children in the community at large. The availability of alternative diagnostic and treatment facilities in the area (table 3) has limited the admission of children with primary mental retardation.

Table 3 refers to the discharge disposition of patients at CMHU. Seventy-two percent returned home. Another 10% were placed in foster or group homes, and 7% entered programs in residential [illegible] ities. The CMHU offers short- and inte[illegible] intervention in a setting close to the hom[illegible] criteria for community psychiatric servi[illegible] immediate intervention, and discharge [illegible] setting).

[1]In the "personality disorder" category the largest number of children fit into the "unsocialized aggressive" category. "Developmental disorder" refers to developmental hyperactivity and variants of developmental delays and deviations, including atypical ego development. "Reactive or crisis disorder" includes children who were from pathological family environments (involving deprivation, abuse, and difficulties in limit-setting) or were undergoing reactions to sudden disruptions, such as the death of a parent or sibling, physical illness, or moving. In the "reactive or crisis disorder" category we saw 1 terminal patient who was admitted postoperatively with craniopharyngioma; he was admitted because of severe hyperphagia and a reactive behavior disorder.

TABLE 1
Age of Patients Admitted to Huntington Intercom[illegible] Hospital Children's Mental Health Unit

	Number (N=117)			
Age Range	Male	Female	Total	Percent
3–5 years	11	5	16	14
6–9 years	40	8	48	41
10–13 years	40	13	53	45

TABLE 2
Distribution of Diagnostic Categories at the [illegible] community Hospital Children's Mental Health Unit

	Admission Diagnosis		Di[illegible]	[illegible]
Disorder	Number (N=117)	Percent	N[illegible] (N[illegible]	[illegible] Percent
Psychosis	9	8		8
Personality disorder	33	28	[illegible]	19
Primary neurological disorder with secondary behavior problems	9	8		10
Neurosis	10	9	[illegible]	9
Reactive or crisis disorder	29	25	[illegible]	26
Developmental disorder	22	19	[illegible]	20
Psychophysiological disorder	5	4		5
Mental retardation	0			2

*Final discharge diagnosis was not available for 11 patie[illegible]

TABLE 3
Discharge Disposition of Patients Admitted to [illegible] community Hospital Children's Mental Health Unit

Disposition	Number (N=130)	Pe[illegible]
Returned home	94	72
Placement		
Foster homes	13	10
Foster homes	7	5
Group treatment homes	6	[illegible]
Residential treatment facilities	9	[illegible]
Death	1	1

*The original 117 patients studied plus 13 for whom d[illegible] available at the time of follow-up.

DISCUSSION

Community Needs

In 1971 psychiatric hospital units fo[illegible] counted for an estimated 9% of all inpati[illegible] sodes among children under 18 in state, c[illegible] ty hospitals (4). A typical inpatient treat[illegible]

children and adolescents has fewer than 50 beds; is city, county, or state operated; and admits either adolescents only or both children and adolescents. Significantly, only 8% of psychiatric inpatient units restrict their admissions to children under 13. Therefore, less than 1% of all psychiatric episodes took place in what might be properly termed a children's psychiatric hospital unit.

The average age of admitted patients in the general hospital children's inpatient unit is 15, the average age in the children's psychiatric hospital unit is 13, and the average age in the residential treatment center is 14. This contrasts markedly with the average age in the CMHU described here—9.7 years. Clearly, there is a paucity of units devoted exclusively to the care of younger children, resulting in a restriction of adequate attention to their special needs. Appropriate program development for this group—age 3–13—constitutes a special need.

An Alternative to Residential Treatment Centers

Conclusions from the NIMH report on children's hospital units for emotionally disturbed children (4) indicated that general hospital inpatient units and children's psychiatric hospitals had fewer exclusions of suicidal and psychotic children than did residential treatment centers. This is appropriate due to the special status of the community unit as a resource for crisis intervention. The general hospital setting offers a wider variety of trained specialists for the assessment and treatment of the disturbed child than does the residential treatment center. Therefore, the hospital setting is more amenable to the diagnosis and treatment of the more complex and problematic cases.

The report noted that units restricting services to children had a longer length of stay and a slower turnover rate than did adolescent units. This has not been our experience. Our data indicate that the average length of stay (51 days) and the patient turnover ratio compare favorably with units restricting services to adolescents and are explained by the emphasis on psychiatric diagnosis and treatment as opposed to long-term rehabilitation.

A CMHU in the community appears to be an efficient service model representing the coordinated application of the highest level of professional training to both treatment-management planning and meaningful discharge and follow-up care.

Part II of this study will follow up children admitted to the CMHU in the areas of functional improvement, developmental progress, need for readmission, and need for reliance on longer term facilities.

CONCLUSIONS

The concept of residential treatment for children has evolved in part out of the necessity to remove the most severely disturbed children from their families and community. The roots of many existing institutions derive from foundling homes, orphanages, and religious and charitable homes for children and youths. The concept of community responsibility for the rearing of the child has been extended now to a concept of intervention in the child's development at critical junctures where family, school, and existing community support systems fail.

Since the end of World War II, the community general hospital has matured as an institution of service and leadership in providing comprehensive health care. The development of adult psychiatric inpatient services parallels that progress. At present, the majority of acute psychiatric admissions are referred to general and community hospital facilities.

For several reasons, there has not been a parallel development of inpatient facilities for children in the community. Ideologically, the child guidance clinic with its multidisciplinary-team approach has been home- and family-centered. Despite innovative outreach to schools and social agencies, significant intervention and comprehensive clinical data-gathering are inhibited by the appointment structure and recurrent problems in staff communication. Consequently, many seriously disturbed children and families are maintained for months and years without definite focus in all determinants of the clinical problem or referral to alternative management options (7). The available models for residential treatment have posed limits to our conceptualization, leaning primarily on state hospital units or various therapeutic residential schools located outside urban areas.

The community general hospital is a natural focus for a diagnostic and short-term child psychiatric treatment unit. Its advantages are 1) proximity to major population areas, 2) ready availability of large numbers of staff physicians and specialists for diagnosis and treatment, 3) easy accessibility to related social agencies, and 4) outpatient treatment programs for ultimate referral. The attending staff of child psychiatrists provides the same leadership as other medical and surgical specialties within the hospital and, in conjunction with the supporting staff, coordinate a meaningful diagnostic and treatment program. The major emphasis for any particular child may be on careful diagnostic delineation, intensive crisis resolution for child and family, redirection of an inadequate management and treatment program, and the setting of realistic limits and goals for the child's later development. These functions are unquestionably valuable and, unfortunately, are frequently not performed optimally in current existing models of child guidance centers, private outpatient treatment clinics, and individual practices.

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Extrapyramidal Side Effects with Lithium Treatment

BY JOHN KANE, M.D., ARTHUR RIFKIN, M.D., FREDERIC QUITKIN, M.D., AND DONALD F. KLEIN, M.D.

The authors neurologically examined 38 patients who had received lithium carbonate for from 3 months to 5 years to determine the presence of extrapyramidal side effects. No patient had received neuroleptics for 3 months before examination. Definite cogwheeling was found in 2 patients. The results confirm previously reported occurrence of cogwheeling in lithium-treated patients.

LITHIUM CARBONATE, now an established treatment in the prophylaxis of certain affective disorders (1), continues to be applied experimentally in the treatment of other conditions (2). Considerable experience has been accumulated with regard to potential toxicity (3), yet the first report suggesting that extrapyramidal symptoms (EPS) might frequently occur with lithium treatment (4) appeared only recently.

Shopsin and Gershon (4) examined 27 patients taking lithium and reported that 15 of 20 patients on lithium for over 1 year showed evidence of cogwheeling. None of these patients had received neuroleptics during their lithium treatment. Intravenous administration of 4 mg of benztropine mesylate to 9 randomly selected patients with cogwheeling resulted in some improvement in 2 patients and no change in 7. The cogwheeling they observed occurred in the absence of other extrapyramidal symptoms (EPS) ... symptoms except intermittent tremor. ... suggested that the presence of cogwhe... lated to length of treatment (no patient ... ium for less than 8 months manifested ... rather than age, sex, dosage, blood level, ...

Using the Simpson Angus Scale (5) an... al item for cogwheeling, Branchey and ... examined 36 outpatients receiving lithi... months to 7 years. No patient had rece... tics for at least 6 months before exami... taking antidepressants. In 3 patients who ... ing lithium for 2 or more years, some ... wheeling was present (each scored 1 ... scale), together with limb rigidity and tre... er patients had been taking lithium for 2 y... Six other patients had rigidity (not ... type) and tremor; 15 had tremor but ... these, 4 also had positive glabella tap), a... ity but no tremor. No patient received ... than 1 on any scale item related to rigi...

This study, undertaken to determine ... of EPS in patients receiving lithium trea... from prior reports in that all examinatio... out by a rater blind to the patient's diagn... ment.

METHOD

Thirty-eight male and female outpatie... 36 years) who had received lithium ca... least 3 months without concomitant neu... examined for EPS. The examination w... by a physician (A.R.) blind to the patien...

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(i.e., patients receiving lithium and patients receiving neuroleptics were examined). Patients were rated on the following 7 items from the Simpson Angus Scale (5): gait, arm dropping, shoulder shaking, elbow rigidity, fixation of position or wrist rigidity, tremor, and salivation. We added items for cogwheeling dystonia and akathisia and two items to assess akinesia (spontaneity of speech, gestures), all on a 5-point scale. Cogwheeling was measured by moving the patient's forearm while the patient remained relaxed in order to assess the amount of fluctuating resistance.

The sample consisted of 36 bipolar manic-depressive patients and 2 schizo-affective patients, diagnosed according to the *Research Diagnostic Criteria* of Spitzer and associates (7). Seven patients were receiving imipramine (range, 50–250 mg/day) in addition to lithium carbonate. Two patients were receiving diazepam up to 10 mg/day.

Patients were receiving a mean lithium dose of 1300 mg/day (SD, 396; range, 900–2700). The mean serum lithium level was 0.79 mEq/liter (SD, 0.22; range 0.50–1.30). Mean duration of lithium treatment was 15.6 months (SD, 13.2; range, 3–60).

RESULTS

Of the 38 patients examined, only 2 manifested evidence of cogwheel rigidity, and both of them manifested additional signs of extrapyramidal disorder, although of minor significance. The most frequent positive rating was for tremor (N=17), which was fine and rapid and usually of mild to moderate severity. Several of the patients with tremor were subjectively unaware of it before the examination. Few patients complained of drug side effects. Neither of the 2 patients exhibiting cogwheel rigidity were receiving any medication other than lithium carbonate.

One patient received several abnormal ratings (gait, +1; arm dropping, +3; elbow rigidity, +2; wrist rigidity, +2; tremor, +3; and akathisia, +1). None of these abnormalities responded to benztropine mesylate, 2 mg I.M. We referred this patient for further neurologic workup, which led to a diagnosis of normal pressure hydrocephalus. This was the only patient who received ratings greater than 1 on any item other than tremor or cogwheeling.

Six other patients received ratings of 1 on the following items: akathisia (N=2), spontaneity of speech (N=1), wrist rigidity (N=1), arm dropping (N=1), and gait (N=1). We feel that scores of 1 are not clinically significant and cannot be considered as definite abnormalities. One patient with +1 akathisia was receiving 100 mg/day of imipramine and the patient with +1 arm dropping was receiving 50 mg/day of imipramine.

We explored in great detail the medication histories of the 2 patients exhibiting cogwheeling with the patient, from chart review, and by contacting previous physicians. One patient had been exposed to neuroleptics for 6 weeks during the treatment of an acute manic episode, which had occurred 8 months before neurologic examination. This patient was reexamined on several occasions during the 6 months after neurologic examination, and the cogwheeling persisted at the same level of intensity. The patient did not experience a sensation of rigidity. The other patient had received several courses of neuroleptic treatment, including chlorpromazine, trifluoperazine, thioridazine, and fluphenazine decanoate. She had not received any neuroleptic medication for 5 months before neurologic examination. It is conceivable that, despite a 5-month interval, the last medication she had received, an injection of 25 mg of fluphenazine decanoate, contributed in some way to the development of persistent cogwheeling; however, the patient continued to manifest this sign 4 months after the original examination (i.e., 9 months after the injection). Neither of these subjects had a rating greater than 2 on any other item. Both patients were suffering from bipolar illness. The first was 19 years old and had been receiving lithium for 8 months (dose, 1800 mg/day; blood level, 0.8 mEq/liter); the second was 55 years old and had been receiving lithium for 20 months (dose, 1200 mg/day; blood level, 0.9 mEq/liter). Both were subsequently treated on two separate occasions with benztropine mesylate, 2 mg I.M., and examined 1 hour after treatment. Cogwheeling did not improve in either patient.

DISCUSSION

Our findings are consistent with those of Branchey and associates (6), who found cogwheeling in 3 of 36 patients receiving lithium carbonate. This report differs from previous studies in that our examinations were carried out by a rater blind to the patient's medication. Even in the physical examination of patients, considerable subjectivity and variation in interexaminer reliability occurs (8).

Our finding of a relatively high prevalence of tremor is consistent with the reports of others (4, 9). Since the tremor that occurs frequently with lithium treatment does not respond to antiparkinsonian drugs, it has been felt that the tremor is not related to the extrapyramidal system (3).

The occurrence of cogwheel rigidity, however, is surprising and difficult to explain. Although lithium has been reported to produce a variety of side effects (9), extrapyramidal involvement, except in toxic states, has rarely been reported. Despite its low prevalence, cogwheeling as a side effect of lithium treatment requires explanation. Further investigation of potential neurologic side effects with lithium treatment is of clinical and heuristic importance.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Psychiatric Illness in Relatives of Patients with Parkinson's Disease: An Expanded Survey

BY ANDREW WINOKUR, M.D., PH.D., JOHN DUGAN, JOE MENDELS, M.D., AND HOWARD I. HURTIG, M.D.

Several recent reports have suggested that there may be an association between Parkinson's disease and some forms of depressive illness (1–3). This prompted us to study the prevalence of affective disorders in first-degree relatives of patients with Parkinson's disease. It was proposed that the genetic factors predisposing to these two conditions might overlap, and that the lifetime prevalence of depressive illness in the first-degree relative of probands with parkinsonism might be greater than that in the general population. In order to test this hypothesis, a pilot study involving seven patients with Parkinson's disease was carried out, and a 24% frequency of primary affective disorder was observed in the first-degree relatives of these patients (4). On the basis of these data, we have studied a larger number of subjects but have been unable to replicate our previous findings of a high frequency of affective disorder in the relatives of patients with Parkinson's disease.

Method

Patients were selected from the Movement Disorder Clinic and the Neurology Group Private Practice of the Hospital of the University of Pennsylvania. The subjects were 25 consecutive patients who agreed to participate in the study, had minimal or no dementia, and had a first-degree relative available to accompany them for the interview. The patient, at least one first-degree relative, and, in many cases, the patient's spouse were interviewed together in a semi-structured format. Information was obtained for every first-degree relative regarding possible indications of affective disorder, schizophrenia, and alcoholism. Attempts were made to contact any living relative whose history was suggestive of affective disorder. Interviewed family members were diagnosed according to the *Research Diagnostic Criteria (RDC)* (5) and noninterviewed relatives according to the *Family History-Research Diagnostic Criteria (FH-RDC)* (6).

Results and Discussion

The 25 probands had a total of 113 male and 109 female first-degree relatives. Of these relatives, only 4 males (3.5%) and 7 females (6.4%) had clear histories of affective disorder. Even when the criteria for inclusion were eased to take into account relatives who had suggestive but incomplete histories of affective disorder, only 10 female relatives (9.2%) were identified. It should also be noted that few unequivocal cases of schizophrenia and alcoholism were encountered in these relatives.

It is reasonable to consolidate the data obtained from the 7 patients reported on previously with the 25 probands described in the current publication, since all 32 patients were obtained from the same clinic and were studied according to the same protocol. Information was thus available for a total of 138 female first-degree relatives of the 32 probands with Parkinson's disease. Fourteen of these female relatives (10.1%) had clear histories of affective disorders. Figures for the lifetime prevalence of depressive illness for fe-

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males in the general population have been estimated to be as high as 9% (7).

Thus, even with the inclusion of the original probands in our total of 32 patients with Parkinson's disease, we are unable to substantiate the earlier suggestion that relatives of such patients might show a higher than expected lifetime prevalence of affective disorder.

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Caffeine-Induced Delirium During Prolonged Competitive Stress

BY VERNER STILLNER, M.D., M.P.H., MICHAEL K. POPKIN, M.D., AND CHESTER M. PIERCE, M.D.

Excessive consumption of caffeine is a practice commonly used to overcome drowsiness and forestall sleep. Ritchie (1) has contended that dosages of 1000 mg of caffeine are capable of producing untoward reactions referrable in large part to the central nervous system. Such responses are said to entail restlessness and excitement, which may progress to mild delirium with sensory disturbances. McManamy and Schube (2) described a caffeine-induced psychotic state in a 1936 case report. Caffeine has also recently received attention as an etiologic factor in certain anxiety states (3). However, a review of the literature to date has not revealed other clinical reports corroborating Ritchie's contentions. This paper is a self-reported account of a caffeine-induced delirium (consistent with Ritchie's description) that occurred during a prolonged competitive stress situation.

Case Report

Mr. A, a 28-year-old single Alaskan fisherman and trapper, was entered in the 1977 Iditarod Trail International Sled Dog Race. The Iditarod spans 1,049 miles between Anchorage and Nome, following a route used decades ago by runners bringing diphtheria serum to Nome. Each March since 1973, "mushers" have proceeded overland by sled with dog teams for prize money (4).

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This work was supported by a grant from Roche Psychiatric Services Institute, Nutley, N.J.

The authors wish to thank the Iditarod Trail Committee and the racers.

In the third week of the 1977 competition, V[illegible]te an of two races, chose to mush continuously [illegible] h[illegible] 48 hours. He was only 2–4 days from Nome [illegible] he racers, was unwilling to yield time to sleep [illegible] ic, juncture of the race. Following an evening [illegible] of chops, two cups of brewed coffee, and three [illegible] resumed mushing in subzero temperatures [illegible] or winds. Despite this intake of an estimated 270–[illegible] ca feine (5) with his meal, he experienced increa[illegible] staying awake. Two hours after the meal, he in[illegible] of caffeine (2 tablets of Vivarin, an over-the-co[illegible] tion). Approximately 20 minutes later, he ing[illegible] tional 400 mg. Thus, in less than 3 hours he h[illegible] than 1000 mg of caffeine.

Half an hour later, he noted the emergence [illegible] re sion of several symptoms. His hands became t[illegible] he experienced a pronounced buzzing in his [illegible] ceived his miner's headlamp as emitting no m[illegible] row band of light. The ascent of a long hill se[illegible] were a flat plain riddled with white stars." V[illegible] and he tumbled twice from his sled in a sho[illegible] experienced doubt as to whether he was reall[illegible] feared being alone. This veteran driver, accus[illegible] cult conditions and sleep deprivation durin[illegible] managed to continue mushing. With the light[illegible] the passage of an interval of 6 hours (still wit[illegible] symptoms abated without subsequent diffic[illegible] rence. He arrived in Nome 3 days later and re[illegible] rience to one of us (V.S.) within hours of fin[illegible] status examination in Nome within 24 hour[illegible] pletion of the race revealed no aberrations and[illegible] A toxicology screen was negative. Complet[illegible] and blood chemistries were within normal lim[illegible]

Comment

In two previous races under similar co[illegible] ions th[illegible] man had used as much as 400 mg of caffe[illegible] b[illegible] form in 24 hours without untoward seq[illegible] t[illegible] 1977 race, within 30 minutes of serially c[illegible]

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proximately 1070 mg of caffeine, he noted a variety of symptoms. He experienced tremor, impaired memory, altered levels of consciousness, vertigo, pronounced anxiety, and sensory disturbances, including visual illusions and hallucinations. This picture is consistent with delirium (6).

With the advent of daylight after 6 hours, the symptoms fully remitted before he was able to warm himself or sleep. Hence, sleep deprivation or hypothermia appear insufficient factors to explain a transient and self-limited delirium. Rather, we believe the picture is likely best explained as a caffeine-induced delirium such as that suggested by Ritchie (1). The resolution of the delirium within a brief number of hours would not be inconsistent with the known half-life of caffeine in man following oral intake (7).

Mr. A neither smoked nor drank alcoholic beverages, and his medical and psychiatric history were unremarkable. He reported an average daily caffeine consumption of 270–360 mg. Psychiatric examination in Anchorage shortly before the race gave no evidence of any aberration. In addition, full pre- and postrace screening performed as part of an ongoing investigation of prolonged competitive stress (8) failed to reveal any striking abnormality in a battery of laboratory tests, including CBC, SMA-24, urinalysis, and several endocrine tests. There was no evidence of infectious process, and drug screening both before and after the race was negative, minimizing the likelihood of other toxic factors in the genesis of the delirium. Although other physiological contributors cannot be entirely excluded, it appears that the principal etiologic factor in the delirium was the excessive consumption of caffeine.

This case corroborates Ritchie's contentions about toxic effects of excessive amounts of caffeine and indicates that the common practice of using caffeine to stay awake may give rise to hazardous sequelae. As little as five 200-mg tablets of readily available over-the-counter preparations of caffeine appears sufficient to generate a delirium. Even smaller amounts may lead to sufficient sensory and motor disturbances to prove dangerous in situations such as long-distance driving. Such effects of caffeine merit closer attention.

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Antagonism of Cocaine Highs by Lithium

BY ALVIN J. CRONSON, M.D., AND ABRAHAM FLEMENBAUM, M.D.

Beneficial effects of lithium treatment for amphetamine abuse were reported by one of us (A.F.) in two patients who spontaneously stopped abusing amphetamine while they were taking lithium because they no longer experienced the expected high. A third patient receiving lithium and amphetamines for weight reduction did not experience appetite suppression, highs, or the subjective effects of the amphetamines until lithium was discontinued (1). This concept was further supported by a placebo-controlled double-blind study of nine severely depressed patients. Lithium treatment resulted in a 60% reduction in their activation and euphoric responses to *d*-amphetamine and almost complete abolition of response to L-amphetamine (2).

There is a large body of evidence, both direct and indirect, regarding the beneficial effects of the use of lithium in amphetamine abuse, although the reports are still controversial (3–7).

Recently, Flemenbaum (8) demonstrated an inhibition of hyperactivity and stereotypic behavior in 10 rats (serving as their own controls) given doses of up to 19 mg/kg of cocaine. In this paper, we will report some clinical experiences that appear to support the hypothesis of an inhibition of cocaine highs by lithium.

Case Vignettes

Mr. A, a 28-year-old married man, had been detoxified from heroin and was drug-free. He was stabilized on lithium because he had symptoms of affective disorder. He reported

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he had obtained and used "pure coke" but had noticed no reaction. He had once taken cyclazocine and knew that it blocked the effects of heroin, and he asked whether lithium had a similar effect on cocaine.

Mr. B, a 25-year-old man, was on methadone maintenance, 90 mg/day. He was difficult to manage until he was started on lithium because of drug abuse and arrogant, manipulative, and impulsive behavior. He reported having no response to cocaine when he was receiving lithium, although his companions became "quite high on it."

Mr. C, a 28-year-old former addict and alcoholic, had been placed on lithium to control his mood swings, violent temper, and impulsiveness. He tried cocaine and found to his surprise that he had no reaction. He was grossly obese and thought that this would prevent him from getting a job, so he was given amphetamine on a careful experimental basis. He reported that while the amphetamine controlled his appetite, he felt no high and did not "crash" when the effects wore off, even if he took more medication than was prescribed. He lost 13.6 kg and was only 9.1 kg overweight when he completed treatment.

Mr. D, a 23-year-old single man with a diagnosis of manic depression, was placed on lithium with excellent results: he became stable in his interpersonal relations and employment and stopped abusing hypnotics and alcohol. He went to a Christmas party where a group of people were using cocaine. He had had some brief experiences with cocaine in college and looked forward to getting high, but he had no response to the drug, which "everyone else thought was great."

Ms. E, a 32-year-old single actress with a history of bipolar manic depression, had been diagnosed and successfully treated at several well-known lithium clinics. In spite of her good response, she had multiple rehospitalizations. During her most recent hospitalization, cocaine was discovered in her urine and her lithium level was negligible. Once stabilized, she confided that she liked the cocaine high but could not obtain it while she was taking lithium, so she had discontinued the lithium several times.

Discussion

Jefferson and Greist (3) have noted that "There is presumptive evidence in man that lithium can attenuate the activating and euphorogenic effects of amphetamine, but firm conclusions will not [illegible] until there are more well-designed studies."

Although our report is anecdotal, we [illegible] further illustrates the potential ability of [illegible] hibit drug highs. We recognize that the [illegible] this action may be indirect, i.e., by [illegible] masked affective disorder lithium may [illegible] creased need for the highs obtained from [illegible] We also recognize potential biases of non[illegible] tions, but we conclude that the evidence [illegible] support a potential use of lithium in the [illegible] drug abuse, including cocaine, and that [illegible] serves further scientific evaluation.

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Bromism: Alive in Well

BY IRA BRENNER, M.D.

Bromism was first recognized 50 years ago, and reports abound to remind us that the entity persists (1, 2). In these reports, the source of the ion is usually traced to over-the-counter or prescribed medications (3, 4). To my knowledge, bromide intoxication from contaminated well water, as seen in the [illegible] scribe, has not been reported previously.

Case Report

A 51-year-old woman came to the emerg[illegible] cause of recurrent suicidal impulses, early [illegible] ening, anorexia, and increased alcohol cons[illegible] the previous week. She had not worked that [illegible] had an overwhelming sense of hopelessness [illegible] denied taking any medications, either prescri[illegible] counter. Her history revealed a 12-year epi[illegible]

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alcoholism and depression that required four psychiatric hospitalizations for detoxification and supportive psychotherapy. The most recent hospitalization was 2 years before this admission. There were many stresses in her life, which were seen as precipitants. Previously, she had been diagnosed as having depressive neurosis with characterologic problems, and she had been treated with benzodiazepines. Her medical history included a hysterectomy 13 years previously. She had also sustained a concussion 3 months before admission in a single-car accident. She said she felt "down" at the time but denied suicidal intent. At that time, psychiatric consultation was not obtained and bromide levels were not assessed. Her family history included alcoholism in her father.

Mental status examination revealed a neatly dressed, friendly, alert, and oriented woman with mild restlessness, hand wringing, and a fine tremor. Her nihilistic feelings reached near-delusional proportions. She was apathetic and her affect was restricted to dysphoric expression. Her remote memory was hazy and inconsistent, although her recent memory and recall were grossly intact. Physical examination was unremarkable. Laboratory data, including thyroid studies, B_{12} level, and electrolyte and metabolic values, were essentially normal; the serum bromide was 38.3 mg/100 ml (normal, <5 mg/100 ml).

The patient's mood, affect, memory, and tremor improved quickly following hospitalization. In addition, serial bromide levels showed a decrease without medical treatment, returning to normal within 1 week and remaining at normal levels. Further questioning and history from other sources confirmed the patient's assertion that she took no medication and ate no unusual or treated foods. However, for the past 5 months she had used well water, which was assayed and found to have a bromide level of 2.1 mg/100 ml. (State regulations permit no bromide in drinking water, and the reason for the presence of bromide in the well remains unclear, although industrial wastes are suspected.) After her cognitive functioning and mood improved, she was able to work successfully on underlying dynamic issues during her 3-week hospitalization. She has had no recurrence of symptoms in 5 months of follow-up.

Discussion

The patient presented with an agitated depression. In addition, she displayed subtle evidence of bromide toxicity, including tremor and memory disturbance. She was exposed to the well water for 5 months before her admission, and moved after her discharge from the hospital. She had been using the well for 2 months before her auto accident, and one could speculate that bromide intoxication may have been contributory. Her bromism dissipated within a week without treatment. A return to normal blood levels in such a short time is unexpected, since the half-life of bromide has been reported to be about 12 days (5). However, in that study the dosage produced no symptoms, and "the amount of ammonium bromide administered to the subjects was considered to be sufficiently small as not to disturb the physiological distribution." The clinical applicability of such a study to subacute bromide intoxication is questionable, since other factors such as blood level and duration of ingestion might alter the excretion rate.

Our patient's highest recorded blood level was only 38.3 mg/100 ml, which is below the generally accepted range of toxicity. However, there is disagreement about toxic levels of bromide. Cut-off points range from 50 mg/100 ml (6) to 72 mg/100 ml (7), and some clinicians feel that mental status changes do not occur until even higher levels. A recent report of mania in a patient with a bromide level of 32.5 mg/100 ml (8) was criticized because of the relatively low blood level (9). Indeed, one might argue that our patient would have become symptomatic without bromide ingestion. However, dismissing the data as incidental is as presumptuous as ascribing a cause-effect relationship in such cases. It has been noted that "bromide schizophrenia" primarily occurs in women with premorbid schizoid personalities (4), and our patient may have been susceptible to subtoxic doses of bromide. Given her propensity toward mood changes, it seems reasonable to assume that she could become symptomatic at a lower than expected bromide concentration, whereas others using the well water remained asymptomatic.

It may be that our patient's threshold was decreased and bromide potentiated her characteristic depressive response to psychic stress. A constitutional or acquired biochemical defect that would allow subtoxic levels of the ion to interfere with neurotransmission of biogenic amines could exaggerate or precipitate mood changes in vulnerable people.

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Psychotic Depression and Bronchogenic Carcinoma

BY JONATHAN G. SOLOMON, M.D., AND SANDRA SOLOMON, M.S.

This communication was prompted by a recent case in which a 51-year-old woman with manic-depressive illness, depressed type, presented with what appeared to be her fourth episode of psychotic depression. The episode was similar to her three previous episodes of affective illness, but medical evaluation unexpectedly revealed the presence of a bronchogenic carcinoma, which may have caused or contributed to her symptomatology. This case highlights the importance of medical evaluation of psychiatric patients.

Case Report

The patient, a 51-year-old rural housewife, had her first episode of psychotic depression when she was 35, her second at 39, and her third at 45. Each episode was characterized by depressed mood, anergia, anhedonia, sleep disturbance with early-morning awakening, difficulty in thinking, psychomotor retardation, feelings of hopelessness, loss of libido, and anorexia, with weight losses ranging from 6 to 12 kg. She had never been suicidal, delusional, hallucinatory, or paranoid. Medical history was negative except for her habit of smoking two packages of cigarettes daily. The family history was negative for affective illness and alcoholism. Each depressive episode responded to hospitalization and treatment with tricyclic antidepressants. Typically, she improved gradually over a 4–6 week period and was asymptomatic on discharge from the hospital. She was last discharged on imipramine, 150 mg h.s., but unfortunately refused maintenance chemotherapy "because I don't want to depend on pills." She declined periodic office visits and only consented to see a psychiatrist when her periodic depressive episodes occurred and became so severe that hospitalization was necessary.

Recently she complained of a fourth episode of depression. Her symptoms were similar to those of her three prior episodes and had begun approximately 3–4 months earlier without any evident precipitant. On admission she appeared cachectic, with marked pallor. Her appetite was poor and she had lost 10.2 kg. Psychomotor retardation was noted and she complained of feelings of depression, lack of energy and interest in living, and inability to function at home. There was no evidence of organicity and no history of drug or alcohol abuse. Her husband stated that this was a typical presentation, saying "This is the way she gets."

Routine medical evaluation revealed a hemoglobin of 8.6 mg and hematocrit of 26.0%. A chest film revealed the presence of a bronchogenic carcinoma with metastases. Consultation with an internist was arranged and the patient was transferred to the medical service for appropriate chemotherapy and radiation therapy.

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Discussion

This patient's clinical presentation w psychotic depression and occurred i three prior episodes of similar symptom is unwise to make a diagnosis solely o presenting symptomatology or psychi Medical evaluation is also necessary F uate this patient medically would have c priate diagnosis and treatment.

An organic lesion was not suspecte cause of the typical appearance of affect in a manic-depressive patient. A bronc noma was certainly unexpected in the a monary symptoms such as chest pain, d ness of breath, or hemoptysis. It is un this patient had another bout of affective tion along with the bronchogenic carcino er the malignancy caused or contributed toms, especially the anorexia, weight lo pallor, and anemia. She had not previou mic. Had she been receiving maintenan sant chemotherapy (1) an exacerbation ness would have been less likely to occ toms of weight loss, fatigue, and palle been more likely to be perceived as bei physical cause. Certainly these sympto sence of the psychiatric or emotional co pression would have alerted the clinic organic etiology. Although patients bronchogenic carcinoma often develo secondary to their illness, depression manifestation of a bronchogenic carc tinctly uncommon (2). Depression is tients with diagnosed carcinoma, presu of the realization of the gravity of the physical debilitation (3).

The differential diagnosis between organic illness can be difficult becau symptoms can be the initial manifesta pathology such as an occult malignan pancreatic carcinoma, retroperitoneal ly brain tumors, and of other illnesses, incl sclerosis, pernicious anemia, and endoc such as hyperadrenocorticism, hyperi hypothryoidism (4). Rossman (5) descri of cases in which depression dominate picture and delayed the diagnosis o orders; often, the lesion was an occult m reported that the erroneous diagnosis ease as emotional may occur because medical evaluation, the rarity of the illn vague complaints, the appearance of me before abnormal physical and laboratory

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or the coincidental occurrence of organic disease with psychiatric symptoms.

Wingfield (6) described several patients with unrecognized organic disease causing emotional symptoms that prompted the referral for psychiatric treatment. "Missed" diagnoses included epilepsy, multiple sclerosis, porphyria, neurofibromatosis, and amyotrophic lateral sclerosis. Stokes and associates (7) reported the following illnesses found on a psychiatric unit and initially misdiagnosed as psychiatric or functional: carcinoma, brain tumor, hypoglycemia, meningitis, thyrotoxicosis, myxedema, endocarditis, and pneumonia. Herridge (8) reported that 5% of patients admitted to the psychiatric unit of a general hospital suffered from a previously undiagnosed organic disease that was responsible for the psychiatric symptoms, and Saravay and Koran (9) found that 4% of psychiatric consultations in a large general hospital involved obvious organic disease erroneously diagnosed as psychiatric.

Psychiatrists would be well advised to adopt a holistic approach to patient care, including thorough medical assessment, especially for inpatients and for the frequent patient with affective symptoms.

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"Spice Cabinet" Intoxication

BY ROBERT A. FAGUET, M.D., AND KAY F. ROWLAND, PH.D.

A recent clinical report (1) in this journal discussed nitrous oxide abuse by a 15-year-old boy who used aerosol cans of whipped cream to obtain the gas. Another equally dangerous high has come to our attention: nutmeg ingestion, which we have labeled "spice cabinet" intoxication.

Case Report

A 19-year-old student, after reading about the mind-altering properties of nutmeg in a popular "consumer's-guide" book about drugs, decided to experiment. He went to the kitchen spice cabinet, took down a can of ground nutmeg, and mixed a tablespoon of it with orange juice. The combination was distasteful, and he was able to consume it only with some difficulty.

Within 2 hours he was "in trouble." He felt flushed, dizzy, nauseated, and his mouth became parched. His heart pounded and a "tremendous" headache overcame him. Gradually his extremities became numb, his perception of time and space changed, and he became detached and restless. At one point he reported significant confusion and said he was "on the verge of visual hallucinations." Twelve hours later, after very little sleep, the intoxication was over, leaving him lethargic, achy, and subdued.

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Nutmeg is among the oldest spices in use. It is derived from the seeds of an aromatic evergreen tree, *Myristica fragrans*, which is native to the Spice Islands. Myristicine, the psychoactive ingredient, is found in the oil of the seeds and also exists in the seed coat, which is processed separately to produce mace. Nutmeg has been used medically as a stimulant and tonic for the stomach, but a 1525 account by Richard Banckes suggests its psychoactive properties: "To cleanse the brain of superfluous humours, take a quantity of maces and chew them well" (2, p. 98). For years, prison officials have known that inmates go on "nutmeg jags" when other drugs are unavailable.

Richard Evans Schultes, an early investigator of medicinal plants, described myristicine intoxication via snuffing. His first-person description is very much like the case reported here:

> The dose was snuffed at 5:00 p.m. Within 15 minutes, a drawing sensation was felt over the eyes, followed very shortly by a strong tingling in the fingers and toes. Then came a severe headache. Within half an hour, my feet and hands were numb. Walking became almost as difficult as when I had beriberi. I felt nauseated until 8:00 p.m. when I took to my hammock, overcome with drowsiness. Strangely, this was accompanied by muscular excitation, except in my hands and feet. About 9:30 p.m. I fell into a fitful sleep which continued with frequent awakening until

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morning, when a strong headache continued until noon. A profuse sweating and, probably, fever persisted throughout the night. My pupils were strongly dilated during the first few hours of intoxication. Though performed under primitive conditions in the jungle, this experiment does, I think, indicate the great strength of the snuff. The larger dose used by the witch-doctors causes them to see visions in colors, which I did not experience. During their dreams, they emit wild shouts that are interpreted by their assistants. They admit that using this substance is a dangerous practice, one of their number from the Puivave tribe having died during Yakee intoxication. (2, pp. 99–100)

Nutmeg abuse can be serious. Not only are the psychic effects disturbing and dangerous, but poisoning and death due to fatty degeneration of the liver have been reported (2). Fortunately, in larger quantities nutmeg is unpalatable and the side effects
unpleasant. However, as in the case of th
student, advertisement in popular "dru
lead some young people to experiment.
one such book, titled *Recreational*
"Those looking to add a bit of spice
sometimes become curious about nutme
Regrettably.

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Selection Criteria for Dialysis and Renal Transplant

BY STANLEY RABINOWITZ, PH.D., AND HERMANUS I.J. VAN DER SPUY, PH.D.

In a number of countries there are limited dialysis and transplantation facilities, and not all patients can be selected for renal treatment. Those who must make the selection are burdened with the moral dilemma of deciding who is to live and who is to be left to die. With no clearly established medical, social, psychiatric, or psychological criteria for selection, the decision is difficult and must inevitably include the subjective impressions of the selection team.

Researchers (1–3) have indicated that patients with end-stage kidney disease face three broad areas of stress. Physically, they face the stress of physiological changes and discomfort, including adherence to a strict diet and restrictions in fluid intake. Psychologically, they face the stress of increased dependency on the dialysis machine and the renal team. Socially and personally, they face increased financial and emotional dependency, frustration of their instinctual drives, and various associated limitations.

There is no general agreement on the factors determining favorable adjustment to dialysis. While earlier investigators (4, 5) suggested that average or above average intelligence was an important prerequisite for favorable dialysis adjustment, recent in
have presented conflicting evidence, alth
erally accepted that mentally retarded p
suitable for renal programs.

There has been comprehensive resea
ing personality factors involved in fav
adjustment (4, 5, 7). Although no clear
criteria are available, it seems general
positive features for good adjustment in
open admission of anxiety and hostility
quate range of dependency adaptation.
tors seem to include overdependency,
and frequent psychosomatic complain

The aim of the present study was to
criteria of selection for a renal progra
term dialysis or future transplantation)
a sample of accepted and nonaccepted p
hypothesized that patients with whom
team can more readily identify are mo
accepted for the program. Intellectua
social class, and personality factors were

Method

Subjects. Seventeen patients accepte
program at the Groote Schuur Hospital
South Africa, were compared on psycho
mographic variables with 11 rejected pa
jects volunteered for the study. All 28
judged as potentially acceptable on med
grounds. Those unacceptable on med
grounds alone were excluded from
sample.

The decision of acceptance or rejecti

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The authors wish to thank Drs. E. Nash and G. Thatcher, Consultant Psychiatrists to the Renal Department, Groote Schuur Hospital, Cape Town, South Africa, for their cooperation and assistance.

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gram was made by a team that consisted of surgeons, a psychiatrist, other physicians, a clinical psychologist (S.R.), and a social worker. Other than identifying them as an upper-middle-class group of professionals, no more detailed investigation of their religious or political beliefs or their psychological characteristics was undertaken. Because of the sensitive nature of the study, such investigations were not regarded as feasible.

Psychological measures and procedure. The groups, which were controlled for age, sex, and race, were contrasted on the following psychosocial measures: the Wechsler Bellevue Adult Intelligence Scale (verbal subtests), Standard Progressive Matrices, Rorschach Inkblot Test, and social class and education. Performance IQ subtests could not be administered because some of the patients were bedridden. However, the Standard Progressive Matrices, often regarded as a measure of general intellectual ability (8), served as the measure of general intellectual level of functioning. The Progressive Matrices percentile scores were converted to IQ equivalents according to the conversion table of Peck (9).

Social class levels were rated according to the scoring system developed by the Child Guidance Clinic of the University of Cape Town. Scores were assigned on a range of 1–6 in ascending order for each class division so that persons falling in class 6 (unskilled workers) obtained a score of 6, persons falling in Class 5 (semiskilled workers) obtained a score of 5, and so on. Education was scored on the basis of one point for each year. The Rorschach was scored according to the quantitative proportions of Klopfer, and the following personality dimensions were studied: 1) inner resources and impulse life; 2) affectional needs, 3) control factors, 4) emotional reactivity to the environment, and 5) intellectual manner of approach. The researchers also used the Rorschach scoring system developed by van der Spuy (10).

Results

Significant differences were found between the accepted and rejected groups on three measures (see table 1). The accepted sample had a significantly higher verbal IQ than the rejected group. Analysis of the subscales revealed significantly higher scores by the accepted sample on all the subtests except digit span. Both social class and education were significantly higher for the accepted than for the rejected group.

No significant differences between the groups on any of the personality variables were revealed by the results of the Rorschach.

Although there was a 5-point difference in favor of the selected group based on the Progressive Matrices IQ score, this is not statistically significant, whereas the selected group scored more than 20 points higher on the Wechsler verbal IQ. Verbal intellectual functioning is much more affected by cultural factors than a performance scale like the Progressive Matrices. The results would suggest that social and educational background emphasized and magnified any differences in intellectual potential that might have existed in the two groups.

TABLE 1
Comparison of Patients Accepted and Rejected for a Renal Program

	Accepted Group (N=17)		Rejected Group (N=11)*		Analysis	
Variable	Mean	SD	Mean	SD	t	p
Wechsler verbal IQ	95.87	19.89	74.6	22.59	2.51	<.02
Progressive matrices IQ	87.75	11.85	82.81	9.09	1.16	n.s.
Social class	4.00	1.27	5.09	1.13	−2.3	<.05
Educational level (years)	8.4	2.85	5.00	3.31	2.9	<.01

*One of the accepted patients and 1 of the rejected patients would not cooperate and failed to complete the WAIS. One of the accepted patients could not adequately complete the Standard Progressive Matrices because of poor eyesight.

Discussion

The results of the present study support the hypothesis that those patients with whom the selection team can more readily identify are more likely to be accepted for the renal program. The accepted sample showed significantly higher scores than the rejected group on measures of verbal intelligence, social class, and educational levels, while no significant differences were found between the groups on measures of general intellectual level of functioning and on personality variables.

These results seem to suggest that selection for the program was probably based on clearly perceivable but superficial characteristics such as verbal intelligence, social class, and education. The assumption is that the upper-middle-class professional selection team can identify more readily with these characteristics. In the absence of clear-cut criteria for selection, the team was probably influenced primarily by these superficial variables. However disturbing these findings might be, they do not imply condemnation of the selection team. In the absence of clear-cut selection criteria, many people would probably use similar subjective criteria without being aware they were doing so. Similar subjective and perhaps subconscious criteria probably operate in a wide range of selection situations, e.g., employment or medical school admission.

Despite the many difficulties and problems associated with the development of objective criteria for selection, we believe that research should be undertaken in order to establish more exact criteria. Until this is done, we are convinced that selection for treatment will continue to be made on the superficial variables we have outlined.

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Successful Treatment of Severe Anxiety Attacks with Tricyclic Antidepressants: A Potential Mechanism of Action

BY KENNETH JOBSON, M.D., MARKKU LINNOILA, M.D., PH.D., JOHN GILLAM, M.D., AND JOHN L. SULLIVAN, M.D.

A correlation between plasma tricyclic antidepressant levels and treatment responses has been demonstrated in moderate to severe depressions (1, 2). A curvilinear relationship has been reported for the secondary amines (1), whereas a linear relationship has been observed for the tertiary amines (2). The doses necessary to reach therapeutic levels have generally varied between 2 and 4 mg/kg of body weight, depending on which drug has been administered to which patient (1, 2). Low-dose tricyclic treatment (doses below 1 mg/kg) has been reported to be effective in the treatment of severe paroxysmal phobic anxiety (3).

Using gas chromatography we measured the plasma tricyclic levels during steady state kinetics in three severely anxious patients. All three patients had suffered from severe panic attacks, agoraphobia, and somatic anxiety symptoms for more than a year before the study. The symptoms had been disabling and were not alleviated by standard therapeutic doses of benzodiazepine medication. Low doses of tricyclic antidepressants induced a dramatic resolution of symptoms in these patients. When the patients stopped their medication all symptoms returned to their previous level of severity, and when the medication was r symptoms again disappeared. Plasma t during the initial management period ar table 1.

This dramatic resolution of severe an three patients with plasma tricyclic le low those reported for successful mana vere depressions makes it reasonable t mechanism of action for the tricyclics i of anxiety attacks that differs from the cepted mechanism in the treatment Drugs other than tricyclics reported to the treatment of severe anxiety attack monoamine oxidase inhibitors and prop All three classes of drugs can block β- ceptors (6, 7). We have found MAO in the most clinically effective agents in t severe anxiety attacks and propranol fective. Propranolol is the most pure blocker of these drugs, so we do not sug of central beta receptors as the mechan action in the treatment of severe anxiety tral adrenergic alpha-receptor blockade able in the treatment of anxiety (8), bu pha-receptor blockade after tricyclic a in man has been suggested to occur at higher than those necessary in the treatm sion (6). Therefore, adrenergic alpha-re ade is an unlikely mechanism of actio cyclics in treating severe anxiety attack

Both MAO inhibitors and tricyclic a have been reported to inhibit MAO in h in vivo (9). In addition, the degree of M

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TABLE 1
Three Patients Treated with Tricyclics for Severe Anxiety

Patient Number	Sex	Drug	Dose (mg/day)	Plasma Level (ng/ml)
1	Male	Imipramine	25	Imipramine, 50 Desipramine, >5<10
2	Male	Imipramine	25	Imipramine, 10 Desipramine, >5<10
3	Male	Amitriptyline	25	Amitriptyline, >5<10 Nortriptyline, >5<10

in human platelets seen after tricyclic administration is generally lower than that seen after MAO inhibitors (9). This is the same order of potency that we have seen in the treatment of anxiety attacks. Since in vivo MAO inhibition in human platelets can occur at very low tricyclic plasma levels and has been reported to correlate with the sedative effects of the tricyclics (9), MAO inhibition may be the mechanism that mediates the management of severe anxiety attacks.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Associ... requests for information, etc. Letters should be submitted in duplicate to the Editor, who ma... regarding publication. Letters must be typed double spaced throughout and should not contain ... words and 5 pertinent references. Criticisms of published articles will automatically be sent to th... response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we ... writers of the disposition of letters or return those which are not printed.*

Abnormal Gaits in Tardive Dyskinesia

SIR: Tardive dyskinesia continues to present problems of both a practical and theoretical nature. The continuing attention this problem has received has resulted in a broadening of the definition of the syndrome and a further awareness of its complexity and importance. We would like to bring attention to the presence of abnormal gaits in patients suffering from severe tardive dyskinesia. The gaits were noted as patients walked about the wards and hospital grounds. When further examined, the patients showed other signs of dyskinesias (e.g., buccolingual masticatory movements and choreoathetoid movements of the limbs). Abnormalities of posture have been reported previously (1), but we are not aware of any detailed descriptions or any comments about its frequency.

We examined 42 patients selected as having at least moderate symptoms of tardive dyskinesia (e.g., oral abnormalities and choreoathetoid movements of the limbs). Of these 42 patients, 18 (43%) had "peculiar" gaits. The gaits of 9 (22%) were considered frankly abnormal, i.e., neurological gaits, characterized as follows:

1. Broad-based gait, i.e., a wide gait with a wide arm swing and an unsteady gait. Of the 7 patients in this category, 3 had pelvic thrusting (forward movements of the pelvis on walking), and 2 walked predominantly on their heels, giving the appearance of a steppage gait.
2. What appeared to be a spastic gait, with the dragging of one foot, was seen in 2 patients.

The remaining 9 patients had what seemed to be an abnormal gait, but it was not typical of any described neurological gait.

More detailed descriptions of the gaits included unsteady, wide-based gait; marked swinging of arms; broad-based, unsteady, pelvic thrusts similar to a pregnancy strut; walking on heels; deliberate, careful wide steps; broad-based, unsteady walking on heels; stiff, unsteady gait, dragging feet; stamping type gait; and so forth. In some patients there was a cerebellar quality, while others had wide arm swing movements reminiscent of ballistic movements. A neurologist who saw 1 patient for evaluation described the patient as having "dystonic movements of the facial muscles together with occasional grunting but without rigidity of the muscles of the trunk or extremities with a mildly abnormal gait. The patient preambulated with a protrusion of the upper chest muscles and a somewhat steppage gait. The reflexes are normal and it would seem that these choreiform and dystonic movements are a result of tranquilizing drugs, i.e., tardive dyskinesia."

It should be emphasized that these observations come from a highly selected group of patients, and ... to the attention of *Journal* readers as further ... complexity of tardive dyskinesia and also as ... in the diagnosis of this condition, although ... should, of course, be made long before t... progressed to this stage.

REFERENCE

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GEORGE M. S...
RAM K. SHRIV...
Ora...

Work Stress

SIR: For the last two years we have be... number of seminars and workshops for gov... and private employees experiencing wo... have included such people as nursing ex... managers, and rank-and-file employees. I... not required to have problems, and many ... the workshops with an intent to learn som... than to seek remedial measures.

We have observed in surveys of the partic... two-thirds of work stresses stem from ot... lems. Another third report stresses that ar... sures. It would thus be useful for the he... understand sources of and coping techniq... of pressure. We found that work stresses o... at one time or another, even among partici... identified problems. We were chagrined to ... health professionals frequently were not a... to counsel people experiencing work stress... of their lack of managerial perspectives ... indicate that mental health centers are p... most other organizations in terms of the ... tensity of work stresses, and that the men... sionals themselves are often unable to cop... job stresses.

We found a number of realizations to be ... people experiencing stress at work. First is ... fact that political relationships are inherent ... tion. One must understand that power, in ... thority exist in any organization. A second ...

the notion of coping with stress using only "fight or flight" reactions is insufficient. "Fight or flight" is more appropriate to the lower animals; man is more able to "fight, flight, or take things in stride." A third realization we found helpful is the need for individuals to be willing to listen to others experiencing work stresses in the same organization. Perhaps this is consistent with the systems viewpoint, which sees stresses as cumulative and interactive. When one lowers the total amount of stresses in an organization, one is helping oneself as well. Fourth is respect for one's coworkers, even when liking others has become difficult if not impossible, or when there is a need to discipline, criticize, or disagree.

An interesting observation occurred with respect to the so-called burning-out syndrome, which seems to be almost ubiquitous, with varying intensities and reactions. The people who report being burned-out generally were describing a phenomenon of partial disengagement from their work, loss of motivation, winding down, and turning off which is probably related to a form of taking psychological flight without physical escape from the interaction.

A.J. DY, M.D.
KATHLEEN E. KAY, M.P.H.
Honolulu, Hawaii

On Assessing Violence and Dangerousness in Mental Patients

SIR: In "Crime and Violence Among Mental Patients Reconsidered in View of the New Legal Relationship Between the State and the Mentally Ill" (January 1978 issue), Larry Sosowsky, M.P.P., implied that former San Mateo County Mental Health Division patients are an extraordinary danger to the community because they have a higher incidence of arrest records for criminal behavior, including violent offenses, than the general population. He also implied that this study took place with the approval and assistance of the San Mateo Mental Health Services Division. Neither implication is correct.

Mr. Sosowsky's implied conclusion that San Mateo County patients released from Napa State Hospital subsequently committed violent crimes at a higher rate than the general population is invalid. Although a certain percentage of the patients had arrest records, no data are provided indicating the number of actual convictions, which could be the only acceptable basis for characterizing people as "violent offenders."

The majority of the arrests may well have occurred before the pivotal hospitalization, not after discharge (see p. 35 of the article, indicating a 19-month study period from June 1972 through December 1973).

The broader aspects of this report deal with arrests from January 1966 through March 1974, so that $6\frac{1}{2}$ years of the $8\frac{1}{4}$-year survey period fall before the pivotal hospitalization. The more focused aspects of the report, dealing with rates of violent crime during the "postreform" period (p. 38), counted arrests for a $4\frac{3}{4}$ year period (July 1969–March 1974), 3 years of which were before pivotal hospitalization. Indeed, 28 of the 301 survey individuals had not been discharged as of January 1, 1974 (p. 37).

What the author's data do show is that before pivotal hospitalization this survey group of state hospital admissions had a higher rate of arrests for violent offenses than their counterparts (in terms of age, sex, and race) in the general San Mateo County population, assuming the author's method of estimating those rates is correct.

It is little wonder that high arrest rates are associated with being admitted to Napa State Hospital, since some of these admissions came directly through the criminal justice system. This would include those who experienced a psychotic break while in jail, those who entered "insanity" pleas for their alleged crimes, etc. The author apparently did not segregate the data of these "penal code" cases, and they are not analyzed separately, as good research procedure would require. We estimate that approximately 12% of San Mateo's Napa State Hospital caseload during this pivotal hospitalization period were such penal code cases. Including this group clearly adds bias to Mr. Sosowsky's report.

The procedure for calculating the annual arrest rate figures for San Mateo County and several U.S. cities is not clearly described by the author and backup data are not provided. Without these data, no independent assessment of the rates he used is possible.

This article has received wide notice, was picked up by the Associated Press, and has already been cited in an editorial (1) opposing proposed amendments to the Mental Health Act of one state legislature—amendments which would tighten criteria for involuntary admission to state hospitals. The editorial cited the study's findings to the effect that the crime rate of former mental health patients has increased by a factor of three and a half since passage of California's 1969 legislation limiting the duration of involuntary hospitalization. Thus, the article is being used to perpetuate the stereotype of all mentally ill as criminally dangerous and violent persons, which they are not.

REFERENCE

1. Mental health trends boost crime rate (editorial). Tallahassee Democrat, Jan 28, 1978, p 4A

ROBERT M. RANKIN, M.D., M.P.H.
GARY M. HEYMANN, PH.D.
San Mateo, Calif.

SIR: The data on the dangerousness of the mentally ill presented by Mr. Sosowsky in the January 1978 issue of the *Journal* have important implications for public policy. Specifically, Mr. Sosowsky appears to agree that California commitment and release policies may have become too liberal to effectively protect the public from dangerous mental patients.

It behooves us to take a careful look at this research to ensure that hospitalized patients are as dangerous as the data suggest. In our view there are problems with this research that render its interpretation extremely hazardous.

Mr. Sosowsky examined the arrest rates of persons who are admitted to Napa State Hospital from San Mateo's community mental health program. This group is highly select in that a decision has been made that they are inappropriate for outpatient treatment because they are deemed dangerous to themselves or others. The criteria for assessing dangerousness no doubt include previous criminal history, and the reason for referral was probably an arrest for a criminal offence in many cases.

In view of these considerations, the only conclusion possible is that the admission screening process is working, as it should, to identify truly dangerous persons. Certainly, no inference can be drawn regarding the dangerousness of mental patients from this highly selected group. In fact, the data that Mr. Sosowsky has gathered do not even address the issue he raised in his discussion section regarding the societal costs of

current admission and discharge policies because the crimes of the patients he studied were all committed before their hospitalization.

VERNON L. QUINSEY, PH.D.
MANFRED PRUESSE, PH.D.
Penetanguishene, Ont., Canada

Mr. Sosowsky Replies

SIR: Drs. Rankin and Heymann's comments perplex me. They assert my "implied conclusion that San Mateo County patients released from Napa State Hospital subsequently committed violent crimes at a higher rate than the general population is invalid," offer points to buttress their assertion, note the wide audience my article has received, and then conclude that my article is being used to "perpetuate the stereotype of all mentally ill as criminally dangerous . . . which they are not."

Nowhere in my paper did I indicate I intended to analyze postdischarge arrest activity. At the outset I made it clear that my "report presents the results of [an] earlier one so as to match closely the methods used by Zitrin and associates." I reported findings which confirmed those of Zitrin and associates, suggested an explanatory hypothesis, and offered what continues to strike me as the appropriate warning. For Drs. Rankin and Heymann to declare my "implied conclusion" invalid without meeting squarely the conclusions I stated explicitly seems to me an effort to shift attention away from what I actually said. Certainly they can acquire the unpublished "back-up data" I relied upon, using the references in my paper, in order to conduct that type of independent assessment if they feel it is called for. In 1974 my earlier report was received by a statewide audience and also picked up by the Associated Press. Regardless, since I did not report my data in a manner that addressed the particular dimension of postdischarge arrest activity, no conclusion on this issue, valid or invalid, can yet be drawn from my published findings. I want to add here that I decided to set up table 5 in my paper to reveal prereform/postreform arrest activity rather than preadmission/postdischarge activity in part because my arrest data included only arrests occurring within 3 months of the last admission and, more importantly, in view of the hypothesis I had in mind this type of partition was crucial. Frankly, I now regret that I did not add the additional table with the postdischarge arrest activity I did analyze, although I continue to think this dimension is not essential to my preliminary paper.

Drs. Rankin and Heymann's other points do not seem to support their own assertion. For instance, they claim convictions "could be the only acceptable basis for characterizing people as violent offenders." There is not unanimity on this issue. I relied on arrests because I concur with Zitrin and associates' judgment that records of conviction are a less accurate index of criminal behavior because many offenders are found incompetent to stand trial or are taken out of the court system before that stage of the legal process takes place (1, 2). A recently released report by the New York State Department of Mental Hygiene on the insanity defense seems at first blush to provide empirical substantiation for this judgment: the use of the defense for acquittal in New York rose fourfold to 225 in the period of 1971 to 1976, up from 53 in the period of 1965 to 1971 (3). This increase occurred after the civil commitment law in New York was liberalized. I do intend, and I stated this in my paper, to analyze and report conviction rates—not beca[illegible] much fundamental insight will be added but [illegible] completeness requires it.

One criticism Drs. Rankin and Heymann[illegible] mental. Their observation seems to reflect [illegible] most observers trying to explain the high cri[illegible] ed in current studies (1, 4, 5). My asserted [illegible] this criticism, according to Drs. Quinsey and[illegible] ders interpretation of my data "extremely ha[illegible] observation is that the high crime rates repo[illegible] ably be explained to some extent by the increa[illegible] of arrested persons from the criminal justice [illegible] tal hospitals. This criticism is connected to [illegible] aspect of this entire matter—the current stri[illegible] criteria. The consequence of both of these as[illegible] samples against which current studies calcul[illegible] violence contain a larger proportion of arreste[illegible] was found in the past. It was precisely this [illegible] with the elaboration that many of the individu[illegible] ly considered nondangerous mentally ill are [illegible] from the state hospitals, that the final reviewe[illegible] used in admonishing me to demonstrate that [illegible] did not simply reflect the demographic ch[illegible] stringent admission criteria. Apparently, I fa[illegible] level of doubt to satisfy the final reviewer [illegible] None of the authors of the two letters acknow[illegible] ness of my paragraphs addressing the contr[illegible] diversion and the changes in current admiss[illegible] my theoretical control for these factors. Th[illegible] curious in the case of the letter by Drs. Quins[illegible] since they acknowledge the obligation to [illegible] look" at my research before they assert [illegible] clusion possible is that the admission screen[illegible] working . . . to identify truly dangerous p[illegible] "more liberty—more criminal activity" seem[illegible] sible explanation for some of the increase in[illegible] ty. The critical question remains for me, how[illegible]

I am aware that what economists chara[illegible] reto-optimal solution may be beyond our rea[illegible] the task which I think is presented by the ne[illegible] ship between the state and the mentally ill. [illegible] value absolutely bringing this existential c[illegible] sharp a focus as the scientific method will a[illegible]

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LARRY SOS[illegible]
[illegible]

Sodium Seconal and Methadrine for Mute Pa[illegible]

SIR: In their article "Sodium Amylobarb[illegible] ferential Diagnosis of Confusion" (January [illegible]

Nicholas G. Ward, M.D., and associates described a diagnostic technique that seems to have fallen into disuse since its popularity during World War II. I learned a similar technique from an older psychiatrist (Dr. Reynold Flom) who had been in World War II and later in the Korean War. There was a slight modification in technique: sodium seconal was used instead of sodium amylobarbitone, and 20 mg of methadrine was added. I have had fairly good results in using this approach with mute patients where the diagnosis was in doubt after the patient had been carefully examined to rule out organic etiology. The following are three case examples.

A 17-year-old high school student had suddenly become mute at home. After 3 days' hospitalization he refused to speak, although he cooperated with hospital routine. Associated signs and symptoms included insomnia, restless sleep, rigid posture, and staring off into space. There was no family history of psychiatric disorder. Following the injection of 250 mg of sodium seconal and 20 mg of methadrine, the patient began to speak and answer questions. Florid delusional material was obtained, and a mental status exam supported the diagnosis of functional psychosis. Within a few days after beginning phenothiazine medication, the patient began to speak. He made an uneventful recovery over the next several weeks and was able to return to his class at high school.

A 39-year-old evangelical clergyman visiting the Twin Cities from his home in Canada suddenly became mute, immobile, and unresponsive during a church service. The parish leaders brought him to the hospital for psychiatric care after praying over him for approximately 24 hours. History revealed that he had had a few "breakdowns" treated by his community general practitioner in Canada. During the first 24 hours of hospitalization, he did not respond to questions and refused all food and fluid. He became severely dehydrated, so intravenous fluids were started. After administration of 300 mg of sodium seconal and 20 mg of methadrine, he verbalized paranoid delusional material as well as auditory and visual hallucinations. He responded to treatment with phenothiazines and was able to return to Canada in two weeks.

A 24-year-old disheveled woman was brought to the hospital by police who had found her confusedly wandering the streets of the city at night. She was mute and refused to communicate in any fashion. Physical and laboratory evaluation were entirely normal. When further information could not be obtained after she had been in the hospital for 48 hours, 200 mg of sodium seconal and 20 mg of methadrine were administered. However, despite the medication, she remained mute. About 3 days later, the police were able to ascertain her identity from friends with whom she lived. She was a heavy user of many different drugs and had had several previous psychiatric admissions for psychosis. She did not respond to brief hospitalization and phenothiazine medication and was transferred to a state psychiatric hospital for more extended care.

These few cases indicate that the method can be extremely useful for diagnosis but is not inevitably successful. The methadrine can induce tachycardia and extrasystoles, so it probably should not be used in those with a history of cardiovascular disease. The advantage of the longer-acting sodium seconal is that interviews may be continued over an hour or two in responsive patients. As noted by the authors, slow injection is necessary to avoid overdose or laryngeal spasm. Resuscitation equipment should be at hand.

JOSEPH WESTERMEYER, M.D., PH.D.
Minneapolis, Minn.

Defining "Schizophreniform Psychoses"

SIR: I have received letters concerning the dichotomy of the broad term "schizophrenia" in typical (process) schizophrenias and schizophreniform psychoses, which I proposed in 1937 (1). I think it may be of interest to emphasize that the schizophreniform psychoses are not a collection of special types of psychoses. Only psychoses characterized by very typical initial symptoms should be diagnosed as "schizophrenias." If these symptoms are not present but the patient has, among other symptoms, hallucinations and/or delusions, the diagnosis should be schizophreniform psychosis. It was found in the International Pilot Study of Schizophrenia that in 9 countries the symptoms of "schizophrenia" corresponded so well that it is now possible to work with a transcultural symptomatology. It is of the greatest significance in research, diagnosis, and therapy of schizophrenia that all researchers use these symptom criteria when diagnosing "schizophrenia"; cases similar to this diagnosis are termed schizophreniform psychoses. In Scandinavia these psychoses are now considered as special types of reactive psychoses similar to schizophrenia.

I hope this information may be of help to researchers in this area.

REFERENCE

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G. LANGFELDT, M.D.
Oslo, Norway

"HMC" Treatment Recalled

SIR: "Methadone in Schizophrenic Rage: A Case Study" (February 1978 issue) by Gilbert H. Berken, M.D., and associates documents the use of methadone maintenance in a case of schizophrenic rage. It might be of interest to recall that narcotic drugs were used for acute schizophrenic or manic patients in rage before the appearance of barbiturates and phenothiazines. Around the time I began medical practice in the early 1960s, an older psychiatrist taught me the use of an old nostrum, HMC (hyoscine, morphine, and cacalyptus). It was available in two doses, one with a half grain and the other with a quarter grain. One dosage was all that was ordinarily required when phenothiazine medication was started at the same time; in rare instances HMC was continued for a day or two. It had the advantage of calming acutely disturbed patients without inducing the severe extrapyramidal side effects so commonly seen with high doses of phenothiazine and similar antipsychotic medications. In the narcotophobic era of the later 1960s and early 1970s, this useful treatment modality apparently fell out of favor. Now

that we are in a more dispassionate period, it might be time to reconsider judicious use of this treatment.

JOSEPH WESTERMEYER, M.D.
Minneapolis, Minn.

Phenothiazines and Night Terrors

SIR: In their report "Pavor Nocturnus in a Schizophrenic Patient: A Review and Case Study" (February 1978 issue), Malcolm N. McLeod, M.D., and Paula Fisher, M.D., reported that phenothiazines were successful in controlling the patient's chronic schizophrenic symptoms but did not control his night terrors, while diazepam, 15 mg h.s., provided excellent control of this very disturbing problem. I wish to point out that these observations were predictable in light of recent findings in sleep research. Since it is known that phenothiazines tend to increase stage 4 sleep, one should not expect them to alleviate pavor nocturnus, which is associated with stage 4 sleep. In fact, phenothiazines might well produce or exacerbate this problem, particularly when given in a single daily bedtime dose (1). Benzodiazepines are known to suppress stage 4 sleep (2) and have been shown to be effective in controlling pavor nocturnus (3). When one also considers their margin of safety, benzodiazepines should be the drugs of first choice in treating pavor nocturnus.

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3. Fisher C, Kohn E, Edwards A, et al: A psychophysiological study of nightmares and night terrors. Arch Gen Psychiatry 28:252–259, 1973

WILFRID L. PILETTE, M.D.
Worcester, Mass.

Drs. McLeod and Fisher Reply

SIR: We agree with Dr. Pilette that phenothiazines may exacerbate pavor nocturnus. Further, we did not expect chlorpromazine to control the patient's pavor nocturnus; we simply reported that these drugs did not control the night terrors. Indeed, we felt that the pavor nocturnus attacks were exacerbated by increasing doses of chlorpromazine, but this evidence was not strong enough to report. Our patient was treated several years ago, long before the report that single doses of a tricyclic or neuroleptic exacerbate pavor nocturnus (1). Thus, our observations were not as "predictable" as Dr. Pilette suggests.

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MALCOLM N. MCLEOD, M.D.
PAULA FISHER, M.D.
Chapel Hill, N.C.

False Positives in the Differential Diagnosis of [illegible] Amytal

SIR: I read with interest the recent article by [illegible] Ward, M.D., and associates, "Sodium Amy[illegible] the Differential Diagnosis of Confusion" [illegible] issue). Their conclusion that confusion wh[illegible] the use of sodium amylobarbitone probably [illegible] etiology is in agreement with my clinical exp[illegible]

However, there is one major source of pote[illegible] tives (i.e., organic confusion clearing under [illegible] dium amylobarbitone) that was not mentioned [illegible] and may be a significant source of diagnost[illegible] the normalizing effect that hypnotics may h[illegible] dysrhythmias. Detre and Jarecki (1) discusse[illegible] hypnotics may have a normalizing effect on [illegible] (1). A patient whose confusion is caused by [illegible] cerebral dysfunction may improve rather th[illegible] when given a hypnotic. Unless this is recog[illegible] mistake such a clearing as evidence of a funct[illegible] and not recognize that the hypnotic has serve[illegible] improve the organic condition (dysrhythmi[illegible] possibility exists that one of the cases des[illegible] thors as functional confusion may have been j[illegible] positive.

Their second case report described a 36-ye[illegible] a history of grand mal and petit mal epilepsy [illegible] with bizarre, agitated behavior, loose associ[illegible] ideation, and disorientation to place. Upon re[illegible] of sodium amylobarbitone I.V. the patient's [illegible] noted to clear, and he was then treated with [illegible] 30 mg of phenobarbital t.i.d. Although the au[illegible] that this represented a schizophrenic illnes[illegible] tion described also seems quite compatible [illegible] ictal psychosis which responded to the antic[illegible] erties of the intravenous barbiturate.

Dr. Ward and associates stated that "Psych[illegible] to epilepsy was ruled out, because psychosis [illegible] has been reported only in psychomotor epile[illegible] cases 'there is never a continuous spike and [illegible] as was seen in this patient." Wells and as[illegible] scribed two cases of transient ictal psychos[illegible] others from the literature which show that suc[illegible] present a varied clinical picture that can mi[illegible] hysterical, and schizophrenic psychosis as w[illegible] These were associated with continuous or ne[illegible] petit mal variant discharges.

If this patient did exhibit a continuous spik[illegible] charge, this would be characteristic of stat[illegible] amytal is described in the dosage used by t[illegible] treatment for status epilepticus (3). Therefo[illegible] initial improvement could be attributed to cer[illegible] zure activity and his continued improvemen[illegible] sulted from the anticonvulsant effects of th[illegible] rather than the thiothixene. His history of [illegible] izations may represent previous episodes of [illegible] that had also gone unrecognized.

Whether this is actually the case or not, the [illegible] positive is an important point that should [illegible] when using intravenous hypnotics for such [illegible] poses.

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STEWART SHEVITZ, M.D.
Nashville, Tenn.

Dr. Ward and Associates Reply

SIR: Dr. Shevitz's point is well taken. Transient ictal psychosis may be a source of false positives with the amytal test. Past and present improvements in the second patient we described have appeared to correlate better with the institution of antipsychotic medication than with the development of therapeutically effective barbiturate levels. However, this is only a clinical observation, and the possibility of status epilepticus in that patient still exists.

We have also considered the possibility of limbic system seizures as reported by Monroe (1) as a source of false positives. Such seizures may not give abnormal EEGs under normal testing but may still respond to intravenous amytal.

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NICHOLAS G. WARD, M.D.
DAVID B. ROWLETT, M.D.
PATRICK BURKE, M.B., CH.B., PH.D.
Seattle, Wash.

Diagnostic Guidelines for Demented Patients

SIR: In his article "Chronic Brain Disease: An Overview" (January 1978 issue) Charles E. Wells, M.D., suggested guidelines for the evaluation of the demented patient before or instead of neurological consultation; these suggestions are welcome but deserve comment. Dr. Wells recommended a CT scan as a screening procedure in his basic diagnostic battery and does not suggest examination of the cerebrospinal fluid (CSF). There seem to be both diagnostic and logistic considerations in this regard.

There are some diagnoses that can be made by CT scan even with a negative CSF exam (e.g., normal pressure hydrocephalus), but there are others in which the CSF will give positive findings with a negative CT scan (e.g., CNS infection, early metastatic disease, multiple sclerosis). In two series quoted, Marsden and Harrison (1) and Freemon (2) both examined CSF as part of their protocols. In the former study, 1 out of 36 cases of identifiable causes of dementia was obtained by CSF exam, and in the latter, 2 out of 60. Additionally, Marsden and Harrison found no vitamin B_{12} deficiency, and Freemon concluded, "Unrewarding tests included . . . vitamin B_{12} and folate levels."

The CT scan is not readily available in all areas of the country, and its cost, while decreasing in high-availability areas, must remain a consideration for some patients. Similarly, the facilities to do a lumbar puncture may not exist in many psychiatrists' offices, although enlistment of hospital outpatient facilities (as well as house staff) may be a partial solution.

It would thus be of interest to learn the reasons for Dr. Wells' exclusion of CSF examination in his suggestions, as well as his inclusion of B_{12} and folate levels.

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ALAN F. JAVEL, M.D.
Martinez, Calif.

Dr. Wells Replies

SIR: I am grateful to Dr. Javel for his comments and for the opportunity to explain more fully the reasons for my suggestions.

The most important reason why I did not and do not advise *routine* lumbar puncture for demented patients is its low yield of specifically useful information. I did not mean to suggest that I never use this diagnostic procedure or that it is not on occasion very useful, but I reserve its use for those patients whose history or clinical findings suggest that examination of the CSF may be of specific diagnostic or therapeutic value. In only 1 patient (with syphilis) out of 166 in the two series mentioned by Dr. Javel did CSF findings provide an exact diagnosis. As Dr. Freemon reported, this patient had a positive serum VDRL and an Argyll Robertson pupil, either of which would have made examination of the CSF mandatory in that patient. As Dr. Javel notes, CSF examination provides exact diagnostic information only in CSF infections, early metastatic disease, and multiple sclerosis. These disorders rarely present as dementing syndromes per se, and even more rarely without other clinical features pointing to the correct diagnosis.

Even though the diagnostic yield is low with lumbar puncture in dementia, I would probably accept its routine use were the procedure noninvasive, inexpensive, and attended by no significant morbidity. It is indeed for these reasons that I have suggested determination of B_{12} and folate levels routinely in dementia even though the yield is low. However, lumbar puncture does not meet these criteria. It is invasive, expensive (in terms of time and equipment) for both physician and patient, and attended by considerable morbidity (the pain of the procedure itself and the posttap headache).

I realize that some believe the omission of the routine CSF examination is unjustified. I am ready to change my recommendations if good evidence for such routine use is forthcoming. However, studies to date do not, in my opinion, support this position.

CHARLES E. WELLS, M.D.
Nashville, Tenn.

Depression in Chronic Schizophrenia

SIR: We read with interest "The Assessment of Depression: A Model for Quality Review of Emergency Psychiatry" by Frederic Kass, M.D., and associates (February 1978 issue). The results of this study come as no surprise to us.

In 1975 we conducted a similar study at West Valley Mental Health Clinic, an affiliate of the UCLA School of Medicine. In our study, 96 schizophrenic outpatients with clinically diagnosed "flattened" affect were psychometrically tested using the Zung Self-Rating Depression Scale (SDS) to determine specifically whether a latent underlying depression existed in a significant number of these patients. These selected patients had denied feelings of depression and did not appear to us to be depressed. The SDS was administered to each patient. Test results clearly indicated that all subjects were experiencing moderate to severe depression. Specifically, scores for the 96 patients ranged from 54–88, with a mean of 67 (SD=13.44).

Now that the existence of depression in chronic schizophrenia has "come out of the closet" we would hope that clinicians will take this into account in the diagnosis and treatment of the affect as well as the thought disorder. This should improve the overall functioning of schizophrenic patients.

Reprint requests or additional information concerning our study should be directed to us at the address below.

HARVEY AGER, M.D.
LOUIS N. MILSTEIN, PH.D.
Lafayette Clinic
951 East Lafayette
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Differential Diagnosis in Recognizing Alcoholism

SIR: We would like to share with your readers an incident at our hospital which points up the need for extreme care in differential diagnosis. Recently, five patients diagnosed as psychotic were admitted to our acute care psychiatric unit. Each patient was prescribed a major tranquilizer. One typical case report follows:

> A 52-year-old woman was admitted and diagnosed as a gravely disabled paranoid schizophrenic and placed on haloperidol, 5 mg t.i.d. On later questioning by a team consisting of a psychiatrist, a psychologist, a clinical pharmacist, and a social worker, she initially denied all but occasional use of alcohol. After more intense questioning, she admitted to very heavy drinking and three convictions for driving while intoxicated, one of which involved an accident in which she received a head injury. At this point all neuroleptic drugs were discontinued. The patient was placed on chlordiazepoxide, 10 mg t.i.d. and 25 mg h.s.; thiamine, 100 mg/day; and flurazepam, 30 mg h.s. Laboratory values were normal except for total protein 5.5 (normal=6.0–8.3) and globulin 1.9 (normal=2.0–3.5). The patient's mental condition greatly improved and she was discharged to continue her therapy on an outpatient basis shortly thereafter.

Great care should be taken in the differential diagnosis of alcoholism and schizophrenia at the time of admission because there is substantial evidence that the major tranquilizers used in the treatment of psychoses can lower the seizure threshold during alcohol withdrawal and thus possibly precipitate a grand mal seizure (1). Before one prescribes a major tranquilizer for a newly admitted patient, a procedure of very thorough questioning, evaluation, and history taking should be exercised during the initial interview to rule out the possibility of alcoholism.

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S. S
D.S. NOOR

A Question on Haloperidol Treatment for Del

SIR: Although it was timely and importa
and Research Report "Rapid Treatment of De
cally Ill Patients" by David P. Moore, M.D. (
issue) has left us with some inquiries and
Moore reports a case of a supposedly delirio
all we as readers know is that the patient on t
tal day "became agitated, struck a nurse, and
nasogastric tube," and that "physical and labo
nations failed to reveal anything that could
change in the patient's clinical state." Pres
basis, "a diagnosis of delirium of unknown
made." One could assume many reasons for
appropriate behavior. Matters important to re
history, whether a drug screen was performed,
tory examinations were done, and the doses a
treatment with morphine, diazepam, and any
tions administered during the hospitalization
withdrawal reactions to drugs are common
rium in hospitalized patients. For example,
Chan have reported a withdrawal reaction
to 8 days after discontinuing diazepam (1). S
cohol withdrawal can appear as late as 10 d
nence commences (2). Toxic reactions to d
cholinergic properties are worth mention
drugs are ubiquitous. Both morphine and d
were administered before the onset of the u
have anticholinergic properties. Haloperido
anticholinergic effects, although they are no
nounced as those of the sedating phenothia
chlorpromazine and thioridazine. Physostig
reported to be effective in treating overdose
pounds mentioned except alcohol and morp

Of equal concern is the implication that pa
ium of unknown etiology may be treated
safely with haloperidol. Certainly a delirium
ic reaction to phenothiazines, butyrophe
cholinergic compounds would not be effecti
treated with haloperidol. A better conclus
from this case report and a literature review
etiology of a delirium, other organic brain s
type of "cerebral insufficiency" is being se
symptoms with haloperidol may be conside
ment. We are certain Dr. Moore would agre
pression of any psychiatric symptom or syn
chotropic medication may forestall diagnos
of its etiology, with potentially serious or fat
(4).

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NEAL R. CUTLER, M.D.
JON F. HEISER, M.D.
Orange, Calif.

Dr. Moore Replies

SIR: I would like to respond to some of Drs. Cutler and Heiser's inquiries and concerns. I do agree with them that suppression of symptoms may forestall diagnostic measures and, in fact, stated in my paper that "Optimum management of delirium includes diagnosis and treatment of the underlying cause of the cerebral insufficiency and symptomatic treatment of the delirium itself." Space limitations precluded a full description of the diagnostic measures taken in this case, which included complete physical examination, detailed history with regard to prior drug use, urinalysis, complete blood count, SMA-18, prothrombin time, arterial blood gases, chest X-ray, ECG; EEG, lumbar puncture, and cultures of urine, sputum, and blood. None of the measures proved enlightening. The patient's systemic medications included only morphine and diazepam, the dosages of which had been unchanged for the 4 days before the onset of the delirium. Thus the only diagnosis I could make was delirium of unknown etiology.

Drs. Cutler and Heiser's point regarding the danger of treating delirium secondary to anticholinergics or to antipsychotics with haloperidol is well taken, and I did not mean to imply that it should be used in these conditions. However, I can think of few other conditions, with the possible exception of withdrawal syndromes, in which it would be dangerous to use haloperidol. As I noted in my paper, "Controlled studies are needed."

My own concern with the tone of their letter is that it might lead some physicians to withhold drug treatment in a critical situation until all diagnostic studies were done. A full diagnostic workup must be done on all delirius patients, but in those who present with potentially life-threatening behavior, as was the case in the patient I described, management must be prompt and effective. Haloperidol appears, with the exceptions mentioned above, to be the drug of choice in such situations.

DAVID P. MOORE, M.D.
Louisville, Ky.

Pretrial Commitment

SIR: We found the recent article by Jeffrey L. Geller, M.D., and Eric D. Lister, M.D., "The Process of Criminal Commitment for Pretrial Psychiatric Examination: An Evaluation" (January 1978 issue) and the "Comment" by Alan Stone, M.D., (January 1978 issue) an intriguing and timely commentary on the process of pretrial commitment in Massachusetts. In the neighboring state of Vermont, the practices of psychiatric observation are somewhat different.

Vermont's 450,000 residents are served by one state facility at Waterbury. Unlike Worcester State Hospital, Vermont State Hospital is the only hospital in the state that accepts court-ordered referrals. Therefore, our sample is truly representative of all such referrals from the entire population of alleged offenders, apart from a small minority that are done on an outpatient basis or in correction facilities. The evaluations are done by an independent forensic consultant appointed by the hospital. These psychiatrists formulate their opinions within an initial (maximum) period of 60 days. In an attempt to highlight the use of pretrial commitment in Vermont, we will briefly describe some preliminary findings of an investigation for the 5-year period of 1972–1976.

We found that in a sizable percentage of the court referrals sent to the Vermont State Hospital, there was reason to suspect a need for hospitalization and a legitimate concern for the mental status of the client. For example, 40% of the individuals referred for pretrial commitment evaluation had previously been hospitalized for psychiatric problems. At the time of hospitalization, most of these patients had been diagnosed as psychotic. We also found that the most frequent referrals, in proportion to the base rate of crimes in the state, were for serious offenses, such as arson and crimes causing harm to individuals. Another indication of the appropriateness of the pretrial commitment procedure is, as Drs. Geller and Lister point out, the number of cases that result in commitment. In our hospital, about one-third of observations end in commitment, most because of incompetence to stand trial and some because of insanity at the time of the crime and the need for further hospital treatment. In contrast, Drs. Geller and Lister found that less than 3% of their sample of referrals ended in commitment. Finally, we found that the individuals committed under these statutes did not become "forgotten" and remain for extended periods of time in the hospital. Their average hospital stay was 260 days. Following hospital treatment, all of these individuals were given supportive aftercare planning—43% had community mental health contacts (70% with individual counselors) and 11% had vocational rehabilitation.

We found, as was suggested by Dr. Stone, that judges sometimes have differing criteria for the use of the mental health laws. For instance, there was a disproportionate number of referrals from some counties with similar types and rates of crimes. We found that there were no dramatic differences in terms of previous hospitalizations, diagnosis, or disposition between these referrals and those from other counties. Thus, the most plausible explanation for this difference is the criteria used by the local court.

Furthermore, looking at the admission rates by type of admission (voluntary, physician's emergency certificate, observation) for the state hospital over a 5-year period, we noted some supportive data for Dr. Stone's balloon theory—"If one form of confinement is reduced, then another will expand." Those counties that have the highest referral rate for observations have the lowest rate for physician's emergency certificates. In addition, when the rate of voluntary admissions declines, the emergency referral rate increases. This suggests that there is a small but significant segment of the counties' population that is in need of hospitalization and will obtain these services through one process or another.

Finally, as Dr. Stone noted, pretrial commitment is sometimes used not only by the judge or state but also by defense lawyers attempting to delay the case or to otherwise benefit their clients. We found that about 21% of the referrals to the hospital were initiated by the patient or his legal representative. In such cases, it seems difficult to argue that the state is using this mechanism to sequester deviants.

Thus we found that in the state of Vermont, pretrial com-

mitment is not used only to remove noxious offenders and sequester deviants. There seem to be three reasons for referral: 1) a legitimate concern for the mental status of the alleged offender, 2) a strategy of the defense, and 3) a means for the court to seek more appropriate dispositions for alcoholics, drug addicts, or recalcitrant offenders. No doubt there are occasional abusers, but we believe these cases call for a more informed interpretation of the existing laws, not an indictment of psychiatry or the judicial system.

BRUJ M. SARAN, M.D., PH.D.
MICHAEL KLEIN
ELLIOTT BENAY
Waterbury, Vt.

SIR: Drs. Geller and Lister in "The Process of Criminal Commitment for Pretrial Psychiatric Examination: An Evaluation" (January 1978 issue) and Dr. Stone in his "Comment" helped to bring the continuing problem of pretrial commitment into the light of professional examination. Their opinions and comment once again highlight the residual social control function of the public mental hospital, a latent function that predates the role of treatment and persists in spite of criticism. The treatment functions of these hospitals have been increasingly decentralized in general hospitals and community mental health centers. However, hospitals like Worcester State Hospital remain, and they clearly serve some important residual function to the society which maintains them.

Drs. Geller, Lister, and Stone remind us that the public mental hospital is only partly a medical institution. Pretrial commitments, like civil commitments, are made by a judge. Physicians at Worcester State Hospital do not determine whether or not these committed individuals should be patients. They do not control the admission of these people or their discharge. When the "evaluation" is complete, the "patient" remains in the hospital until called by the court. Incarceration and medical treatment are subject to a complex set of governmental regulations quite apart from medical considerations.

It is ironic that simultaneous with the preparation of Geller and Lister's report, Worcester State Hospital experienced the loss of its medical directorship. Late in 1977 the position of the medical superintendent was eliminated, and Dr. David J. Myerson resigned as superintendent of Worcester State Hospital. He relinquished the governance of the hospital to a Department of Mental Health Regional Executive Committee, headed by a nonphysician. This ended almost 150 years of medical superintendency at Worcester, a tradition begun by Samuel Woodward, first President of the Association of Medical Superintendents of American Institutions for the Insane, the forerunner of the American Psychiatric Association.

This event marked both the end of an era and a return to nonmedical authority in mental hospitals. Before the nineteenth century, hospital superintendents were nonphysicians appointed by civil or ecclesiastical authorities to govern hospitals. Physicians were employees who ministered to the medical needs of patients hospitalized for a variety of social problems. The surrender of control over commitment and the decline of medical superintendency herald a return to this role for the physician and underscore the latent social control function of the public mental hospital.

It took two "outsiders" to bring this problem to professional attention. Drs. Geller and Lister rotated to Worcester State Hospital from Beth Israel Hospital for six months. Lacking prior socialization in the latent functi[on] of the public mental hospital, they were struck by the [dis]parity between the law and actual practice.

More studies like these are required. Like [these] they make that which is latent become more r[eal] remind us of the power of unspoken social forc[es] heed them or they will recur to awaken us i[n] the night.

HOWARD H. GOLDMAN, M[.D.]
Wor[cester]

Feedback for Part I of the Boards

SIR: The purpose of the American Board of [Psychiatry and] Neurology is to certify competence as well [as] areas of deficiency. Currently, written feedb[ack] on request to those examinees who fail Part [II] tifying examination. No feedback is given for [Part I].

Part I is a multiple-choice examination, so [it] be provided in a computerized format. The P[sychiatry and Neurology] provided referenced feedback in the past; [this] erenced feedback should be provided to *all* Pa[rt I] [ex]aminees.

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GEORGE MIDDLEKAUFF, [M.D.]
Chi[cago]

Dr. Rudy Replies

SIR: I am pleased that Dr. Middlekauff [of the] American Board of Psychiatry and Neurology [has] lished a feedback mechanism for Part II (the [oral] providing written information to examinees [after] very well aware of the need to provide feedb[ack] Part I (the written examination), and we are [also] arate the reporting of this examination into ca[tegories] would advise candidates of their strengths [and] However, it is unlikely that we will be able [to] erenced feedback in the near future inasmuch [as] developing an adequate bank of questions.

LESTER H. [RUDY, M.D.]
Exec[utive]
American Board [of Psychiatry]
and Ne[urology]
[Chicago]

Therapeutic Usefulness of Ergotamine Tartrate [in Narcolepsy]

SIR: We have recently encountered 4 cases [of] and periodic somnolence in which manifest[ations of] sleep attacks, cataplexy, hypnagogic hall[ucinations] sleep paralysis were markedly improved [with] tartrate only, Cafergot (each containing 1 mg [of] tartrate and 100 mg of anhydrous caffeine), and [Cafergot] lets (each containing 0.1 mg of Bellafoline, 0.[25] mine tartrate, and 20.0 mg of phenobarbital).

Subsequent follow-up in those cases has [shown]

teresting findings on day-to-day and diurnal variations of serum serotonin concentration. This letter represents a brief preliminary report of one of those cases.

A 24-year-old man with typical signs and symptoms of narcolepsy was treated with ergotamine (3–6 mg/day as tartrate), with a remarkably gratifying clinical response in several days. The patient's serum serotonin level was 70 ng/ml at 8:00 p.m. and 98 ng/ml at 8:00 a.m. on Jan. 28, 1977, and 68 ng/ml at noon and 83 ng/ml at 4:00 p.m. the next day. These levels were noticeably lower than a mean value of 148 ± 50 ng/ml, obtained from 49 normal subjects at our clinic, and remarkably similar to changes in serum serotonin concentration usually observed following administration of tricyclic antidepressants, which are considered by many to be the drugs of first choice in the management of narcolepsy (2). The pattern of diurnal variation per se did not differ significantly from the control group, and it is suggested that the variations in serum serotonin value and the therapeutic response may be related to pharmacologic actions of ergotamine. Serotonin metabolism in the brain has an important role in the hypothesis of arousal-sleep mechanisms (3), and the facts of lowered serum serotonin concentration and alleviation in manifestations of narcoleptic paroxysms do not seem to be in conflict with this hypothesis.

Although ergotamine has generally been believed to be in all likelihood devoid of significant effects on the CNS (except its activity of stimulating the vomiting center), it might be conceivable that ergotamine or its metabolite interferes with serotonin metabolism in the brain of narcoleptics, given its noticeable structural similarity to its competitive analogue, LSD. Studies designed to explore this possibility are in progress.

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YOSHIHIRO KANEKO, M.D.
YUKIO TAKAHASHI, M.D.
MOTOHISA KANEKO, M.D.
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Fukushima, Japan

Proper Knowledge of Psychotropic Medications

SIR: In their article "The Physician's Knowledge of Psychotropic Drugs: Preliminary Results" (January 1978 issue), Richard M. Gottlieb, M.D., and associates did well to show that psychiatrists and psychiatric residents had very low levels of knowledge about the physiology, pharmacology, and side effects of the most commonly prescribed drug in America, diazepam (1). However, this should in no way lead one to believe that psychiatrists are not interested in learning about proper psychotropic prescribing.

Recently, at Brotman Memorial Hospital, a survey designed by the pharmacy department to measure psychiatrists' journal preferences was sent to 160 psychiatrists on our staff. Of the respondents, 89.7% of the psychiatrists submitted that they would appreciate a "hospital" newsletter containing abstracts of important psychopharmacological issues and latest developments. The pharmacy department subsequently developed a monthly two-page newsletter to meet this request. All of the major medical journals as well as 24 psychiatric journals are reviewed for psychotropic-related articles which are then summarized and incorporated into the newsletter.

We feel, as Dr. Gottlieb and associates concluded, that the challenge of teaching proper use of psychotropic medications "may not be met in medical education today and that training programs be upgraded to meet this need." It must be remembered, moreover, that learning is an ongoing process which must be continually updated. With such a voluminous amount of psychiatric literature to read and a great number of patients to see, one can easily understand how a psychiatrist may lag behind in updating his knowledge of psychotropic drug usage. Thus, a brief hospital newsletter reviewing the current literature is one way to meet the continuing education needs of psychiatrists by helping them to keep abreast of important psychopharmacological issues.

GARY E. PAKES, PHARM.D.
Culver City, Calif.

Cinchonism in a Patient for Whom Lithium Was Prescribed

SIR: A 40-year-old banker was begun on lithium carbonate for hypomania. He was maintained on two 300-mg tablets t.i.d. A serum lithium level obtained on Monday morning was 1.0 mEq/liter. Monday evening, the patient opened a new bottle of the medication he had just obtained from the pharmacy. Tuesday afternoon, the patient thought that some fellow employees were laughing at him. Wednesday morning at 5:00 a.m. the patient awoke and began to believe that he was about to lose his job, his house, and his family because of his illness. At work, he continued to be anxious, fearful, and depressed. To his supervisor, the patient's appearance was similar to that observed 5 weeks previously, when the patient had first been hospitalized. The patient called his psychiatrist, who advised him to try to stick it out another 24 hours until his next scheduled appointment but to come in if the problem became more intense.

The mystery was solved a few minutes later when the pharmacist discovered that he had accidentally substituted 200-mg quinidine tablets for the lithium. The pharmacist reported that the quinidine bottle and tablets were similar in appearance to the lithium bottle and tablets and that the two bottles were side by side on the stock shelf. On subsequent questioning, the patient admitted to severe tinnitus.

The patient was advised to discontinue the quinidine and to see his family physician. He recovered within 24 hours and has since been asymptomatic on 1800 mg/day of lithium carbonate.

JOHN M. RATHBUN, M.D.
Cumberland, Wis.

Differentiating Grief, Mourning, and Bereavement

SIR: "The Grieving Spouse" (January 1978 issue) by Milton Greenblatt, M.D., is a useful compilation of several well-known statistical and clinical studies of grief and mourning.

One of the difficulties I encountered in reading his article, however, was the almost interchangeable use of the terms "grief," "mourning," and "bereavement." One heading in the article suggests that there might be a distinction between these terms: "Grief as a Process and the Phases of Mourning," but unfortunately this distinction is never clearly spelled out.

In my own clinical practice I have found it helpful to distinguish between grief and mourning as follows: Grief is the sequence of affective, cognitive, and physiological states that follows directly after an irretrievable loss; mourning, on the other hand, is a complex and lengthy process that begins with denial of the loss and, in its optimum course, proceeds toward the acceptance of both the external loss and the integration of multiple intrapsychic shifts (1).

The clinical distinction between grief and mourning was anticipated by Freud (2) in an addendum to his paper on anxiety in which he characterized grief as a "longingful cathexis" toward the lost object. Bowlby (3), whose work on childhood separation experiences is often quoted and summarized, also studied the literature on loss reactions in animals and noted that a substantial portion of behavior in primates who had lost siblings and parents related to seeking and retaining nurturing support from their immediate environment. Such behavior appeared to be defensive and protective and might be phylogenetically related to the heightened sympathetic response observed by Parkes (4). If the clinical distinction between grief and mourning is valid, then the process of grief would appear to serve the function of protecting the organism and preparing it for the longer process of mourning. In humans, grief might well hold the same relationship to mourning as anxiety does to the mobilization of defense mechanisms, namely, a necessary preparation.

The usefulness of such a distinction becomes apparent when applied to three conditions that Dr. Greenblatt mentioned in his article: delayed, suppressed, and pathological grief; and a fourth one described by Helene Deutsch (5), absent grief. These conditions should be understood not as isolated pathological phenomena but as abnormal or maladaptive defensive maneuvers that occur in individ[illegible] backgrounds, psychodynamics, and persona[illegible] Thus a schizophrenic might experience an ab[illegible] predominantly somatized grief reaction and [illegible] longer process of mourning, the reason being [illegible] never attained the developmental milestone [illegible] constancy. By contrast, an obsessive neuro[illegible] pass through normal grief but founder in the [illegible] ess because of excessive ambivalence tow[illegible] spouse. The main point of these examples is t[illegible] logical" nature of the process would depe[illegible] more on the preloss state of functioning than [illegible] timing, level of affective expression, verbal [illegible] gruence with social expectations observed in [illegible]

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JOHN S. G[illegible], M D
D[illegible]

Dr. Greenblatt Replies

SIR: I am pleased with Dr. Graves' perce[illegible] my article and I would like to thank him for g[illegible] such a careful reading.

MILTON GRE[illegible], M D
Los A[illegible]

BOOK REVIEWS

Psychotherapy Versus Iatrogeny: A Confrontation for Physicians, *by Nikola Schipkowensky. Detroit, Mich., Wayne State University Press, 1977, 494 pp., $30.00.*

Although this book is expensive, it is well worth the investment. It is a most interesting book divided into eight sections discussing different aspects of iatrogeny. The first section is an excellent historical review, including references to Hippocrates and Plato and emphasizing that Hippocrates recommended wise restraint in speaking about the dangers of health and life in the presence of the patient to avoid "iatropsychogeny." Schipkowensky then discusses iatrogeny in German, French, Anglo-American, and Slavic psychiatry, pointing out that there is relatively little emphasis on the pathology caused by physicians' attitudes.

He discusses hypnotism as an example of the danger of new psychotherapeutic methods and refers to a number of theories put forward by those using "magnetism." Leibault and Bernheim reported cures of 95% of their patients, while Janet had a complete recovery in relatively few (7%) of his hypnotically treated patients. (It was my good fortune in 1936 to spend some time talking with Janet about hypnotism. Perhaps part of his lack of success was his questioning attitude about hypnosis.)

In a fascinating chapter titled "The Physician and Belles Lettres" Schipkowensky describes Montaigne as "the founder of theoretical antimedicine." He then describes in considerable detail many other authors who rejected medicine in principle. He points out that "a genuine doctor is not afraid of the mirrors in which many authors want to show him his image; however, he has the right to demand a realistic reflection, both of his science and of his activity. . . . A true mirror keeps the doctor from iatrogenic and iatropathic mistakes." In referring to various literary criticisms of physicians, the author points out that all physicians are threatened by this potential and that, as a result, all physicians should be extraordinarily careful not to take advantage of their patients in any way.

The chapter on the ideology of iatrogeny provides a somewhat philosophical point of view, with particular reference to magic and its power to produce disease and even death. Schipkowensky also discusses religious iatrogeny, focusing on religious cures as well as on the development of disease through guilt concerning sins. He provides a long discussion of Freudian psychoanalysis as a "model of psychotherapeutic iatrogeny." He gives Freud credit for a brilliant mind and "cleverness in neutralizing his critics as one who functions best in adversity." He is particularly critical of the transference theory: "To interpret all interpersonal relations as transference phenomena is one of many assertions Freud made without any foundation" (p. 143).

In his section on organolocalistic (mechanistic) iatrogeny the author reviews the literature from ancient times to the mid-twentieth century in a very interesting way, giving several examples of the way in which physicians can cause anxiety symptoms by resorting primarily to apparent physical findings. In the section on character and iatrogeny he discusses personality as material (somatic) in content, ideal (psyche) in its form, biological in its origin, and social in its development. When physicians criticize each other in the patient's presence concerning diagnosis, treatment, and prognosis, they do great harm to the patient. With this we could certainly all agree.

As a logical succession to ideology, the author's next chapter deals with methods of communicating medical topics that may do more harm than good. He points out that medical students often develop symptoms discussed in lectures or at the bedside. By the same token the physician's attitude or a careless word may plant the seeds of a serious anxiety or a phobic attack. He presents many case histories demonstrating how physicians, by inadvertent comment or by presumptive diagnosis, may cause a patient to believe that there is a serious condition present. On the other hand, the author points out that some patients cannot be told the true nature of an incurable condition.

In the chapter on clinical iatrogeny the author states that anxiety neurosis "evolves" in three clinical forms: phobia, hypochondriasis, and apprehension of harm to the individual's social values. He includes neurasthenia as a pathological reaction of personality, as well as hysteria, which he describes as psychotic in pattern when its existence is questioned. He describes cases in which symptoms of these conditions resulted from an inappropriate treatment procedure carried out by physicians. Patients with psychosomatic and physical diseases who hear inappropriate comments by a physician may also develop iatrogenic reactions that cause their symptoms to be more severe. He believes that "psychogenous death" occurs more frequently than is usually believed, especially when the patient feels he or she is in a hopeless situation.

Under the heading "Anti-Psychiatry" Schipkowensky describes in detail Rosenhan's article "On Being Sane in Insane Places" (1). If Schipkowensky were alive today I am sure he would have included the ABC television program "Madness and Medicine" as an unfortunate production with a substantial antipsychiatry bias. In such instances there is just enough substance for the psychiatrist to be aware of some important facts, as he or she would from Rosenhan's article, yet enough misinformation that it could result in serious harm. This section of the book is of particular interest, especially the author's comments about Szasz and his writings.

In the section on the occurrence of iatrogeny in psychotherapy the author devotes a great deal of space to both direct and indirect criticism of psychoanalysis, especially Freudian psychoanalysis. He presents a long and interesting discussion of "psychogeny" in the home and school under the heading of "didascalogeny and mathetogeny." He emphasizes the selection of good teachers because, with parents, the teacher represents the greatest influence on a child at a time when his or her personality is most easily molded. Schipkowensky describes cases in which teachers have exercised a serious negative influence, resulting in psychiatric symptoms. He discusses parental influences under the head-

ing of "Oppressive and Excessive Didascalogeny" and gives many examples of this type.

In the chapter on liberating psychotherapy the author summarizes the aims of psychotherapy as follows: 1) healing of pathological reactions of the patient's personality, 2) stimulation and the making of a harmonious personality in pathological developments, 3) rehabilitation after changes of character and/or disability in process psychoses and in somatic disease, 4) readaptation of antisocial and asocial individuals to the community of life, 5) rehabilitation of all mental patients, aiming at complete maturing of their personalities by widening their spiritual horizon, 6) alleviation of the condition of incurable patients and spiritual assistance to those doomed to long agony. This is a very interesting chapter and well worth reading.

In Schipkowensky's very short chapter "Conclusion" he makes a particularly important point: "Whatever the kind of treatment the psychic influence of the physician on healthy individuals as well as sick ones cannot be ignored" (p. 476). This influence, of course, can be either positive or negative. He points out that iatrogeny is sometimes embedded in a healer's personality; therefore, physicians should always have as much self-knowledge as possible and should judge the patient's personality so that they can treat the patient in accordance with his or her personality peculiarities.

This book is a very important contribution and should be available to every physician as well as to others who do psychotherapy. I would express one serious criticism of the book—Schipkowensky's bias against psychoanalysis. This book would be valuable even if used only for reference to the very extensive bibliography Schipkowensky provides. Although written in a very readable way, the book is so extensive that it should be used as a reference work.

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RAYMOND W. WAGGONER, M.D., SC.D.
Ann Arbor, Mich.

Female Psychology: Contemporary Psychoanalytic Views, *edited by Harold P. Blum, M.D. New York, N.Y., International Universities Press, 1977, 438 pp., $22.50.*

Freud (1) concluded his lecture on femininity with the statement that his knowledge of the subject was incomplete. He advised those who wished to know more about it to wait until science could offer deeper and more coherent information. On the basis of new information, the contributors to this volume have evaluated and revised Freud's theories and offer perspectives of their own.

Several of the authors in this book disagree with Freud's assumptions that female identity is not established until the child has passed through the phallic phase and that her entrance into the oedipal phase at about age three is based on her discovery that she has no penis. They cite evidence that feminine gender identity is established early in life and is determined by the interaction of anatomy, biology, parental attitudes, and the child's capacity to organize internal and external experiences. There is some disagreement as to the relative importance of these factors, especially the impact of the absence of the penis, although all agree that it is a significant factor in the resolution of the young girl's [illegible] plex.

Galenson and Roiphe differ with Freud [illegible] which a girl becomes aware of genital differ[illegible] lieve this occurs during the pre-oedipal st[illegible] and 18 months), but they agree with him that [illegible] castration anxiety are major organizers of t[illegible] women. To Kleeman, parental attitudes and [illegible] nitive development are most important. Sto[illegible] importance of parental sex assignment and o[illegible] titudes, and he also notes the impact of a b[illegible] in the establishment of femininity. From [illegible] tions of three little girls, Parens and associa[illegible] the girl's oedipal phase is not initiated by c[illegible] but is associated with gender-related chan[illegible] with the nature of her object relations, and [illegible] dispositions. The wish to have a baby, the [illegible] pendent on the wish for a penis.

Clower's observations about pleasurable [illegible] girls during latency challenges Freud's ass[illegible] relinquishment of pleasure in her body is a [illegible] the establishment of femininity. According [illegible] transition from adolescence to womanhood [illegible] resolve her ambivalent ties to her parents i[illegible] her wish to be a mother. The tendency to c[illegible] hood in contemporary society is a reality fac[illegible] es the difficulty in establishing a feminine [illegible] berg presents suggestive evidence that duri[illegible] ner genital sensations, which in early life ha[illegible] a sense of femininity, are reintegrated inte[illegible] femininity.

Societal attitudes toward women are com[illegible] to understand. Lerner suggests that the adul[illegible] the child of the female genitalia is evidence [illegible] discomfort associated with female sexuality [illegible] women, especially of their procreative cap[illegible] ject of a paper by Jaffe. Although society's [illegible] women is clearly ambivalent, the tendency [illegible] has gained support from Freud's characteri[illegible] as inferior, narcissistic, and masochistic. S[illegible] ception to this view, demonstrating its origi[illegible] oretical and social biases. Blum examines [illegible] female masochism and moral inferiority ar[illegible] they are derived from unwarranted notio[illegible] sexuality. For Grossman and Stewart, penis [illegible] phor that can reflect disappointment and ra[illegible] at various levels of psychosexual developm[illegible] as a symptom to be analyzed and not a m[illegible] ultimate conflict. The interaction between [illegible] and other determinants of femininity is [illegible] Ticho's paper on autonomy and Applegarth[illegible] bition.

In his discussion of female orgasm, Moor[illegible] ing point that conclusions about the nature [illegible] ences based only on direct observations are [illegible] It is necessary to know the psychic repres[illegible] ated with the orgasm in order to understand [illegible] servations about sleep orgasms support the [illegible] may have varied meanings. Barglow and Sc[illegible] cal of the literature on women that relies too [illegible] ological data and does not consider intrapsyc[illegible] explain the conditions of women.

The comprehensive view of the multiple [illegible] female psychology presented in this book [illegible] definitive statement about what female ps[illegible] should be is not possible. The many facto[illegible]

femininity can be integrated into varied patterns. This perspective can facilitate the efforts of the therapist to assist the female patient in resolving her conflicts and establishing an identity that suits her. Although this book offers many valuable insights, the tentative nature of some of the reported findings suggests that there are areas for future investigation.

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REBECCA Z. SOLOMON, M.D.
Hartford, Conn.

Marihuana Research Findings: 1976. NIDA Research Monograph 14, *edited by Robert C. Petersen, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1977, 225 pp., no price listed.*

Physicians, particularly psychiatrists, are often asked for expert opinions on matters with social and political consequences. If the issue is outside our expertise, we should say so; if we are merely ignorant, we should either discover the known or investigate the unknown. When we speak, we should be careful to distinguish our expert opinions from our voices as independent citizens.

One area in which psychiatrists profess expertise is the impact of drug use on physical and psychological health. Because of the strong and emotionally held opinions in this area and its importance in many lives, a broad and secure data base is essential, particularly with regard to the so-called recreational drugs. Until recently, the public debates about decriminalization of marijuana were lacking in information. However, as *Marihuana Research Findings: 1976* evidences, the debate has spawned numerous projects (many supported by the National Institute on Drug Abuse) and, at last, some hints of useful information.

Following an introductory and summarizing chapter by the editor, this volume contains detailed reviews of the epidemiology of marijuana use; its chemistry, metabolism, toxicology, pharmacology, and acute and chronic effects on unlearned and learned animal behavior; and its effect on humans, with special attention given to the genetic and immune systems as well as to therapeutic potential. These are written in clear, simple English by active, expert researchers. Even the descriptions of metabolism and toxicology were clear to me.

The use of marijuana has markedly increased since the late 1960s, spreading from the counter culture to a broad cross-section of youth—a majority of those 18 to 25 years old have used the drug. It appears to have moved from fad to cultural pattern. If the user avoids such tasks as driving while intoxicated, occasional or minimal regular use of marijuana as a social euphoriant has not been shown to have lasting deleterious effects. Nor does marijuana use inevitably lead to use of other, more dangerous illicit drugs or to violent or criminal behavior. As with the use of alcohol and tobacco, frequent use of high potency compounds may be associated with deleterious effects that have not yet been demonstrated. In addition, idiosyncratic physiological and psychological responses may occur.

This brief summary does not do justice to the rich content of this slim, readable book. It is an important volume for all of us in our roles as experts and citizens. I eagerly await the next annual report.

WILLIAM A. FROSCH, M.D.
New York, N.Y.

Cognitive-Behavior Modification: An Integrative Approach, *by Donald Meichenbaum. New York, N.Y., Plenum Press, 1977, 293 pp., $14.95.*

Cognitive behavior modification deals with the intrapsychic or cognitive aspects of behavior therapy and is a recent, important thrust within the field (1–4). This area of behavior therapy is so new that it has not been included in two recent comprehensive reviews of psychiatry (5, 6).

The book is a theoretical study heavily dependent on Meichenbaum's own research. The first three chapters concern the technique of self-instructional training. This technique has been successfully applied in the treatment of hyperactive, impulsive children, socially isolated children, and adult schizophrenic patients. In the fourth chapter Dr. Meichenbaum stresses the importance of cognitive factors in systematic desensitization, modeling, assertive training, and aversive conditioning. He presents evidence suggesting that the traditional behavior therapy techniques would have their clinical effectiveness improved if cognitive techniques were integrated into the treatment plan.

The technique of stress inoculation training (a method used to enable patients to better deal with stress), which has been successfully applied to phobics, alcoholics, and socially withdrawn children, is discussed in chapter five. In chapter six, Dr. Meichenbaum compares his cognitive restructuring techniques with those of Aaron Beck, Albert Ellis, Thomas D'Zurilla, and Marvin Goldfried. In chapter seven there is a discussion of internal dialogue (what the patient says to himself or herself) and its importance in both behavior change and the human body's physiological response to stress. Dr. Meichenbaum presents his cognitive theory of behavior change in chapter eight, and in the last chapter he provides a method of assessing patients that uses internal dialogue and images (fantasies) as the primary data.

Dr. Meichenbaum sets forth not only firm experimental support for his techniques but also a practical guide as to how to assess and treat patients within the framework of cognitive behavior modification. He offers convincing evidence for adding cognitive behavior modification skills to our present regimen.

This book is not a comprehensive review of the literature, and little time is spent on thought stopping, covert sensitization, covert reinforcement, covert extinction, and attribution theory. Despite these omissions, the book is well written and offers additional clinical strategies that all psychiatrists could use.

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GEORGE W. MIDDLEKAUFF, M.D., PH.D.
Chicago, Ill.

Essays in Neurochemistry and Neuropharmacology, Vol. 1, *edited by M.B.H. Youdim, D.F. Sharman, W. Lovenberg, and J.R. Lagnado. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1977, 195 pp., $21.50.*

The explosion of research and knowledge in the neurosciences has been paralleled by an abundance of new books and journals to record recent successes and provide portents of the future. It is no longer uncommon to convene international conferences for the major purpose of producing one or more volumes, to be rapidly published by photo-offset, lest the information contained be obsolete before it reaches its audience. It is in this context that *Essays in Neurochemistry and Neuropharmacology* must be considered, especially since it is the first of what will be a very large series indeed, judging from the articles planned for future volumes.

The projected series is aimed at researchers and graduate students "who wish to familiarize themselves with current specific problems in neurochemistry and neuropharmacology." The aim is for each article to represent a more personalized statement by the author(s), including the author's own views on trends for future research.

This first volume consists of six articles. J.M. Saavedra discusses the role of methylating enzymes in the synthesis and catabolism of compounds relevant to the nervous system. G.G.S. Collin deals with the role of the sulfated amino acid taurine in the mammalian CNS. H. Thoenen and A. Otten write on the molecular basis of transsynaptic regulation of the synthesis of macromolecules. K.F. Tipton and associates describe the role of aldehyde metabolites of biogenic amines in the brain. A.J. Kastin and associates discuss CNS and pituitary effects of hypothalamic peptides and melanocytes-stimulating hormone. Finally, E.D. Bird and L.L. Iversen review neurochemical research in Huntington's chorea.

Judging from these six articles, the editors were only partially successful in achieving their aims. The depth of scholarship is by and large appropriate for an introduction to new areas for advanced students or researchers. References are limited in most articles, but they are still abundant enough to enable an interested reader to get a good start on the primary literature. (Examination of the references, however, reveals that most of the articles were probably completed in early 1976—two years ago. A publication lag of this magnitude hampers but does not eliminate the worth of this type of volume, which is aimed at giving readers a good start in a field.) The articles contain elements of the kind of essay the editors hoped for, but they are usually only appendages to the main business of reviewing the literature. However, some of the more personal remarks are of a high order, especially Thoenen and Otten's discussion of the role of transsynaptic regulation in the adaptation and plasticity of the nervous system.

All of the reviews are well written, and it is probable that the book would be useful to readers interested in this field. The authors and editors have been highly successful in producing readable, clear, concise statements [illegible] without getting lost in minutiae. They also [illegible] uncertainty.

Future volumes in the series will include [illegible] on the biochemistry of mental illness, [illegible] chemistry, and mode of action of indoles [illegible] cinogens. These might be of greater inter[illegible] psychiatrist who would like an introduction [illegible] search in the neurosciences.

HERBERT Y. M[illegible]

Family Constellation: Its Effects on Personality and Behavior, 3rd ed., *by Walter Toman, Ph.D. New York, Springer Publishing Co., 1976, 325 pp., $[illegible] paper).*

Dr. Toman outlines the natural environment in which brothers and sisters influence each other's personality development, describes basic trait clusters for ten sibship positions, gives guidelines for generating additional categories, and expounds a fascinating and heuristic theory of complementarity (attraction of unlikes) of sibships in relation to marriage, parent-child interactions, and [illegible] achieves quantification of the measures of sibship [illegible] conflict and of the effect of loss of a family member, expressing, in algebraic terms, the degree of [illegible] flict and the disorganization of constellations, [illegible] so provides a complete and valuable interview guide for securing sibship constellation data. Three case histories illustrate how the personality trait clusters obtained by retrospective sibship constellation and related data (e.g., change of family, change of residence, illnesses, secession, religion) can be used in consultation regarding [illegible] vocational goals, and general effectiveness [illegible]

Because of weaknesses in the research design and statistical analyses of the experimental data [illegible] ing the theory of attraction of complements [illegible] gories and because of the diametrically opposite [illegible] experiments by other researchers, the most [illegible] sumption one can make from this book is [illegible] and direction of the influence of complementary [illegible] constellations on the compatibility of marriage [illegible] ships have not been firmly established. Descriptions of personality trait clusters demand the use of unambiguous, precise, culturally unbiased adjectives, a difficult task. Toman's colloquial style often fails to meet [illegible] determined, he has derived the trait clusters [illegible] ing clinical observations rather than experimental [illegible] to support his statements. The procedure of [illegible] ality traits of sibship categories in sibships [illegible] from combinations of constellations of sibships [illegible] warranted. The discussion of the effect of [illegible] spacing between siblings is in each case inadequate.

Toman's treatise on family constellation would have been improved considerably had he included descriptions of child-rearing practices by which, for example, the youngest child could be given some of the leadership qualities of the eldest and the eldest some of the "freedom" of the youngest. Educators would appreciated specific discussion of preventive methods, and psychotherapists would have welcomed discussion of differential treatment approaches [illegible] ship constellation effects.

From the studies and experiences of a lifetime, Toman has produced a volume full of sagacious insights, suggestive material for the psychotherapist and educator, and heuristic hypotheses for the behavioral science researcher.

HERMAN J.P. SCHUBERT, M.E., PH.D.
Buffalo, N.Y.

Current Developments in Psychopharmacology, Vol. 4, *edited by Walter B. Essman and L. Valzelli. New York, N.Y., SP Books (Spectrum Publications), 1977, 264 pp., $25.00.*

This volume is one of a series of annual publications consisting of invited contributions from distinguished scientists and clinicians actively working in different areas of psychopharmacology. Volume four has nine chapters; four deal with basic or preclinical issues and five deal with clinical problems.

One of the most informative chapters of the book is that of Simon on the distribution and properties of opiate receptors in the CNS and the identification and characterization of endogenous ligands, endorphin peptides, for opiate receptors. On the basis of these new findings, systematic studies have been directed to provide evidence for a qualitative or quantitative change in opiate receptors during chronic treatment of animals with opiates, but the question of how opiate receptors in the brain are affected by the development of tolerance and physical dependence has remained open. In the meantime, as discussed by Blasig and Herz, precipitated opiate withdrawal in rats seems to offer a suitable alternative approach for studying problems concerning physical dependence. Since the problem of precipitated withdrawal depends in part on the kind of antagonist applied, the precipitated withdrawal method may become a useful link between molecular and behavioral approaches to the study of opiate dependence.

The possible role of catecholamines in the regulation of various central nervous functions and behavior has been reviewed with emphasis on information important for the mode of action of some psychotropic drugs. Although further support to the view that schizophrenic symptoms may be mediated via dopamine-carrying neurons is given, Engel and Carlsson, on the basis of some new experimental data, put forward the hypothesis that there is a common neurochemical basis, involving the catecholamines, for the primary reinforcing properties of dependency-producing drugs. Considering that the catecholamine neurons form a great number of neuronal pathways in the brain, the majority of which originate in the lower brain stem projecting to widespread areas of the brain and the spinal cord, it is not inconceivable that catecholamines are involved in the regulation of various behavioral and physiological responses and conditions, such as drug dependence and schizophrenia.

Identification of an enzyme capable of using cofactor 5-methyltetrahydrofolate in producing tetrahydro-β-carbolines from tryptamine analogs resulted in a new interest in the biological effects of naturally occurring (e.g., harmine, harmol, harmaline, harmolol, yohimbine) and synthetic (e.g., 2,9-dimethyl-β-carbolinium iodide, usually referred to as DMCI) β-carboline substances that in the past had been studied only because of their hallucinogenic, tremor-inducing, and monoamine-oxidase-inhibiting effects. Although the enzymatic formation of tetrahydro-β-carbolines remains to be demonstrated in vivo, Ho believes that the new findings reopened the possibility that a psychotic state may result from a shunt of the metabolic pathway of serotonin and the consequent formation and accumulation of a psychotomimetic compound.

There is a short and concise chapter on recent advances in genetics with psychopharmacological significance. Of particular clinical relevance are findings related to genetic factors that seem to influence the response to alcohol and the therapeutic responsiveness to lithium salts, monoamine oxidase inhibitors, and tricyclic antidepressants. On the basis of the steadily accumulating data, Mendlewicz asserts that "a knowledge of the contributions of genetic and environmental influences in drug metabolism and drug response is an essential research area that will hopefully lead to the discovery of predicting factors in the determination of success or failure of treatment."

This volume also contains a comprehensive review of the toxicology of tricyclic antidepressants by Fiori, a critical review of long-term treatment with neuroleptics by Shepherd and Watt, a long-overdue review of the pharmacological management of hyperkinesis by Silbergeld, and a review of current trends in the psychopharmacology of aging by Sathananthan, Ferris, and Gershon. According to Silbergeld, "hyperkinesis" denotes a diverse group of behavior disorders in which some order can probably be imposed by separating hyperkinetic children according to their pharmacological response. Amphetamine- and methylphenidate-responsive hyperkinesis is one of the most systematically studied subtypes, and extrapolation from the experimental pharmacology of these drugs has led to the original hypothesis that decreased monoaminergic function is the underlying defect at least in certain hyperkinetic states. The fundamental problem with this hypothesis is that the mechanisms of experimental hypermotility and the reversal of amphetamine's behavioral stimulation are not compatible. The conjunction of altered response to amphetamine and hypermotility has not been experimentally produced except in one case of chronic exposure to inorganic lead, which is associated with reduced acetylcholine release and increased catecholamine functions. Whether this new model of hyperkinesis offers any advantages over the existing ones remains to be seen.

In summary, this convenient volume gives a good selection of articles on current developments in psychopharmacology. Although written primarily for the teacher and research worker rather than for the student, some of the chapters contain material that could help the practicing psychiatrist in his or her everyday work.

THOMAS A. BAN, M.D.
Nashville, Tenn.

Primer of Lithium Therapy, *by James W. Jefferson, M.D., and John H. Greist, M.D. Baltimore, Md., Williams & Wilkins Co., 1977, 206 pp., $10.50 (paper).*

Although not quite ready to say, "If you know lithium, you know psychiatry," one is impressed by the number of areas of psychiatry involved in the study of this simple element. Not only is there an abundant literature detailing its physiology and pharmacology, lithium research and clinical use have also stimulated investigation in such apparently diverse areas as genetics and forensic psychiatry. Its clinical use is prompting a thoughtful reassessment of time-honored assumptions about diagnostic groupings as well as of the relationship between diagnosis and etiology.

This clearly written book, packed with carefully evaluated

summaries, is a noteworthy addition to the exponentially increasing lithium literature. Drs. Jefferson and Greist have done the clinician a singular kindness by providing well-organized information from their Lithium Consultation Computer Program. The summaries of relevant articles are valuable in their own right, but the authors' analysis is invaluable.

Among the many positive aspects of this book is the thorough discussion of differential diagnosis and its importance in terms of predicting both therapeutic response and lithium toxicity. The authors update the recommendations concerning the physical and laboratory evaluation of the patient for whom lithium is considered. They go beyond the usual warnings about drug interactions and offer pragmatic suggestions about the management of patients with complex pharmacological needs. The section on the treatment of lithium intoxication is up-to-date, discarding approaches that, although theoretically sound, have proven of little practical value. The authors' suggested guidelines are particularly welcome because few of us have obtained first-hand experience in the treatment of a large number of patients with severe lithium intoxication.

In a book such as this there have to be a few things that one would like included or developed more extensively. The authors indicate that lithium may be an effective antidepressant in certain instances, but they do not comment on the studies comparing lithium with tricyclic antidepressants. The chapter on the general pharmacology of lithium is not of the same quality as the other sections, in part due to its brevity. However, several good references on this subject are given.

I have one, perhaps gratuitous warning: in our search for certainty in an uncertain world, we long for *the* definitive text. This one is excellent, but it will soon be dated as additional information becomes available about the long-term benefits and hazards of this important pharmacological agent.

CHARLES W. BOREN, M.D.
Hartford, Conn.

Psychiatric Medicine, *edited by Gene Usdin, M.D. New York, N.Y., Brunner/Mazel, 1977, 486 pp., $20.00.*

This book is a consolidation of papers presented at the annual scientific meeting of the American College of Psychiatrists in February 1977. To some browsers the title would suggest a general textbook of psychiatry. In this instance, however, "psychiatric medicine" encompasses the topics of psychosomatic or psychophysiological psychiatry, with special attention to the field of consultation and liaison psychiatry.

The book is divided loosely into sections such as Psychobiology and Liaison Psychiatry, but it is basically a series of papers on special topics. This gives an initial impression of fragmentation. It is only after concluding the book that one is aware that, taken as a whole, it is a good overview of historical trends and special topics of current interest. Portions of the book would appeal only to special interests, but the reader who completes the whole volume has a working knowledge of the field and is equipped to explore in depth a number of significant areas. As Martin states in his epilogue, "This meeting of the College reported no one great discovery, but maturely presented concepts of illness and health based on sound data."

As a general psychiatrist, I found several papers most useful. Weiner's introductory pa... chobiology of Human Disease: An Overview ... encyclopedic survey of the theoretical and ... tempts to link psychic factors to organic il... concluding that adaptive failure is a key f... bibliographies are comprehensive, but We... and Reiser are especially helpful in docume... ground of their subjects.

Greenhill's paper, "The Developmen... grams," would be a valuable resource for t... administrator setting out to establish a ... Greenhill's historical approach to the su...ject... acknowledgment of problems and failed pr... feeling that a realistic approach and an acce... are useful in the organization of such prog a... cal position on the role of the psychiatrist ... ganic illness is interesting. Perhaps at var... opinions, he rejects the feasibility of a "psyc... rolled into one" and concludes that the ... demonstrate that his primary identification ... try, with its rich content to be applied at mu... cal, biosocial, and medical end points."

In his chapter, "The Challenge of Newe... ings," Reiser proceeds from older theories ... date the reader's knowledge about the mech... in psychosomatic illness. He surveys the w... 20-year periods and concludes with a thi... current state of the art.

John Wing's paper, "The Management o... in the Community," was presented as the S... Award Lecture. Wing begins with an extre... torical review of the concept of schizophre... tempts to arrive at a diagnostic standard F... extensive experience in the management of t... ic patient outside the hospital, exploring se... that make for success or failure of manage... sizing the necessity for reasonable expecta...

My selection of papers for comment sh... from the considerable interest of other chap... It is essentially a collection, but reflective ... whole book identifies it as an effective revie... is less tightly organized than a textbook on ... would be, but this is perhaps offset by the fa... is free to pursue subjects of special interest

Rev... *Ph...*

Behavior Therapy in Clinical Practice: Deci... cedure and Outcome, *by Ernest G. Poser P... Ill., Charles C Thomas, 1977, 164 pp., $13...*

Over the last decade more than a dozen ... been published on the theory and practice o... py. Some of these stress basic principles ... others stress specific behavior therapy pro... grams as they apply to psychiatric disorders ... able volume has a different focus and organ... describes eight specific inpatients who we... behavioral unit he directed at the Douglas H... Montreal, Que., Canada.

Each case is described in some detail, ... tient's family background, personal history, ... the problem dealt with. Dr. Poser then prov...

analysis of the patient's difficulty, which may include behaviorally oriented tests, a delineation of the treatment goals, and a description of the various treatment methods employed. An important aspect of these case reports is an evaluation of the effectiveness of each treatment component along with a follow-up evaluation, which in most cases extends from 5 to 10 years. The latter is unusual in case reports and of great value, even though these are selected cases.

This book gives the reader a perspective on the outcome of behavior therapy with patients sufficiently impaired to require hospitalization and also provides information on possible cycles in the patient's maladaptive behavior and the need for early intervention should relapse occur. The eight cases provide the author an opportunity to describe the application of 15 different behavioral procedures, since some patients required more than one approach. These range from relaxation methods and modified systematic desensitization for the treatment of phobic states to aversive conditioning and differential social reinforcement for the amelioration of such socially disapproved behaviors as sexual exhibitionism.

This volume is not a substitute for a text that develops in a systematic and comprehensive way the basic principles of learning and social conditioning. Nor does it take the place of a text that describes the chief behavior therapy procedures for the treatment of various disorders. Rather, it complements such texts by providing in greater detail than one usually finds a description, behavioral assessment, plan of treatment, and follow-up of a number of patients. This conveys vividly to the reader the essence of a behavioral approach to specific problems impeding the restoration of functioning in hospitalized psychiatric patients.

JOHN PAUL BRADY, M.D.
Philadelphia, Pa.

Clinical Biofeedback: A Procedural Manual, *by Kenneth R. Gaarder, M.D., and Penelope S. Montgomery, M.S. Baltimore, Md., Williams & Wilkins Co., 1977, 222 pp., $12.95 (paper).*

This is one of the first books about the practical aspects of clinical biofeedback training. Gaarder and Montgomery have had an active clinical biofeedback practice at the University of Texas at San Antonio. In this book they draw on their extensive clinical experience to give us a working account of the biofeedback training process.

This book aims to provide enough information on biofeedback training to enable practitioners to incorporate biofeedback techniques into their clinical work. The book begins with a consideration of the scientific foundations of biofeedback in experimental laboratory studies. It then moves on to practical considerations, including choice of patients for biofeedback training, structuring a treatment program, assessment of ongoing therapy, and treatment strategies. Two final chapters are of particular interest to readers with relatively little background in electronics. One deals with instrumentation techniques and the other with the function, safety, and specifications of various types of biofeedback equipment. Three useful appendices are included: a list of professional resources in the biofeedback area, questionnaires routinely used in training, and a script for relaxation training.

Gaarder and Montgomery's major theme is that successful biofeedback treatment depends on many factors common to other therapy approaches. Biofeedback is not presented as some form of fancy electronic panacea. Rather, it is discussed as a method of behavioral intervention that needs to be placed in the context of psychotherapy. For example, the need to develop a therapeutic relationship with the patient is strongly emphasized. In addition, practitioners are encouraged to take into account the patient's environment and such related factors as secondary gain, competing behaviors, and the role of significant others. The stress placed on the complexity of clinical biofeedback training is refreshing. Most practicing biofeedback clinicians realize that, as Gaarder and Montgomery point out, the simplicity of such training is more illusory than real.

A second theme receiving a great deal of attention in this book is the need to use biofeedback in combination with other procedures, such as relaxation or autogenic training. The authors note that a combination of procedures often facilitates transfer of skills learned in the laboratory to the patient's natural environment.

One question about the entire clinical biofeedback area that applies equally to this book is, Can the physiological changes achieved through biofeedback training be attributed to feedback itself? When a broad comprehensive approach to patient care such as the one outlined in this book is employed, it is difficult, if not impossible, to determine which elements of the treatment package are responsible for behavior change.

The major weaknesses of this book stem primarily from the fact that biofeedback is a relatively recent area of research and practice. Data on many of the problems discussed in this book (e.g., selection of patients) are only now becoming available.

Many clinicians will find this book helpful as a practical handbook. In and of itself, the book does not provide the detailed coverage of the biofeedback area needed by someone seriously interested in applied clinical research. The book is clearly and concisely written and could be used as a training manual for technicians or research assistants. It meets the pressing need for making practical information on biofeedback training readily available to the medical community.

The promise of biofeedback is that it may provide a mechanism for the behavioral treatment of medical disorders. It is clear that the practical problems delineated by Gaarder and Montgomery need to be systematically addressed before this promise is fulfilled.

FRANCIS J. KEEFE, PH.D.
Boston, Mass.

Ethical Issues in Sex Therapy and Research, *edited by William H. Masters, M.D., Virginia E. Johnson, and Robert C. Kolodny, M.D. Boston, Mass., Little, Brown and Co., 1977, 219 pp., $12.50.*

This book is the report of a conference sponsored by the Reproductive Biology Research Foundation held in January 1976. The participants were all distinguished scientists, moralists, and mental health professionals. The topics were wide-ranging but largely focused on the concerns involved in research on sexual matters as well as the moral and ethical problems in sex therapy. It is a significant contribution to our understanding of the burgeoning issues that have emanated from the epochal contributions of the sex researchers, from Freud to Masters and Johnson, that have revolutionized the attitudes of the practitioner as well as the public with regard to sex behavior. The widespread use of the techniques of sex

therapy that have grown out of these developments raises a number of moral and ethical issues in the practice and training of sex therapists. Who should be a sex therapist and how should he or she be trained? What are the problems of anonymity, confidentiality, informed consent, and insurance coverage? Many far-reaching consequences have grown out of advances in dealing with sexual dysfunctioning. This volume touches on all of these matters and deals with them intelligently and cogently.

It was generally agreed at the conference that research in sex behavior does not differ in any real sense from research or experimentation in other biochemical or behavioral contexts. It requires the full understanding of the issue of informed consent and the other safeguards of responsible investigation. This means a valid research design that is aware of the prevailing cultural attitudes toward sex behavior. There was a range of viewpoints expressed by the participants on all of these questions.

There was also agreement on the unique situation in sex therapy for the possibility of patient-therapist interaction to be complicated by sexual activity. Everyone strongly agreed on the unethical and destructive quality of sex between patient and therapist and the failure or inability of either patient or therapist to become aware of the transferential or countertransferential subtleties in such relationships. Nothing but a total injunction against such activity will encourage respect and trust in the treatment process.

The issue of training was also dealt with at the conference, and the need for adequate attitudinal growth as well as technical competence was stressed. The requisite background of the therapist depends on the nature and intensity of the sexual dysfunction. With minimal dysfunction, education or simple behavioral modification may suffice, but in the more extreme disorders, characterological inventories and biochemical studies may be required, necessitating sophisticated medical and psychological skills.

This was a trail-blazing conference and will surely be repeated at regular intervals. The report should be required reading for any practitioner who has the slightest brush with sexual problems in his or her practice.

LEON SALZMAN, M.D.
Washington, D.C.

Hero, Artist, Sage, or Saint? A Survey of Views on What Is Variously Called Mental Health, Normality, Maturity, Self-Actualization, and Human Fulfillment, *by Richard W. Coan. New York, N.Y., Columbia University Press, 1977, 316 pp., $20.00; $6.50 (paper).*

Professor Coan has written a scholarly, readable work that examines the evolution and elaboration of the basic ideas about the ideal person or the optimal personality. He examines such basic dichotomies in the traditions of Western thought as intellect versus emotions, individualism versus love, and materialism versus spiritualism, which we have inherited from such diverse sources as the ancient Greek philosophers, Christianity, the Renaissance, and the Enlightenment. He then reviews the ideas of the optimal person as found in the writings of the major figures in the history of modern psychology—Freud, Jung, Alder, Rank, and Fromm, to name just a few.

He presents a review of his own research, which involves a multivariate analysis of the optimal personality realm. He argues that all the ingredients of our concepts of the ideal condition can be reduced to five basic mode[illegible] fillment: 1) efficiency in the intellectual, soci[illegible] realm, 2) creativity, 3) inner harmony, a co[illegible] tioning of all parts of one's being, 4) relatedne[illegible] tion toward positive interaction with others, a[illegible] dence, the experienced dissolution of one's se[illegible] uality. His data and logical considerations po[illegible] of polarities in these categories, often neces[illegible] choice between mutually desirable but incom[illegible] All in all, we are in his debt for this schol[illegible] catholic presentation of the currents of th[illegible] have contributed to our ideas about what co[illegible] personality function.

FRANCIS J. K[illegible]
[illegible]

The Gay Academic, *edited by Louie Crew. [illegible] Calif., ETC Publications, 1978, 427 pp., $15.[illegible]*

This is a collection of essays by 27 men a[illegible] define themselves as "gay academics." Mo[illegible] them are members of college faculties, seve[illegible] students, and 3 are ordained members of [illegible] book is divided into 10 essentially disparat[illegible] ering general academic issues, history, libr[illegible] guistics, literature, philosophy, psychology, [illegible] ology, science, sociology, and political scie[illegible] According to the editor, the book "is intended as a sam[illegible] scholarship spawned in the Gay Liberation M[illegible] is often the case in collections of this kind, [illegible] quite uneven, and some of it is of very pa[illegible] such as the scholarly chapter presenting [illegible] Richard Burton's terminal essay in his transl[illegible] bian Nights or the short literary essay on Ha[illegible] the words "casque" and "cask" in several [illegible]

Some interesting differences in points of [illegible] gay liberation movement emerge in some of [illegible] Stanley contributes a rather shrill antim[illegible] ionated chapter (e.g., "If people could be [illegible] wanted to be, without the intense social co[illegible] would be lesbians and homosexuals") [illegible] cooperation between lesbians and homosex[illegible] several other essays emphasize the imp[illegible] working together for common goals. Edgar [illegible] tributes a charming and witty chapter in wh[illegible] things, he comments on the difficulties he ha[illegible] an advocate of the concept that male virgi[illegible] with a reasonably happy life. His "consis[illegible] est" in sex has been treated "as the unfo[illegible] homosexuals and heterosexuals alike. [illegible] France's comment that "chastity is the [illegible] versions," Friedenberg observes wryly, "[illegible] takes all kinds, doesn't it?"

Perhaps the most interesting chapter (fo[illegible] Crampton's moving and scholarly essay on [illegible] from Leviticus to Hitler" documenting the [illegible] homosexuals have been brutalized and murd[illegible] ancient Hebrews around 550 B.C. and the[illegible] later with Christianity's coming to powe[illegible] rope. Genocidal laws against homosexuals [illegible] criminal codes of France until 1791, of En[illegible] and of Scotland as late as 1889. Both male a[illegible] sexuals were subject to capital punishmen[illegible] the American colonies, but the death penal[illegible]

revoked after the Revolution. The most recent episode of gay genocide cited by Crompton was in Hitler's Germany, when more than 50,000 homosexuals in Germany alone were arrested and sent to perish in concentration camps. When those seized by the Nazis in occupied countries are added, estimates of the number of homosexuals who died "from illness, neglect, medical experiments, and the gas chamber" range from 100,000 to more than 400,000.

The section on psychology contains only two chapters, one by De Cecco and Shively on "Conflicts Over Rights and Needs in Homosexual Relationships," and the other by Mark Freedman (now deceased) on "Toward a Gay Psychology." The former deals with some of the conflicts encountered in homosexual relationships and the use of negotiation as a way of ensuring the democratic rights of the individuals concerned. Freedman's article claims that there are "at least four major areas in which gays often function better than nongays." He defines these as 1) inner direction/centering (discovering and living by their own values), 2) sex roles (a wider range), 3) sexuality (more openness, sensuality, and freedom), and 4) identity/self-disclosure (more candidness and less defensiveness, at least among homosexuals who have "come out"). He concludes with the hope that out of the experience of emerging gay groups a special gay psychology will develop that will contribute to a better understanding of the gay community.

In summary, *The Gay Academic* is a potpourri of uneven quality. With the exception of a handful of chapters, it will be of only moderate interest to most psychiatrists.

JUDD MARMOR, M.D.
Los Angeles, Calif.

The Family Crucible, *by Augustus Y. Napier, Ph.D., with Carl A. Whitaker, M.D. New York, N.Y., Harper & Row, 1978, 293 pp., $11.95.*

This book is a delightfully novel way of presenting family therapy—novel in several senses of the word. Augustus Napier has devised a new method admirably suited for his intended purpose of capturing the "feel" of family therapy carried out with Carl Whitaker. With literary skill and psychological sophistication, Napier has made real the therapy with one family; the actual dialogue and episodes are a composite from several families, selected to illustrate the processes of the Whitaker approach to family therapy. Paragraphs and chapters on basic concepts, theory, and methods of family therapy clarify and highlight but do not disrupt the absorbing tale of the primary family. In my view, the fictionalized narrative succeeds in providing a more accurate portrayal of family therapy with Whitaker and Napier than could a factual account of any single, inevitably atypical case. This narrative is far more readable and vivid than would be a bare-bones presentation of abstract or technical principles.

Although Whitaker's personal qualities as a family therapist are widely known through his many presentations at workshops and symposia, his emphasis on growth through personal struggle is greatly illuminated by the longitudinal view of this case description. This presentation makes room for describing the context and timing of his therapeutic interventions and hence should help make the rationale of his "experiential" approach more accurately understood than is possible from vignettes.

Whitaker's therapy is marvelously packed with dilemmas and paradox. On the one hand, he draws unabashedly on the power accorded the grandfatherly old-time physician in order to win the initial "battle for structure" in the therapeutic relationship. On the other hand, having provided the security of this framework, he respectfully waits for the family to take the initiative and responsibility in accord with their readiness and ability to begin to change. While exerting great personal strength and firmness, he relies on a cotherapist relationship to free himself—and the family—for expressions of fantasy, warmth, and what he disarmingly calls "craziness."

In the cotherapy described here, both Whitaker and Napier make contributions that are independent but complementary, distinctive but supportive. Unlike most present-day psychotherapists, Whitaker and Napier maintain an interest in both intrapersonal and interpersonal levels of functioning, both of the individual as a person and the family as a system. While focusing on the immediate experience in the therapy itself, the authors bring to life a historical perspective toward transgenerational origins, for example, through direct "consultations" with families of origin. Most valuably, this narrative gives a sense of therapy as a process with phases, moving in a spiral course from family unit to individual, from one person to another, from present to past, and from playful good humor to surges of violent intensity.

Professional readers need to be warned that this volume misleadingly appears to be addressed to the general public. It differs from our usual publications in that it is highly absorbing and readable. It also explicitly discusses the gains and hazards of family therapy from the perspective of family members, who are given an inside look at the difficult, complex, and sometimes scary process. Nonetheless, I hope that clinicians of diverse persuasions will enter into this book and may glimpse the special gains that await therapists who take the risks of engaging themselves in this approach to family therapy.

LYMAN C. WYNNE, M.D., PH.D.
Rochester, N.Y.

A Reassessment of the Concept of Criminality: An Analysis of Criminal Behavior in Terms of Individual and Current Environment Interaction. The Application of a Stochastic Model, *by Eggert Petersen. Copenhagen, Denmark, Munksgaard (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 134 pp., $32.50.*

There is an inclination to deplore evidences of increasing criminality today. In the main, this evidence consists of reports of conventional offense rates, which are derived from counts of the number of offenses occurring within a specified area during a stipulated period, measured against the estimated number of residents of the area, usually obtained from standard census data. Leaving aside questions concerning the validity of such data, it should be clear they have limited usefulness in the development and/or assessment of theories of criminality, although they are often used to support the contention that the criminal justice system is not very effective in modifying or controlling criminality.

This scholarly study of criminality among men in the Danish Navy hypothesizes that criminality does not characterize the individual but is, rather, the effect of interaction between the individual and his environment. To test his thesis, Petersen examines criminality against the backdrop of the military society and the special characteristics and aims of its judicial

system. On the basis of a comprehensive review of past studies of criminality in the military, he draws the interesting conclusion that "military criminologists in general seem to regard military criminality as a phenomenon characterizing the criminal individual per se."

In his search for a more dynamic understanding of criminality, the author analyzed data relating to sanctions, service records, results of military psychological tests, biographical data, job satisfaction inventories, and the characteristics of service posts. Within these parameters, the investigation focused on a large number of variables relating to individual servicemen and the service posts to which they were assigned. The job satisfaction inventory that the author devised deserves special recognition as a device for the measurement of individual attitudes.

In his analysis of factors in the individual and in his service posts that might predispose to criminality, the author concludes that

> there is no single factor at the bottom of naval criminality, nor only a complex of personal factors, but there is an interaction between person and service post which it has proved possible to describe by means of a probability model, which comprises a person parameter (sanction pronity) and a service post parameter (sanctioning inclination) that both vary greatly.

In the last chapter, the author makes some practical suggestions for combating naval criminality, mainly in the area of improving job satisfaction at various service posts.

This monograph is recommended as a model for multifactorial investigation into the basis for criminality. No doubt it has implications for the study of other behavioral phenomena as well.

CHARLES E. SMITH, M.D.
Chapel Hill, N.C.

Drug Abuse in Pregnancy and Neonatal Effects, *edited by José Luis Rementeria, M.D. Saint Louis, Mo., C.V. Mosby Co., 1977, 284 pp., $18.95.*

This is a valuable and welcome book. It is a comprehensive overview of current knowledge concerning the effects of a number of drugs of abuse on the developing fetus and on the neonate. Its primary appeal and value will be to the obstetrician and neonatologist working with pregnant addicts and other drug abusers. Other medical and paramedical personnel working with substance abusers will also find the answers to their own and their clients' questions about the effect of a variety of abused substances on the fetus and newborn in this volume.

Although it is a useful compendium, it has its weaknesses. It is an edited volume, with individual chapters authored by experts. Like many such volumes it suffers from a lack of tight editorial control. It is repetitious, and some chapters could have been combined. For example, chapter 21, "Disposition and Planning Long-Term Follow-Up of the Infant After Release from the Hospital," and chapter 23, "Social Agencies and Their Role in Working with the Infant and Family," go over similar material with only a slight difference in perspective.

There are divergent opinions between authors, and in some instances the editor highlights these, but in other instances the contradictions are neither pointed our nor resolved. Thus one cannot browse through this v... sult a single chapter and be sure that he or she ... authoritative viewpoint. In chapter 3 there is ... omission under cardiac complications. Alth... endocarditis as a complication of heroin abus... no mention is made of fungal endocarditis. Ad... a rare finding, but it is well-known and ar... limited to mainlining heroin addicts. It is imp... it is frequently overlooked and the appropri... procedures are not ordered.

Finally, it would have added to the value o... editor had provided a summary chapter for ... major sections. Such a synthesis would have ... hicle for dealing with contradictory material ... provided a welcome overview of the relat ve ... the findings of the individual authors, mak... more valuable reference.

JAMES T. B...
Cinc...

Studies of the Effectiveness of Treatments fo... Vol. V: Evaluation of Treatment Outcomes ... DARP Admission Cohort, *edited by S.B. ... Dwayne Simpson. Cambridge, Mass., Ballin... Co. (J.B. Lippincott Co.), 1976, 512 pp., $25...*

This volume is the fifth research study de... the Drug Abuse Reporting Program (DARP) on th... treatment for drug dependence by the staff of ... Behavioral Research of Texas Christian Univ... porting program was established by NIMH... studied include 43,943 admissions to 52 feder... treatment agencies from June 1, 1969, thro... 1973, and followed for the fifth year. The ... books in this series studied the admissions ... present volume reports on the 16,000 adm... 1973.

The chapters in each of the six parts of the ... the segments of the research, including the ... treatment, death rates, causes of death for ... and the validation of drug abuse treatment ... amplify work on methadone maintenance, th... munities, outpatient drug-free therapy, and ...

The researchers, using quite complex an... recorded the multiple studies in great deta... samples of patients. The results demonstr... who remained in treatment tended to show p... "no clear cut superiority of any one treatm... any modality was found for all types of pa... adone maintenance showed the best during-tr... ioral outcome record in reducing opioid use ... and increasing employment and productive ... other finding was that "all treatments ... tients in a therapeutic setting for the intende... ration and completion." In striving to anal... authors admit that sometimes "more questi... than were answered."

A good section of this volume from the v... clinical psychiatrist is the study of death rate... death for opiate addicts in treatment. The ov... was 13 per 1,000, with violence and factors ... use contributing to 72% of the deaths. Anoth... tion deals with a method for the classificatio... treatment programs on the basis of scoring ...

an attempt to establish an evaluation technique that can be useful for future research.

All in all, this book is not easy for the clinical psychiatrist to digest. It is lengthy, difficult to follow at times because of the overuse of acronyms, and full of tables (I counted 156); it also places much emphasis on the details of procedures, most of which are geared to the researcher.

MANUEL M. PEARSON, M.D.
Philadelphia, Pa.

Handbook of Physiology: A Critical, Comprehensive Presentation of Physiological Knowledge and Concepts, Section 1: The Nervous System. Vol. I: Cellular Biology of Neurons, Parts 1 and 2, *edited by John M. Brookhart and Vernon B. Mountcastle; Eric R. Kandel, volume editor; Stephen R. Geiger, executive editor. Baltimore, Md., American Physiological Society (Williams & Wilkins Co., distributor), 1977, 1,182 pp., $135.00.*

These two books provide a systematic introduction to the functioning of nerve cells that will be invaluable as a reference work for the graduate student and the scientist who is interested in a modern review of ongoing research in cellular neurobiology. The volumes are not meant for or applicable to the casual reader interested in a review of new knowledge in cell biology of neurons.

The important new findings described in these volumes include the analysis of fine structure of the neuron, further progress in understanding the excitation and conduction in neurons, and the recent findings in synaptic transmission. Perhaps of most importance to psychiatry are the applications of techniques of cell biology to the study of systems of neurons. It is hoped that such studies might lead to an understanding of how the sensory and motor systems are organized and how the neuronal circuits of behavior are modified by learning.

The two parts of volume 1 are an unsurpassed systematic introduction to functioning of nerve cells. They should be available to serious students of the nervous system.

DONALD W. MULDER, M.D.
Rochester, Minn.

Medicolegal Aspects of Hospital Records, 2nd ed., *by Emanuel Hayt, LL.B. Berwyn, Ill., Physicians' Record Co., 1977, 506 pp., $19.80.*

This is a good book, presenting a complicated subject in a systematic and understandable manner. It can be a useful guide for medical records librarians, hospital administrators, and physicians as long as the reader keeps in mind that "no book can be a substitute for competent legal advice" (p. xii).

It covers much more than its title implies. It shows the reader how a lawyer reads and interprets a medical record and how points that medically trained people may consider trivial can come to haunt them if not properly attended to. It provides a guide to courtroom procedures and illustrates the various situations in which medical and other hospital records are used in and by the courts. The systematic presentation and comprehensive discussion of the records hospitals are required to keep are made more interesting and understandable through examples from actual cases decided in the courts. Another important aspect of the book is that it incorporates the requirements of the Joint Commission on Accreditation of Hospitals.

On the negative side, the book was written for use in general hospitals and makes only a few references to situations peculiar to psychiatric institutions. The recently adopted statute dealing with the confidentiality of medical records of alcoholic and drug addicted patients is not included. Although the table of contents is exhaustive, the index leaves a lot to be desired.

MARIJAN HERJANIC, M.D.
St. Louis, Mo.

Handbook of Studies on Depression, *edited by Graham D. Burrows. Amsterdam, the Netherlands, Excerpta Medica (Elsevier/North-Holland Biomedical Press), 1977, 420 pp., $68.00.*

Norman Sartorius, Director of the World Health Organization's Division of Mental Health, has stated that epidemiological surveys have disclosed that on any given day there are one hundred million people with clinically recognizable and treatable depression. He also forecasted that in the next decade there will be a substantial rise in the incidence of this affective disorder. It is not surprising, therefore, that physicians, especially psychiatrists, have become more eager to learn more about depression and its treatment and that the number of books devoted to the etiology, biochemistry, manifestations, and treatment of depression has escalated in the past few years.

This well edited book is the latest and one of the best of the multidisciplinary treatises on depression. It is divided into four sections: the first is devoted to classification, phenomenology, and etiology; the second to treatments; the third to current research; and the last to bereavement, suicide, cardiac effects of tricyclic antidepressants, and the management of drug overdose.

The contributors to this volume are acknowledged international authorities. Each lucidly, concisely, and comprehensively covers his or her area of expertise. The result is a very readable book with a wealth of up-to-date information on all aspects of depression. Having read many of the recent books on depression, I can forthrightly state that, although expensive, this volume offers the reader so much data to increase his or her knowledge of the illness and its therapies, it will be judged a worthwhile investment.

FRANK J. AYD, JR., M.D.
Baltimore, Md.

Psychiatric Diagnosis. The C.M. Hincks Memorial Lectures, *edited by Vivian M. Rakoff, M.A., M.B.B.S., Harvey C. Stancer, Ph.D., M.D., and Henry B. Kedward, M.A., M.D. New York, N.Y., Brunner/Mazel, 1977, 228 pp., $15.00.*

Psychiatric Diagnosis consists of nine papers written by different authors. Reading these consecutively makes one aware of a level of difficulty beyond that considered in each paper; namely, how can the approaches be reconciled? Drs. Spitzer, Sheehy, and Endicott present a clear outline of the multiaxial diagnostic system of *DSM-III*. Dr. Stoller, discussing psychodynamic diagnoses, points out that a diagnostic system which neglects the meaning of a symptom to the

patient is incomplete. Dr. Reitan provides an excellent review of the utility of neuropsychological concepts and testing. He demonstrates that cerebral lesions can give distinct syndromes influencing behavior and that the diagnosis of organic brain syndrome, as conventionally used, cannot describe the multiplicity of specific deficits that may occur.

Dr. Roy Grinker, Sr., describes the necessity of making diagnoses that reflect all available and pertinent data. Dr. Wing's article includes so many issues that it is in itself the basis for a book. A noteworthy concept is that of basing all diagnoses (including multiaxial diagnoses) on objective or at least overt clinical findings; a system for doing so is described. Dr. Wender reviews the concept of a schizophrenic spectrum. He is not evasive in defining often ill-defined terms, such as chronic schizophrenia, borderline schizophrenia, "reactive" schizophrenia, "schizoid" phenomena, and dementia praecox.

Dr. Winokur provides a review of the genetic determination of major affective disorders, the separation between unipolar and bipolar disorders, and the likely existence of two types of unipolar depression. He also gives a concise review of genetic markers for affective disorders. Dr. Van Praag presents the need for making diagnoses in terms of differentiation of biological substrates, particularly in relation to catechol and indole metabolism when defining affective disorder. This dimension, as outlined, is independent of etiology, symptoms, and prognosis. Dr. Feinstein, an internist and epidemiologist, gives an elegant summary of the various patterns of inference applied to available evidence in diagnostic procedures.

The quality of the individual articles is good. Each remains in its area rather than becoming global or criticizing other systems. The articles on neuropsychological deficit, the schizophrenic spectrum, and genetic patterns in affective disorder will be of direct interest to clinicians. This book demonstrates that adequate psychiatric diagnosis requires descriptions in several frameworks and gives the term "multiaxial" meaning and relevance.

MARTIN H. KEELER, M.D.
Houston, Tex.

Religious Encounters with Death: Insights from the History and Anthropology of Religions, *edited by Frank E. Reynolds and Earle H. Waugh. University Park, Pa., Pennsylvania State University Press, 1977, 244 pp., $14.50.*

In view of the current popularity of books dealing with various aspects of death and the dying process, this volume of essays from ethnographic and historical viewpoints is most welcome. The book is primarily the outgrowth of papers presented at the 1973 convention of the American Academy of Religion. Eleven of the 15 essays are based on papers presented at that meeting. The original papers were modified to make the scholarly material more accessible to the nonspecialist reader.

The editors' introduction gives coherence to the essays by considering death in terms of "disjunction, continuity, transition, integration, and transformation." They make the valuable point that although varying cultures produce a "dazzling array of differences," there is an "undeniable core of unity" in the way people deal with death.

Two comparative studies begin the collect[ion] by Mircea Eliade, gives an arresting and helpfu[l] sketch of underlying mythologies of death. [The] paper on death and pilgrimage seemed less [rel]evant to the general topic. The remaining papers concern [...] death in various cultural traditions. Benjamin [...] "Death, Kingship and Royal Ancestors in [...] onstrates the real archetypal connection of [...] royal forebears as a major source of General [...] er. Franke Neumann's paper on the gatekee[per] tures vividly the spiritual tribulations and [...] death of Moctezuma II (Montezuma) at the [...] empire.

The first of the 4 essays on Indian religion[s], Long, describes the way in which Vedic and [...] duism dealt theistically and nontheistically [with] questions concerning human mortality. David [...] ticle on medieval Hinduism presents an [...] cussion of the conquering of death and the [...] al rebirth by fantasying and meditating in [...] death. The enormously elaborate rituals [...] ent-day Hindu families to assure their [...] place in heaven are depicted in David Knipe's [...] Pangborn outlines the Parsis' death rituals [...] connects their death anxiety to the anxiety [...] gard to their probably dying culture. In [...] Shōsan's samurai and Zen death techniques [...] gives a lively account of a samurai warrior [...] who attempted to live in a state of "perpetual [...] of his own death."

Those of us reared in the Western tradition[s] God will feel on more familiar ground in the [...] Judaism. Wolfrom Herrmann considers the [...] mortality as it is described in the Old Testament [...] dilemma of meeting death and yet not actual[ly] movingly portrayed by Pesach Schindler in [...] the Polish Hasidim during the Holocaust.

In the section on Islam, Alford Welch sum[marizes] jor points in the Koran on death and dying [...] some of the Koran's death symbolism with sl[ight] in Shī'a communities and their relevance [for] the terror of death for community members [...] validate the history of this minority Islamic [...] cussed by Earle Waugh.

Glen Davidson's contribution presents inte[resting] on death in three seventeenth-century N[ew] groups—the Huron Indians, the Catholic [...] Puritan New Englanders, but his conceptua[l] forced. In his essay on Lincoln's assass[ination] Capps explicates a tragic mythic dimension o[f] presidency and considers the effect of the [...] frontier mythology.

In general, I found the essays in this book [...] well written except for some undefined terms [...] tion of different cultural attitudes and ri[tuals] death is lucidly set forth. I think that *Religi[ous] with Death* can fulfill the editors' aim of help[ing] develop a "personal 'language' of death." [For] therapist this volume can be additionally val[uable] him or her understand patients in their [...] mortality in themselves and others.

RICHARD C[...], M.D.
New H[aven], Conn.

This Month's Reviewers

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Behavior Modification: Principles and Clinical Applications, 2nd ed., *edited by W. Stewart Agras, M.D. Boston, Mass., Little, Brown and Co., 1978, 301 pp., no price listed (paper).*

The Alcoholism Digest Annual, Vol. 5, 1976–1977. *Rockville, Md., Information Planning Associates, 1978, 491 pp., $35.00.*

Systems Change Strategies in Educational Settings, *by Richard I. Arends and Jane H. Arends. New York, N.Y., Human Sciences Press, 1977, 120 pp., $9.95.*

Professionals and Paraprofessionals, *by Michael J. Austin, Ph.D. New York, N.Y., Human Sciences Press, 1978, 290 pp., no price listed.*

Counseling in Correctional Environments, *by Lawrence A. Bennett, Ph.D., Thomas S. Rosenbaum, Ph.D., and Wayne R. McCullough, Ph.D. New York, N.Y., Human Sciences Press, 1978, 94 pp., $6.95.*

A Bibliography in Dynamic, Familial and Social Psychiatry, *by John Birtchnell and Sheila Hafter Gray. York, England, Society of Clinical Psychiatrists, 1978, 135 pp., £1.00 (paper).*

The Psychiatric Halfway House: A Handbook of Theory and Practice, *by Richard D. Budson, M.D. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 274 pp., $10.95.*

The Sex Role System: Psychological and Sociological Perspectives, *edited by Jane Chetwynd and Oonagh Hartnett. Boston, Mass., Routledge & Kegan Paul, 1978, 182 pp., $6.75 (paper).*

Visual Evoked Potentials in Man: New Developments, *edited by John E. Desmedt. Oxford, England, Clarenden Press, 1977, 556 pp., $50.00.*

Schizophrenia: A Philosophical Reflection on Lacan's Structuralist Interpretation, *by Alphonse de Waelhens; translated by W. Ver Eecke. Pittsburgh, Pa., Duquesne University Press (Atlantic Highlands, N.J., Humanities Press, distributor), 1978, 256 pp., $15.00.*

Short Forms of the MMPI, *by Thomas R. Faschingbauer and Charles S. Newmark. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 179 pp., $15.00.*

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Fear: Learning to Cope, *by Albert G. Forgione, Ph.D., and Richard S. Surwit, Ph.D., with Daniel G. Page. New York, N.Y., Van Nostrand Reinhold Co., 1978, 175 pp., $9.95.*

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Epidemiological Approaches in Child Psychiatry, *edited by P.J. Graham. London, England, Academic Press, 1977, 397 pp., $19.50.*

The Psychological Society: A Critical Analysis of Psychiatry, Psychotherapy, Psychoanalysis and the Psychological Revolution, *by Martin L. Gross. New York, N.Y., Random House, 1978, 354 pp., $10.95.*

The Sex Atlas: A New Illustrated Guide, *by Erwin J. Haeberle, Ph.D., D.A. New York, N.Y., Seabury Press, 1978, 500 pp., $25.00.*

Neuroleptics and Schizophrenia. Handbook of Psychopharmacology, Vol. 10., *edited by Leslie L. Iversen, Susan D. Iversen, and Solomon H. Snyder. New York, N.Y., Plenum Press, 1978, 242 pp., $25.00.*

The Anatomy of Hallucinations, *by Fred H. Johnson. Chicago, Ill., Nelson-Hall, 1978, 226 pp., $16.95.*

The Placebo Effect in Healing, *by Michael Jospe. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 162 pp., $15.95.*

A Primer of Drug Action, 2nd ed., *by Robert M. Julien. San Francisco, Calif., W.H. Freeman & Co., 1978, 278 pp., $14.00; $6.50 (paper).*

History of Behavior Modification: Experimental Foundations of Contemporary Research, *by Alan E. Kazdin, Ph.D. Baltimore, Md., University Park Press, 1978, 445 pp., $24.95.*

Psychology of the Planned Community: The New Town Experience. Community Psychology Series, Vol. 4, American Psychological Association, Division 27, *edited by Donald C. Klein, Ph.D.; Daniel Adelson, series editor. New York, N.Y., Human Sciences Press, 1978, 182 pp., $11.95.*

Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

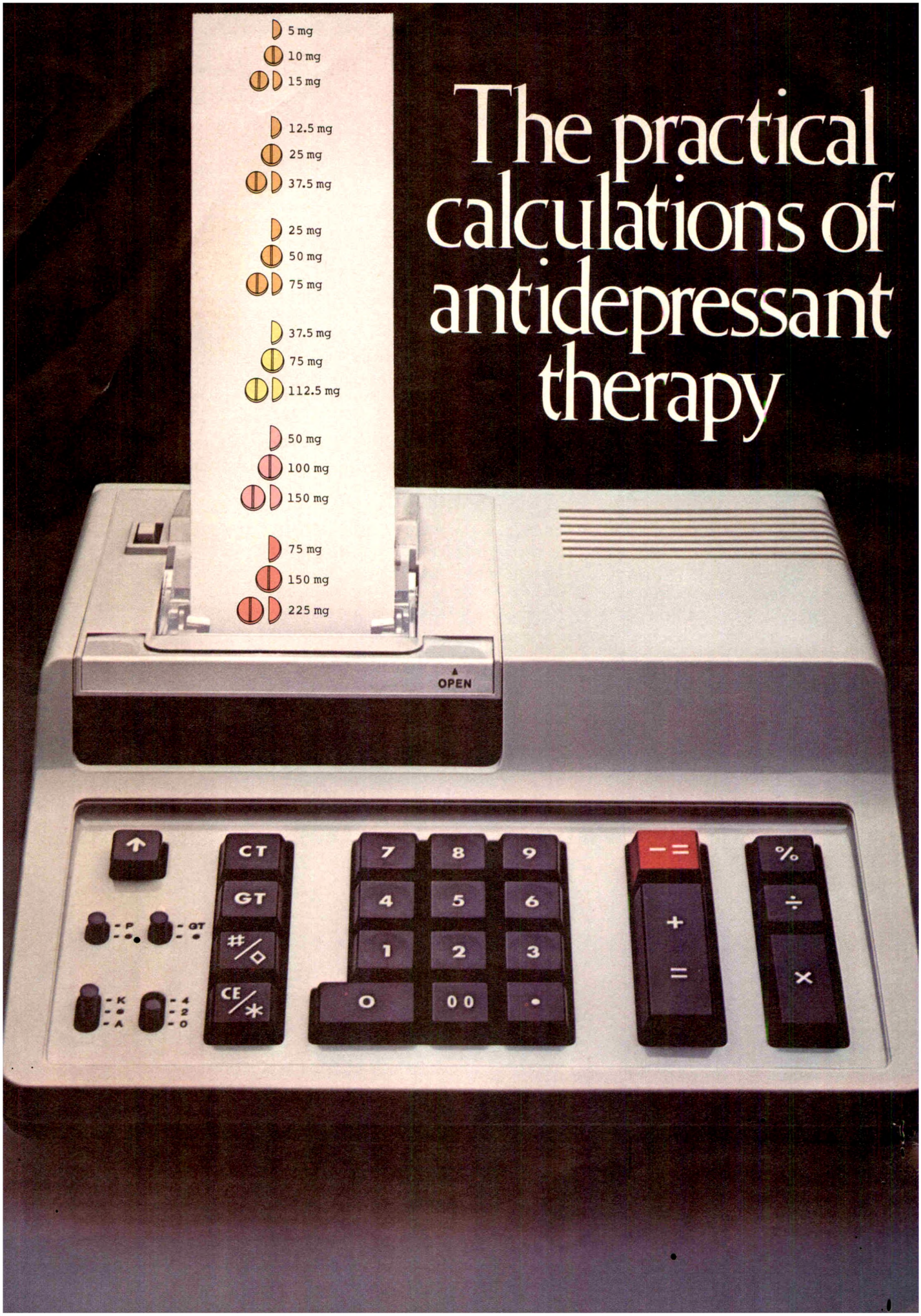
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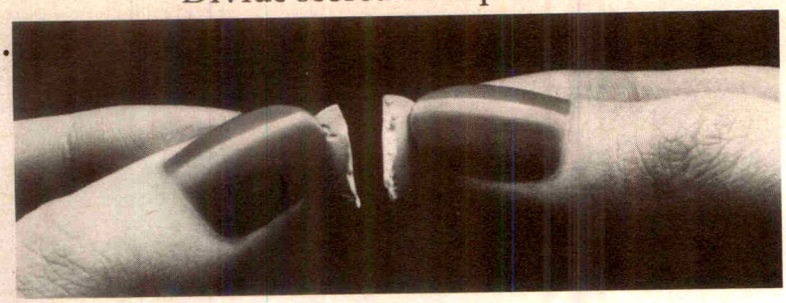
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Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

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Highly effective in a wide range of both acute and chronic psychotic disorders,[1-7] such as hallucinations, delusions, suspiciousness, hostility, mania.

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Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects.[2,4-6,8,9] Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Although some instances of drowsiness have been reported, marked sedation is rare.

Common side effects easily controlled.[1,2,4-7] Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. 34:113 (Feb.) 1973. 2. Ayd, F.J., Jr.: Med. Sci. 18:55 (Oct.) 1967. 3. Rapp, M.S.: Can. Psychiatr. Assoc. J. 15:73 (Feb.) 1970. 4. Howard, J.S.: Dis. Nerv. Syst. 35:458 (Oct.) 1974. 5. Gerle, B.: Clin. Trials J. 3:380 (Feb.) 1966. 6. Abuzzahab, F.S., Sr.: Psychosomatics 11:188 (May-June) 1970. 7. Darling, H.F.: Dis. Nerv. Syst. 34:364 (Oct.-Nov.) 1973. 8. Snyder, S.H., et al: Science 184:1243 (June 21) 1974. 9. Stimmel, C.L.: Dis. Nerv. Syst. 34:219 (Apr.) 1976.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

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A tasteless, odorless, colorless liquid concentrate for better patient compliance: 2 mg per ml.

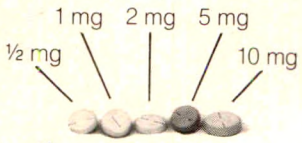
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A rapid-acting injection for psychiatric emergencies: 5 mg per ml,* with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



tablets

5 tablet strengths for convenience in individualizing dosage:



HALDOL® (haloperidol)

tablets/concentrate/injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

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when moderate anxiety
or agitation accompanies depression

dual-action Triavil®

containing perphenazine and amitriptyline HCl

prescribe it...
with good reason



For a brief summary of prescribing information, please see last page of this advertisement.

widely useful in psychiatric practice when moderate anxiety or agitation accompanies depression



Helps treat the total symptom complex—moderate anxiety and coexisting depression.

The antidepressant component is capable of alleviating such symptoms of depression as early morning awakening, anorexia, poor concentration, and feeling of hopelessness.

The tranquilizer component is capable of alleviating symptoms of anxiety, tension, and psychomotor excitement without apparent dulling of mental acuity. Hypnotic effects appear to be minimal, particularly in patients who are permitted to remain active.

The drug may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving.

Depending on the condition being treated, relief of symptoms of anxiety may occur within a few days, but adequate relief of symptoms of depression may take a few weeks or even longer.

Symptomatic relief often increases the patient's ability to return to normal family, social, and vocational activities.

Flexibility of four tablet strengths affords ease of dosage adjustment for better patient management.

Initial therapy for many patients is TRIAVIL® 2-25, containing 2 mg perphenazine and 25 mg amitriptyline HCl, or TRIAVIL® 4-25, containing 4 mg perphenazine and 25 mg amitriptyline HCl, t.i.d. or q.i.d. See full prescribing information for dosage in elderly and adolescent patients.

Treatment with TRIAVIL—a balanced view: TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. The drug may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

a wide range of indications...

patients with moderate to severe anxiety and/or agitation and depressed mood

patients with depression in whom anxiety and/or agitation are severe

patients with depression and anxiety in association with chronic physical disease

schizophrenic patients who have associated depressive symptoms

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containing perphenazine and amitriptyline HCl

prescribe it... with good reason

widely useful in psychiatric practice when moderate anxiety or agitation accompanies depression

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Available:
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TRIAVIL® 2-10: Each tablet contains
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TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

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FOR FLEXIBILITY IN ADJUSTING MAINTENANCE THERAPY
TRIAVIL® 2-10 (or TRIAVIL® 4-10)

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered. J7TR22 (DC6613212)

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Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Associat

This book reports six studies that add to the growing body of data demonstrating the feasi ering mental illness under health insurance. Although in some ways it may be considered a s APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND** Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal emp menting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health b
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- Utilization of care for mental conditions under the Canadian health insurance program the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
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Computerized transaxial tomography (CT scan) is an important new noninvasive technique that can help in determining the etiology of epileptic seizures. Shown here is a single slice from a color-enhanced CT scan of the brain of a patient with epilepsy. The absence of vascular abnormalities, tumor, or other lesions is compatible with a diagnosis of idiopathic epilepsy.

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A single entity with multiple anticonvulsant properties

Primidone itself has anticonvulsant activity. In addition, its major metabolite, phenylethylmalonamide (PEMA), exerts an anticonvulsant effect of its own and also enhances the anticonvulsant action of the second metabolite, phenobarbital. In patients refractory to phenytoin and phenobarbital, primidone may prove effective as the sole therapy.[1]

Multiple indications—grand mal, psychomotor, and focal epileptic seizures

In grand mal, MYSOLINE is comparable in efficacy to phenytoin and phenobarbital.[2,3] In psychomotor seizures, phenobarbital is rarely effective.[1] In focal seizures, phenytoin used alone is not indicated[4]—and, according to Forster,[3] MYSOLINE is markedly more effective than phenobarbital, which should dispel the misconception that MYSOLINE is "just a phenobarbital." (See chart below.)

Efficacy ratings* of anticonvulsants in epilepsy

Agent	Grand Mal	Psychomotor	Focal
Mysoline	++++	++++	++++
Hydantoinates (phenytoin)	++++	+++	+++
Barbiturates (phenobarbital)	++	++	++

*Relative effectiveness, ++++ being most effective.
Adapted from Forster, F. M.[3]

Consider it first for major motor epilepsies

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See next page for Brief Summary and references.

Start seizure control with MYSOLINE® (primidone)

Initial dose in children over 8 years of age and adults: 250 mg daily at bedtime

BRIEF SUMMARY
(For full prescribing information, see package circular.)
MYSOLINE®
Brand of **PRIMIDONE** *Anticonvulsant*

ACTIONS: MYSOLINE raises electro- or chemoshock seizure thresholds or alters seizure patterns in experimental animals. The mechanism(s) of primidone's antiepileptic action is not known.

Primidone *per se* has anticonvulsant activity as do its two metabolites, phenobarbital and phenylethylmalonamide (PEMA). In addition to its anticonvulsant activity, PEMA potentiates that of phenobarbital in experimental animals.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several weeks before it can be assessed.

Usage in pregnancy: The effects of MYSOLINE in human pregnancy and nursing infants are unknown.

Recent reports suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Data are more extensive with respect to diphenylhydantoin and phenobarbital, but these are also the most commonly prescribed anticonvulsants; less systematic or anecdotal reports suggest a possible similar association with the use of all known anticonvulsant drugs.

The reports suggesting an elevated incidence of birth defects in children of drug-treated epileptic women cannot be regarded as adequate to prove a definite cause and effect relationship. There are intrinsic methodologic problems in obtaining adequate data on drug teratogenicity in humans; the possibility also exists that other factors, *e.g.*, genetic factors or the epileptic condition itself, may be more important than drug therapy in leading to birth defects. The great majority of mothers on anticonvulsant medication deliver normal infants. It is important to note that anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and threat to life. In individual cases where the severity and frequency of the seizure disorder are such that the removal of medication does not pose a serious threat to the patient, discontinuation of the drug may be considered prior to and during pregnancy, although it cannot be said with any confidence that even minor seizures do not pose some hazard to the developing embryo or fetus.

The prescribing physician will wish to weigh these considerations in treating or counseling epileptic women of childbearing potential.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

PRECAUTIONS: The total daily dosage should not exceed 2 g. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 g per day. The initial dose is 250 mg. Increments of 250 mg are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 g. A typical dosage schedule for the introduction of MYSOLINE is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg daily at bedtime	*2nd Week* 250 mg b.i.d.
3rd Week 250 mg t.i.d.	*4th Week* 250 mg q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg, with gradual weekly increases of 125 mg a day, to a daily total usually between 500 mg and 750 mg.

In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances: for initiation of combination therapy; during "transfer" therapy; for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.).

HOW SUPPLIED: MYSOLINE Tablets—No. 430—Each tablet contains 250 mg of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg of primidone (scored), in bottles of 100 and 500.
MYSOLINE Suspension—No. 3850—Each 5 ml (teaspoonful) contains 250 mg of primidone, in bottles of 8 fluidounces.

References: 1. Woodbury, D. M., and Fingl, E., in Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics, ed. 5, New York, Macmillan Publishing Co., Inc., 1975, p. 222. **2.** Rodin, E., in Conn, H. F. (ed.): Current Therapy 1977, Philadelphia, W. B. Saunders Company, 1977, p. 716. **3.** Forster, F. M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. **4.** Physicians' Desk Reference, ed. 31, Oradell, N.J., Medical Economics Co., 1977, p. 1175.

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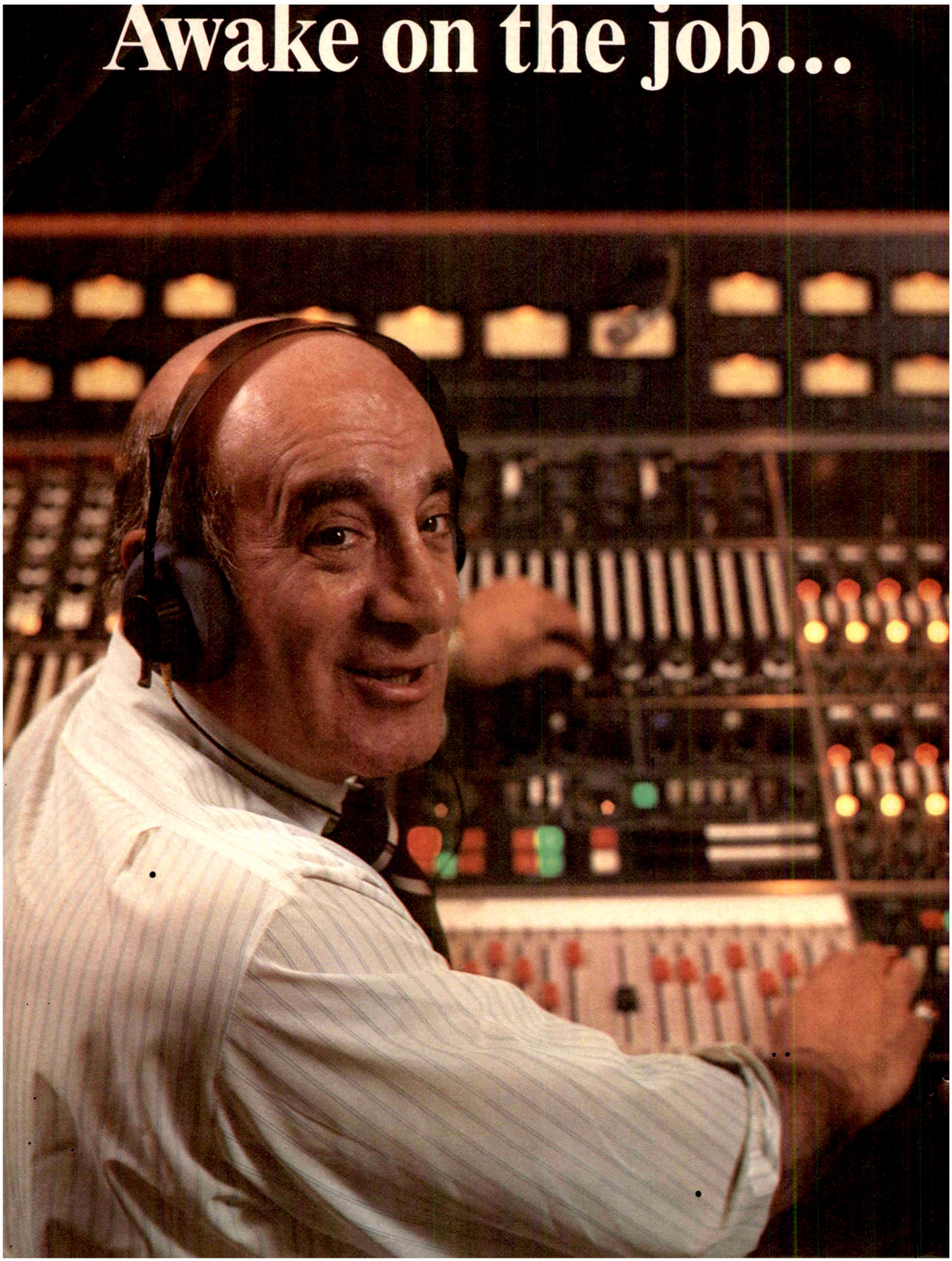
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Brief Summary

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ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

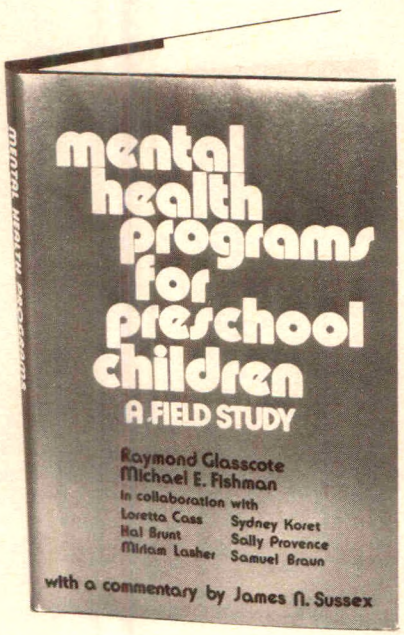
TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

A Timely New Book from The Joint Information Service

This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



An articulate exposition of the immense problems and encouraging accomplishments in a particularly difficult area of endeavor . . . . Beyond their concern with the manifestly ill child, the authors intriguingly explore what may be needed to enhance the mental good health of all young children . . . . A significant contribution.

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25 mg. Capsules

has them both.

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- Patients feel calm, yet remain alert.

For Brief Summary please see following page

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The antidepressant with both daytime and nighttime advantages.

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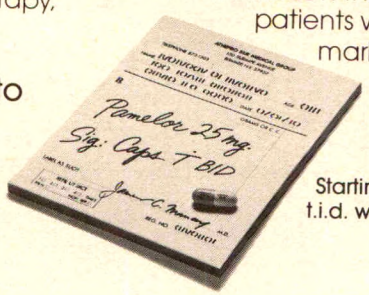
- PAMELOR therapy helps relieve the sleep problems that accompany depression: difficulty in falling asleep, restless sleep, and early-morning awakening. An improved pattern of sleep begins to appear in some patients within the first week of therapy.
- Overall improvement is often observed by the end of the second week. Maximum improvement with PAMELOR therapy, as with other antidepressants, may require a longer period of therapy, particularly in severe depressive illnesses.

PAMELOR therapy allows patients to remain calm yet alert.

- Helps improve depressed mood with a low level of stimulatory effect.
- PAMELOR therapy also has a mild calming action to help relieve the anxiety and tension that are often symptoms of depression. Because PAMELOR therapy rarely causes excessive drowsiness, patients are more alert and better able to function. Nevertheless, they should be cautioned about driving or operating machinery.

PAMELOR therapy is well tolerated.

- In 90 studies in which 818 patients were treated with PAMELOR therapy, over half (54%) of the patients who improved completely or markedly had no side effects at all.



Starting dosage 25 mg. b.i.d. Increase to 25 mg. t.i.d. within the first week as required.

Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose*—25 mg. three or four times daily; dosage should begin at a low level and increase as required. *Elderly and Adolescent*—30 to 50 mg. per day, in divided doses. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

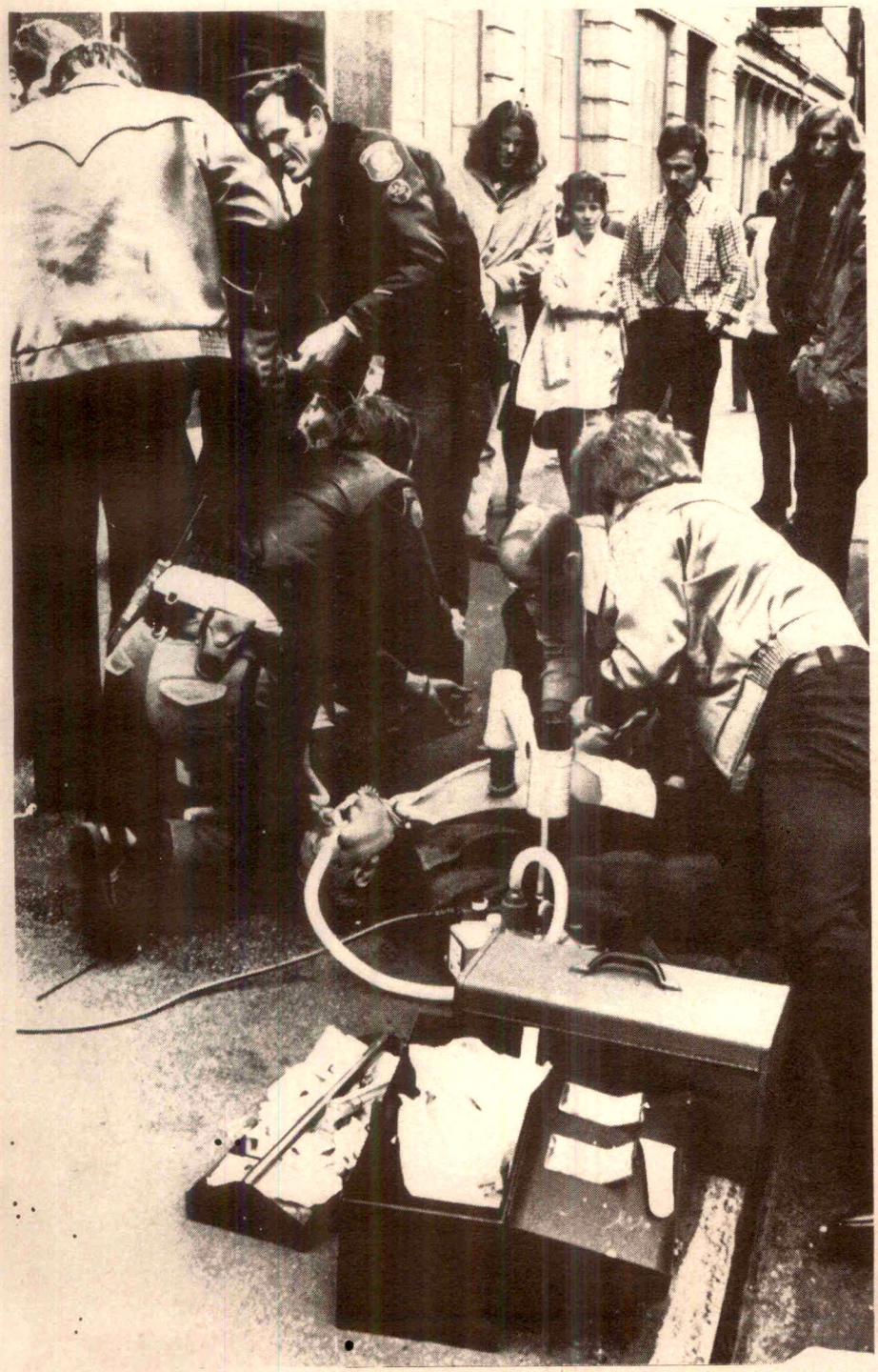
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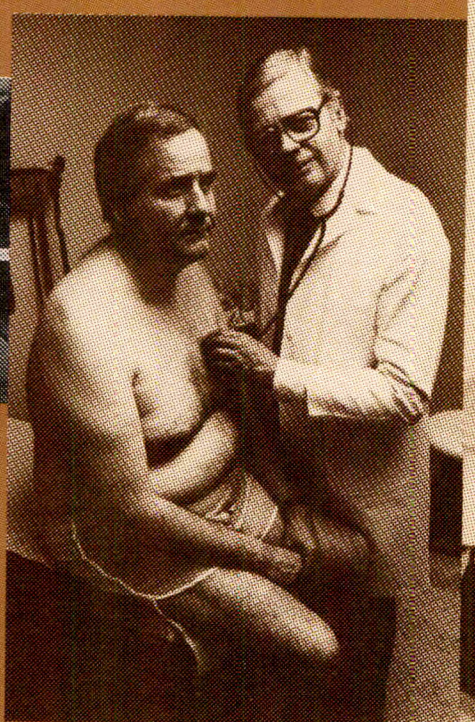
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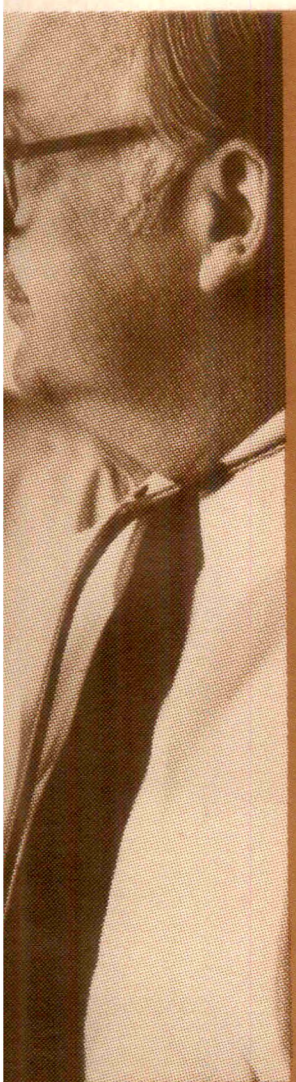


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- ☐ Proven antianxiety performance
- ☐ Minimal effect on mental acuity
- ☐ Predictable patient response
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Librium® (IV)

chlordiazepoxide HCl/Roche



5mg, 10mg, 25mg capsules

synonymous with relief of anxiety

Please see next page for summary of product information.

Librium® 5mg, 10mg, 25mg capsules
chlordiazepoxide HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states. Efficacy beyond four months not established by systematic clinical studies. Periodic reassessment of therapy recommended.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Warn patients that mental and/or physical abilities required for tasks such as driving or operating machinery may be impaired, as may be mental alertness in children, and that concomitant use with alcohol or CNS depressants may have an additive effect. Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Usual Daily Dosage: Individualize for maximum beneficial effects. *Oral–Adults:* Mild and moderate anxiety and tension, 5 or 10 mg *t.i.d.* or *q.i.d.*; severe states, 20 or 25 mg *t.i.d.* or *q.i.d. Geriatric patients:* 5 mg *b.i.d.* to *q.i.d.* (See Precautions.)

Supplied: Librium® (chlordiazepoxide HCl) *Capsules*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10. Libritabs® (chlordiazepoxide) *Tablets*, 5 mg, 10 mg and 25 mg—bottles of 100 and 500. With respect to clinical activity, capsules and tablets are indistinguishable.

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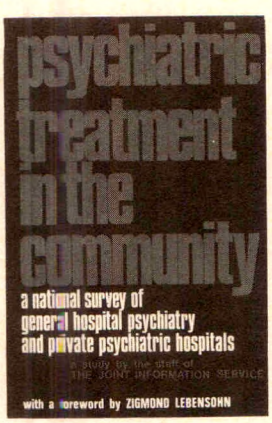
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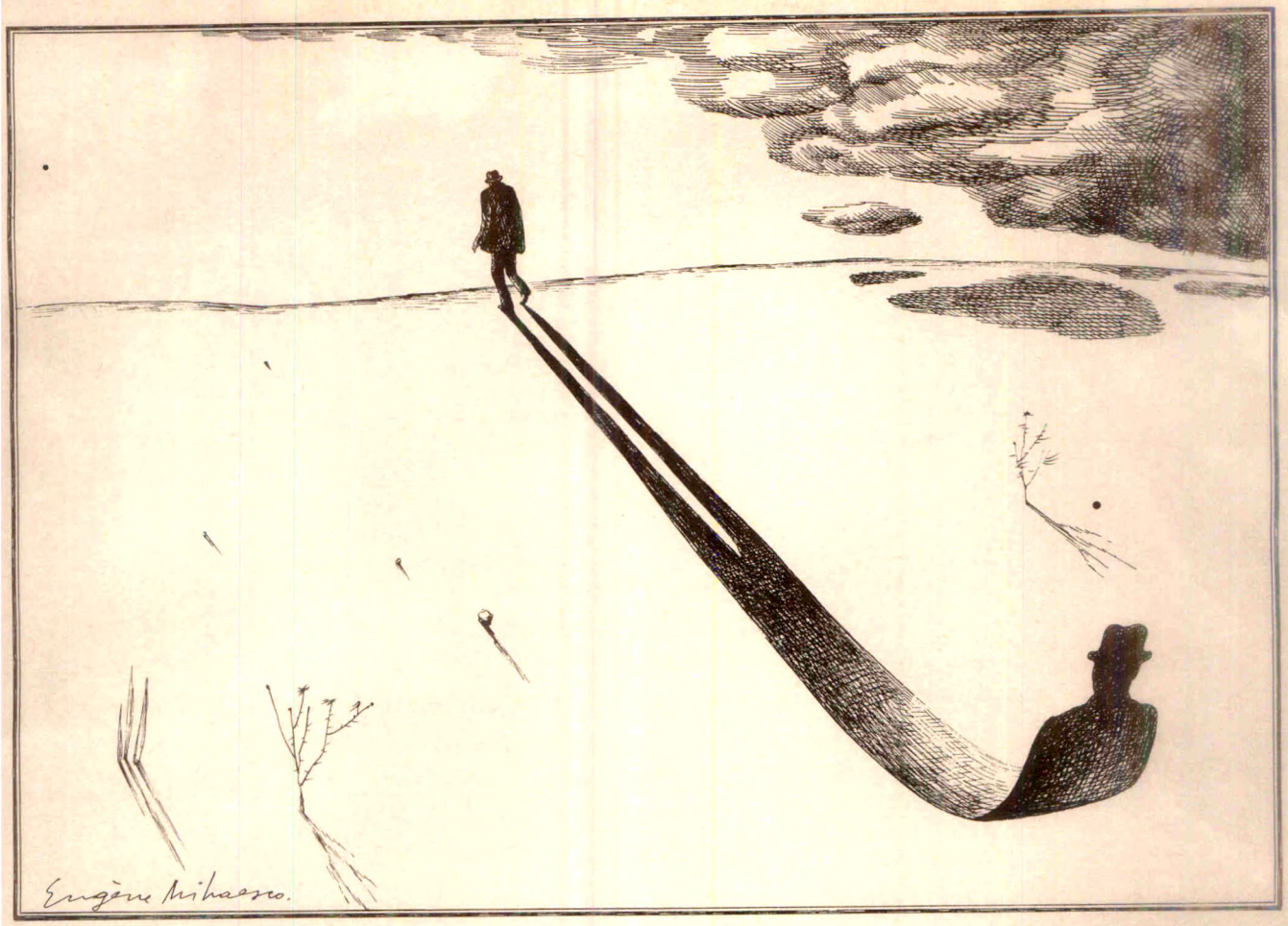
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That is what depressed individuals may feel is the substance of their being. There is no pleasure, no joy – nothing grows – and in the cold shadow of depression their activities are inhibited, while initiative may be eroded or destroyed. The tragedy is that they can see that others are able to live on the brighter side but they, themselves, cannot reach it on their own.

Your experience in treating depression, and Tofranil-**PM** can help light the way.

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Unsurpassed effectiveness among tricyclics in relieving symptoms of anxious depression.

Before prescribing Tofranil-PM, please review a summary of the prescribing information on the back of this page.

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As anxiety, agitation, sleep disturbances, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)

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For complete details, including dosage and administration, please refer to the full prescribing information.

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Prescribing Information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats Oral Dose —
20 mg/kg/day — 10 days, no adverse effect
40 mg/kg/day — 10 days, no adverse effect

Pregnant Mice Oral Dose —
20 mg/kg/day — 10 days, slight increase resorptions
40 mg/kg/day — 10 days, slight increase resorptions

Pregnant Rabbits Oral Dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg/day.
- — Increase to 100 mg/day in three or four days.
- — Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- — An increase to 225 mg/day may be required in patients with severe symptomatology.
- — Some chronic, treatment-resistant patients may require up to 400 mg/day; (however, the long-term safety of 400 mg/day has not been established).

Elderly or debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four times a day.
2. Moderate — 10 mg to 25 mg three or four times a day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

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SUPPLEMENT TO THE AMERICAN JOURNAL OF PSYCHIATRY

Festschrift for Francis J. Braceland, M.D.

Vol. 135, July 1978

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SUPPLEMENT TO THE AMERICAN JOURNAL OF PSYCHIATRY

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FESTSCHRIFT FOR FRANCIS J. BRACELAND, M.D.

Editor, *The American Journal of Psychiatry*, 1965–1978

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Am J Psychiatry 135, July 1978 Supplement

FESTSCHRIFT FOR FRANCIS J. BRACELAND, M.D.

A Fond Letter to the Editor

DEAR FRANK,

What can one give to the Editor who has everything?

That, sir, was the question that plagued your Association as it searched for a suitable way of honoring your thirteen magnificent years as the ninth Editor of our *Journal*. As we have watched you at work, we have discovered the virtues that have guided your editorial pen: the breadth of your knowledge and interests, which, combined with your open-minded tolerance of controversy, have made the *Journal* a truly balanced forum for ideas; your unerring critical sense of what is appropriate for publication; your encouragement of younger writers and your soft answer that turneth away the wrath of the rejected author; your genius for the *mot juste* and the apt quotation; your unfailing wit and humor that have poured oil on troubled editorial waters and nourished the loyalty of your staff. What was there left, we asked ourselves, to bestow on a man with possessions like these?

As the shadows lengthened on our disheartened deliberations, an enthusiastic voice suddenly rang out, "I have it—a *Festschrift*!"

The collective response was as dreary as a Boston winter: "After thirteen years of editing, the last thing in the world Frank needs is more papers."

"Hear me out," replied the protagonist. "These will be no ordinary papers. Not one of them will Frank have seen in manuscript. Not one of them will he be required to read with an editor's critical eye, to send out for review, to appraise for acceptance or rejection. All this will have been done for him in an issue of the *Journal* prepackaged for his personal perusal. Like any ordinary citizen-subscriber, he will be able to sink back in his favorite armchair to read and reflect, to agree or demur, and even, if he is so minded, to write a letter to the Editor. That, I submit, would be a gift unique in the annals of editordom."

The arguments were irrefutable, the problem solved. This, then, is your own special copy of the *Journal*, edited from title page to colophon while you were out of the office. We have tried to select a table of contents to tempt your intellectual palate. Each of these papers, written by one of your countless colleagues and admirers, reflects an area enriched by your life and writings as clinician, scientist, humanist, administrator, editor, and admiral. Ransom Arthur Bunney gether the history and the nature of military on the basis of his experiences in the Navy you have given such long and loyal service. Bunney reviews the remarkable advances in psychiatry that have coincided with your years tor, and to which you have made your own contributions. Robert Butler discusses the aging, which you have always illuminated your youthful humor. Your distinguished leadership one of our country's foremost private mental is signalized by Robert Gibson in a paper to a major issue facing American psychiatry the place of private psychiatric institutions of patients with chronic mental illness. David argument for the vital importance of a knowledge psychiatry's past is underscored by the profound and scientific wisdom with which your own understanding of history have endowed you. Herbert Weiner applies the medical model to try in a paradox of truly Shavian proportions should delight your never-failing appreciation new, the unexpected, the creative.

It will gladden your Editor's heart to know of these authors leapt with enthusiasm at the to join in honoring you, and not one of them not one of them grumbled when faced with an almost impossible deadline for the submission contribution. Your Editorial Staff, too, labored hard and devotedly at the innumerable details of preparing the manuscripts for publication labor of love from us all, this *Festschrift* bears boundless esteem, the heartfelt good wishes deep affection of your Association, of the these papers, of your Associate Editors, of your rial Staff, and, through all of them, of that of readers of the *Journal* you have made among medical publications.

Am J Psychiatry 135, July 1978 Supplement

Reflections on Military Psychiatry

BY RANSOM J. ARTHUR, M.D.

The need for psychiatrists in the military was recognized for the first time during World War I, which involved millions of men in unusually protracted warfare. The policy of treating psychiatric casualties close to the front and returning soldiers to their military units as quickly as possible proved of great significance in the U.S. war effort. During World War II, the Korean conflict, and the war in Viet Nam, military psychiatry made great contributions and learned many lessons, both at home and abroad. The lessons learned by military psychiatry have important applications for the rest of medicine, especially in the fields of stress, crisis therapy, and community psychiatry.

IT IS A PARADOX that psychiatry, which is among the most pacific of disciplines, and the military, which represents organized bellicosity, have their moments of intimacy. Psychiatry and the military appear to be, as King Charles I said of monarch and subject, "clean different things." At first glance it would seem that surgeons, with their blunt, no-nonsense, aggressive style, might be more compatible with the military environment than psychiatrists, who often possess a liberal political outlook and a very high need for autonomy. It is true that the military has had little trouble recruiting surgeons and that surgeons have always been well represented in the ranks of military medical officers who achieve the highest grades and positions. Although the professional activity of the surgeon, particularly the surgery of war trauma, is indeed an integral part of the military mission, it does not quite ever occupy the central zone.

The core of military organizations, whether they are prepared to fight with bows and arrows or with hydrogen bombs, involves such items as leadership, courage, morale, motivation, perseverance, dereliction, malingering, and the age-old problems of drink and breaches of discipline. Psychiatry in the military setting finds itself inexorably drawn into considerations of these eternal verities of military life. Some psychiatrists in the military can concentrate on the purely professional activities of treating psychotic patients without reference to their patients' roles within the military organization. The vast majority of psychiatrists, however, must become involved in the structure of command and discipline whether they wish to or not. What is true for the peacetime military is doubly true for the military at war.

It is difficult to speak of psychiatry in the military before the latter portion of the nineteenth century, although psychiatric problems have always existed. Surely the erratic behavior of Caligula when he placed himself at the head of his Roman legions in the field cried out for psychiatric intervention, but there was to be no formalized military psychiatric assistance for almost another two millennia. Physicians during the American Civil War actively worked with soldiers who had what we would now recognize as various combat syndromes (1). The celebrated neurologist S. Weir Mitchell was an active medical officer during that conflict; he took an interest in the psychological problems of the soldiers in the Union Army. In the twentieth century, it was noted that during the siege of Port Arthur in the Russo-Japanese War of 1905 there were many casualties who seemed to have some kind of neuropsychiatric syndrome. They were diagnosed as syphilitic, but in view of what we learned about siege operations during World War I it seems unlikely that neurosyphilis was the real cause of their symptoms.

WORLD WAR I

It was World War I, the so-called Great War, that gave military psychiatry its initial formal surge. By 1914 psychiatry was a demarcated urban medical specialty; it was no longer primarily the province of mental hospital superintendents as it had been in the previous century. The great medical schools of Europe had professors of psychiatry who were directors of famous psychiatric clinics in such cities as Berlin and Vienna. Kraepelin had made his taxonomic contributions to psychiatry, and Bleuler had further delineated the concept of schizophrenia. The theories of Freud were known internationally, albeit often in a distorted and vulgarized way. The basic ideas of the unconscious and of psychodynamics were in the air. Neurosyphilis was a reasonably well understood entity. It must not be thought that psychiatry was, then or now, a truly high status specialty. However, there was a sufficient body of organized knowledge and a requisite number of working psychiatrists to enable them to deal with the unprecedented psychological problems arising from World War I in a far more systematic way

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than would have been the case had a similarly catastrophic war occurred 50 years earlier.

The Great War involved millions of men in warfare of a protracted character not seen since the sieges of the Middle Ages. The horrors of trench warfare were greater in duration and magnitude than soldiers had previously endured. Major battles of the past, like Gettysburg or Waterloo, lasted two or three days, and even operations such as the siege of Richmond at the end of the American Civil War lasted but a few months. World War I dragged on year after erosive year and exposed men to constant danger, disease, fatigue, and untold horrors. It soon became apparent that there were large numbers of casualties among men who had suffered no obvious physical trauma. Twenty percent of the soldiers discharged for disability from the British Army had such psychiatric disorders as "shell shock," "war neurosis," or "neurasthenia" (2). There were soldiers who had gone blind, who had become unable to speak or hear, who had paralyzed limbs, or who shook so hard from anxiety that they were unable to function.

It was obvious that viewing these thousands of casualties as malingering on a mass scale was not the way to deal with the situation. There were attempts to treat such patients by methods common to the practice of neurology and psychiatry at that time: rest, diet, persuasion, sedatives, and moderate exercise. As one reads the case reports from both the Western Allies and the Central Powers, one is struck by the colorful phenomenological descriptions and the inadequacy of the theoretical framework of understanding (3). Physicians attempted to relate the type, extent, and severity of traumatic experiences and the individual character of the patient to the final outcome, but they had no unifying theory of traumatic neurosis. The syndromes of combat affected not only those who might have been expected to become shirkers but also soldiers and officers of demonstrated courage. Siegfried Sassoon (4), a man of supreme bravery, described his own experiences in a psychiatric facility for convalescent officers in England after three harrowing years in the trenches.

In spite of their desperate need for soldiers, the contending nations were relatively lax in their initial provision and monitoring of manpower. For example, Britain did not introduce conscription during the first year of the war. As the powers gradually became aware of the vast losses attendant on neuropsychiatric casualties, they developed a treatment doctrine pertaining to combat reactions that seemed to be suitable for both man and institution. In the U.S. Army, Cols. Thomas W. Salmon and Pearce Bailey were instrumental in stationing psychiatrists at the divisional level (5).

It was the duty of the psychiatric medical officer to integrate himself fully into the life of his division and to provide psychiatric services close to the front line. Psychiatrists provided rapid treatment of transient combat reactions that could be dealt with by rest, reassurance, provision of hot food, and the briefest kind of recuperation. These psychiatric medical offic[illegible] realized the enduring truth of combat psy[illegible] namely, that if one evacuated patients w[illegible] would otherwise be transient combat reacti[illegible] safe spot several hundred miles behind the lin[illegible] to the zone of the interior, it was virtually in[illegible] to get these soldiers to return to full com[illegible] Treatment close to the front line mobilized [illegible] dier's primary psychological weapon: iden[illegible] with his comrades in his military unit. A soldi[illegible] had a strong sense of guilt about deserting [illegible] while it was under heavy fire, no matter h[illegible] pelling the reason.

The American psychiatric program proved [illegible] markably successful. After initially high leve[illegible] chiatric casualties, the rate for the U.S. Arm[illegible] a rather low order of magnitude. To be sure, [illegible] ordinarily high quality of the vigorous America[illegible] their freshness in comparison with the exhau[illegible] ropeans, and the constant series of victories d[illegible] summer and early autumn of 1918 no doubt co[illegible] to their superb morale and low rate of psyc[illegible] disability. Nevertheless, the efforts of a very [illegible] chiatrists proved of real significance in the fu[illegible] of the American war effort in the European t[illegible] operations.

The war had a profound impact that [illegible] beyond those psychiatrists who were on [illegible] vice. The unremitting horrors of the war [illegible] Sigmund Freud to make considerable modifi[illegible] his thinking about instinct theory and drea[illegible] tion. On the basis of the reported experience[illegible] war, he realized that certain dramatic events [illegible] duce stereotypic dream patterns of a persist[illegible] that had little to do with wish fulfillment. [illegible] after witnessing the appalling carnage of the [illegible] hard to deny the strength of aggressive and ca[illegible] motivational forces in human life. Freud, wh[illegible] in 1856, had spent his entire life in an era [illegible] general European war, let alone a world w[illegible] easy in the peaceful Victorian twilight to reg[illegible] gressive and death-connected aspects of [illegible] tivity as of lesser importance than the libidi[illegible] views could not be held seriously in 1919. F[illegible] *Beyond the Pleasure Principle* (6) after the [illegible]

By 1918 military psychiatry had discove[illegible] ber of elemental principles. The first was [illegible] longed exposure to a situation of constant c[illegible] deprivation could result in various disa[illegible] dromes not brought about by obvious physic[illegible] Second, this type of reaction to prolonge[illegible] stress was very widespread. Third, the factor[illegible] to do with the social support given to a sold[illegible] unit and leaders had important prophylactic [illegible] the prevention of combat reactions. After [illegible] many of these crucial lessons were forgotte[illegible] in the United States, in spite of the extens[illegible] mentation given in volume 10 of the *Medical Depa[illegible] ment of the United States Army in the W[illegible] War*. This volume was apparently read as an impe[illegible]

by the Royal Army Medical Corps in Britain, but its lessons seemed to have vanished from the American consciousness.

In between the wars, during what Robert Graves called "the long weekend," the regular armed forces were kept at a ridiculously low level; the medical departments were correspondingly small (7). There was only a handful of psychiatric specialists in the Army and Navy. There were 35 psychiatrists in the Army, and only 4 of these were diplomates of the American Board of Psychiatry and Neurology. There was no carefully thought-through contingency plan for the treatment and rehabilitation of psychiatric casualties. To anyone unfamiliar with the melancholy and isolated history of psychiatry in general, this glaring omission would seem very odd. Those with a knowledge of the past would know that a very large number of psychiatric casualties could be expected in a general war. Nevertheless, little thought was given to planning in this area. Psychiatric specialists were not highly regarded in either civil or military life, and the kind of cooperation and visionary thinking necessary to adequately prepare a psychiatric program was largely lacking.

WORLD WAR II

Fortunately, many of the leaders of Army and Navy psychiatry during World War II were drawn from a group of experienced civilian psychiatrists. I could mention several hundred contributors by name, but any list of distinguished wartime psychiatrists would do injustice to others whose contributions might have been equally great but not as well recognized. Francis Braceland was one of the true pioneering leaders of modern Navy psychiatry. Already Dean of Loyola Medical School, after commissioning in the Naval Reserve he recruited outstanding psychiatrists for naval service. He became assistant head of the Neuropsychiatric Section (later Branch), formed under the leadership of Captain Kennedy in 1942, and in 1944 was appointed head of the Branch. During the course of the war, under his outstanding leadership, the U.S. Navy expanded from 8 psychiatrists to approximately 980. Throughout that period the Neuropsychiatric Branch enjoyed the confidence and cooperation of the Navy Surgeon General, Admiral McIntire.

Dr. Braceland wrote extensively on problems of Navy psychiatry, including the areas of selection, combat reactions, and institutional variables in the incidence of psychiatric disease in the military (8, 9). Among the important duties that he and others were called on to perform during the course of the war was the crisis therapy of highly placed officers and civilian officials who developed severe psychiatric reactions. Psychiatrists who were given these missions had to work under all the adverse conditions that are connected with the treatment of VIPs, but their therapeutic task was given a special urgency when, for example, the individual was a key member of the team working on the atomic bomb.

Among the major tasks of military psychiatry during both world wars was the psychiatric and psychological screening of individuals with mental handicap or psychiatric disorder of sufficient magnitude to preclude the possibility of successful military service. The screening out of men and women who were grossly impaired was of crucial importance to the effectiveness of the fighting forces (10). A more controversial area, however, was the detection of individuals with personality deficits labeled as immaturity or unstable character. Many psychiatrists prided themselves on being able to distinguish recruits who would not become good soldiers or sailors on the basis of a three-minute interview. There were no studies in wartime that conclusively demonstrated the validity of such predictions. Research on this topic was performed, but it failed to include a control group of individuals who were enlisted in spite of adverse psychiatric opinion with which to compare the psychiatrically cleared group. Postwar research at the Navy Neuropsychiatric Research Unit has cast great doubt on the ability of the psychiatrists to screen out at a level greater than chance those individuals who would later become ineffective sailors and Marines. The studies were carried out with cohorts from 1960 and 1961 (11). However, the study conditions were different from those existing during the war, and it is possible that wartime screening was effective.

When World War II began, the U.S. Army did not immediately build on the carefully worked out psychiatric plans of 1918. Instead, a somewhat ramshackle approach to the problems of psychiatric casualties was initially taken by the Army high command. The number of psychiatric casualties was initially shockingly high. During wartime the incidence of psychosis usually rises rather little (12). In fact, for well over 60 years the incidence rates of psychosis in the U.S. Navy varied scarcely at all under both peacetime and wartime conditions. However, situational reactions, psychoneuroses, and character and behavioral disorders showed great changes in incidence rates according to circumstances. The recorded incidence rates of these conditions depend not only on environmental variables but also on medical administrative policies. It is notorious that line commanders as well as medical commanders influence the diagnoses given to certain categories of military patients.

By 1943, however, both the Army and the Navy had developed a strong cadre of superb psychiatrists, many from civilian life, who were being used, by and large, in a correct manner. There was a strong emphasis on sound combat psychiatry and rapid return to duty. In addition to the psychiatrists stationed at some proximity to front-line fighting, there were others on duty in psychiatric wards, base hospitals, psychiatric hospitals, psychiatric units on hospital ships, in administrative posts at higher headquarters, and in combat air crews in the Army Air Corps, later the U.S. Air Force. In fact, some of the most dramatic, gripping, and terrifying stories of the effects of combat stress

came from psychiatrists who were stationed with the air arm (13). Psychiatry proved itself an important tool in maintaining air crew effectiveness. Flight surgeons, of necessity, had to have at least a rudimentary knowledge of practical psychiatry in order to function effectively. By the end of the war both services had superb psychiatric capability. A roster of leaders of military psychiatry and psychology during the war reads like a "Who's Who" of pre- and postwar American academic psychiatry and clinical psychology. After the war those who had served in posts of responsibility during the war itself were well equipped to move into important academic posts in an era of expansion.

THE LESSONS OF WARTIME PSYCHIATRY

The psychiatric experience of both world wars provided a number of lessons that proved to be of general interest. Military psychiatrists were first to describe the specific features of combat syndromes. They documented the overt anxiety, recurrent nightmares, startle reactions, exhaustion, and other cardinal features of combat stress. Combat reactions are not monolithic. Their subcategories were distinguished by war psychiatrists in the field. For example, patients might be classified as showing simple combat fatigue, hysteric or anxiety reaction type, or psychotic reaction type. In each instance the syndrome was a result of the kind, amount, and duration of stress superimposed on the preexisting mental status and character of the patient, his characteristic coping abilities and mechanisms, and the morale of and support given his unit. Unit morale, in turn, depended very often on excellence of leadership.

Military physicians were also pioneers in the description of the concentration camp syndrome (KZ syndrome), which represents the classic instance of a clearly identifiable syndrome arising from extreme stress. This syndrome developed in a fearsome situation in which there was a pervasive threat of death. The syndrome is characterized by difficulties in mentation, anxiety, depression, insomnia, and gastrointestinal and other persistent physical symptoms (they have persisted for 35 years now) (14). The whole basic idea of crisis and crisis therapy clearly owes much to descriptions of the traumatic disorders of war and their treatment. The increase in interest in therapeutic processes, and most particularly the surge in the popularity of group therapy, as well as the basic thrust of a therapeutic community in psychiatric practice, arose in part out of the wartime experience, particularly that of the English military psychiatrists.

One of the most important phenomena of the war was the exposure of large numbers of nonpsychiatric physicians and future physicians to the experience of military psychiatry. Specialists and general practitioners who had thought of psychiatry in terms of remote alienists or isolated mental hospital work were exposed to a vast variety of psychological disturbances arising in men and women whom they had kn... sane and ordinary. These physicians were ab... fy that stress in the biological, social, and ps... cal sense could bring about terrifying disinteg... personality. Wartime physicians, by workin... cert with a large number of excellent psychia... the opportunity to verify that their psych... leagues were also good physicians, not strar... sentatives of a wholly different profession.

The wartime scientific and technological s... such as the perfection of radar and the ator... gave science and technology an enormous ... in the eyes of the general public. Advances i... such as the discovery and widespread use of ... further enhanced the idea of the seeming ... potential of science. It was a rising tide that ... boats, and psychiatry benefited from the ge... of approbation extended to science and me... 20 years following the end of World War II ... psychiatry enjoyed a golden age. Never b... alas, never since, has psychiatry been so po... popular, and so respectable. Departments o... try flourished in medical schools throughc... tion. State university medical schools that h... ly had one half-time psychiatrist on the fact... oped departments with as many as 100 ... Larger schools had departments that grew ... spondingly greater dimensions. Money flow... all was a sunny morning. It was only with t... tudes of the middle and late 1960s that the ... sun began to decline again, at least tempor... larly, after the first 3 or 4 postwar years of ... demobilization, military psychiatry flourish...

THE KOREAN CONFLICT

During the Korean conflict, the U.S. Arm... absorbed the lessons of the two previous ... century conflicts, had an effective program ... psychiatry based on the fundamental princ... treatment in a setting close to the front-line... rapid return to duty (15). The paradigm of ps... working in close concert with field units wa... to the peacetime military post, at least in ... Psychiatric personnel were expected to pla... role within the military community. These ... giene services within a closed military gro... forerunners of civilian community psychia... my mental health groups offered services n... active duty people but to their families as we... atrists also became involved in a consultative ... unit commanders and other important offic...

RESEARCH

It was during the 1950s and 1960s that the ... Navy fully developed permanent psychiatr... facilities: the Walter Reed Army Institute o...

in Washington, D.C., and the Navy Medical Neuropsychiatric Research Unit (now the Naval Health Research Center) in San Diego, Calif. The major theme of both enterprises is research on the effects of stress. Biological, social, and psychological approaches are used. For example, the work of Mason (16) and his fellow investigators at Walter Reed on the neuroendocrinological aspects of stress represents an important contribution to biological science in general.

The Naval Health Research Center in San Diego has concentrated on research on the epidemiology of psychiatric disorders in the Navy and Marine Corps, the environmental factors influencing health in military personnel, the effect of stress on disease onset and health patterns, the effect of stress on performance in such groups as Navy aviators and under-water demolition team members, as well as research on the most effective patterns of sleep and rest in the maintenance of vigilance and efficient task performance. Other important studies carried out at the Navy unit include studies on the prediction of successful military performance (17). More recently, the Center for the Study of Prisoners of War was developed in San Diego to study all aspects of the captivity experience and develop rehabilitation plans. This center also studied the families of the released prisoners. The mission later expanded to include studies of the stresses and strains of military families in general. Although it is surely correct that many of the studies have an applied focus, most have important implications for a particular area of biobehavioral and psychiatric sciences. The sum of the discoveries represents a body of work in which the medical departments of the Armed Forces can take justifiable pride.

THE VIET NAM CONFLICT

There can be no doubt that the war in Viet Nam, with its malignant effects on life in Southeast Asia and in the United States, had injurious effects on the American military—and on military psychiatry as well (18). It was difficult to maintain the morale of members of the Armed Forces during this troubled period. The psychiatric precepts that had proven so useful in wars with a clearly identified enemy seemed less obviously applicable during a war monumentally unpopular among military and civilian populations alike. Lengthy exposure to prolonged combat and threat of death without any definite hope of escape were not present in Viet Nam. The amount of time that an individual would spend in Viet Nam, provided he survived, was fixed. Combat was often of a highly sporadic character against a foe who, except for an occasional pitched battle, preferred to come and go out of the shadows.

Although the danger of death and mutilation for our men was very real, as the tens of thousands of combat deaths attest, the combat in Viet Nam was much different from that experienced in the trenches of Flanders in 1916, during the siege of Stalingrad in 1942, or during the prolonged battles along the 38th parallel in the Korean conflict. Military psychiatrists had to cope with a new type of patient with new types of stress. No longer was there necessarily a basically motivated soldier fighting as part of a proud and recognized military unit, who felt guilt about leaving his comrades, who might be suffering temporarily from nightmares, anxiety, and exhaustion, but who had a basic desire to return to do his duty. Instead, there were often military patients whose motivation to return to duty was at best ambiguous. The amounts of combat stress received were imprecisely known. There were often various adventitious elements complicating the clinical picture, such as the unprecedentedly large number of men taking drugs. It is not possible to compare directly the psychiatric casualty rates of the Viet Nam conflict with those of previous conflicts. The Viet Nam figures were widely publicized as being lower than those of the past, but it is not clear what significance to attach to data of differing types. The short tours of duty (one year) and peculiar nature of the combat had no exact parallel in previous wars. The special psychiatric problems of Viet Nam veterans were not confined to the wartime period and have continued to loom large in the operation of certain Veterans Administration programs for such individuals (19).

ETHICS

Another problem that arose in an acute form during the time of the Viet Nam conflict was the question of the ethics of military medicine. With the growth of ethical hypersensitivity during the past few years, questions have arisen as to whether it is possible to reconcile one's oath as a physician (implying as this does the total dedication of oneself to the welfare of the individual patient) with the institutional demands of the military, in which the individual is subordinated to an overall group purpose. This ethical conflict is perhaps most marked in military medicine associated with combat. For example, is the surgeon justified in attempting to repair an injury as quickly as possible so that the individual can swiftly return to combat, where he might well be injured again or killed? Is the military psychiatrist justified in rapidly treating combat fatigue? Is the physician ethical in using his patient's guilt about deserting his comrades and his identification with his unit in order to have him quickly returned to combat, where he might soon be killed? Should not the psychiatrist counsel this patient to evade his duty and return to the safety of the zone of the interior? Should not the psychiatrist help the soldier to rationalize that he was in fact fighting in a wicked war? Should not the psychiatrist affirm that all wars are wicked, that the patient's own self-interest lay in expunging any sense of guilt or obligation to others and in seeing, in a clear-eyed way, what is best for him alone? During the Viet Nam conflict such choices posed a Hamlet-like dilemma in which ethical fastidiousness became a

serious concern within the context of an affluent society in the midst of a distant and unpopular war in which national survival was not in jeopardy.

Moral dilemmas of military medical practice were mentioned in works dealing with military psychiatry in both of the world wars but were quickly dismissed. It was felt that there was no violation of the physician's ethical role in his military medical work and that psychiatrists were obliged, as a matter of duty, to restore a man to functional integrity by repairing the ravages of a combat neurosis. It was assumed that, restored to mental health, the soldier would necessarily wish to do his duty.

It must be said that scrupulosity on this issue could only be the product of recent times. In the first place, in centuries before the nineteenth, the social position of physicians, save for a very few, was sufficiently low to make them glad to have a military post without giving serious thought to moral considerations. During the Roman times physicians were slaves, and even in the last century the commission as an officer that the military surgeon might receive was considered a higher honor than his medical qualifications per se. It has only been in the twentieth century that the social and economic status of physicians has risen to a point where it exceeds that of military officers. The major wars of this century have been seen by both sides as ideological struggles in which national survival itself was at stake. The Kaiser and later Hitler and the Japanese militarists were considered so wicked by their enemies that the world wars were thought of as holy missions. Eisenhower spoke of a "crusade in Europe," and the subsequent disclosure of the barbaric activities of the Nazis made this feeling not implausible. It was only with the arrival of a war of stupendous unpopularity involving a nation possessed of unprecedented comfort and affluence that moral sensitivity arose about a myriad of war-related issues. Questions about medical ethics are the subject of innumerable discussions that do not often lead to definitive answers. Most people, through one mental process or another, manage to reconcile their duties as citizens, physicians, and organizational members without serious moral repugnance. In any case, with the ending of America's direct participation in war, the moral issues of combat psychiatry are now moot.

Many institutions in American life suffered an erosion of authority and loss of public goodwill as the result of the Viet Nam conflict. The military, in general, was prominent among the institutions injured. In turn, military psychiatry is in a subdued phase as compared with 15 years ago. The number of psychiatrists in uniform has dwindled considerably, and there are few senior people left. Nevertheless, the situation is not so bad as it was before World War II. Psychiatry is still recognized as an important entity within the military forces. The perennial tensions between psychiatry and the military command continue. Commanding officers still wish to use psychiatric services as a way of disposing of troublemakers. As ever, there are still bizarre fantasies on the part of high-ranking lin[illegible] that psychiatrists are able to read people's [illegible] have the ability to screen out potential failu[illegible] programs in which the failure rate is less than [illegible] thousand. On the other hand, the military is [illegible] more accepting of the possible worth of al[illegible] programs and other psychiatric rehabilitative [illegible] ors for military families and active duty pers[illegible] cluding high-ranking officers. A psychiatrist [illegible] cellent professional and political skills is still [illegible] tion to become a central figure in an isolate[illegible] base in the areas of discipline and morale, pr[illegible] or she makes the necessary effort and has a sy[illegible] ic commander.

Military psychiatry has made important [illegible] tions to psychiatric thinking in general duri[illegible] six decades. Just as Lind, Beaumont, an[illegible] Reed, all distinguished military physicians, [illegible] nal advancements in medicine, psychiatric [illegible] particularly the field of stress, is much indebt[illegible] coveries of military psychiatry in discipline[illegible] from neuroendocrinology to psychoanalysis.

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Drug Therapy and Psychobiological Research Advances in the Psychoses in the Past Decade

BY WILLIAM E. BUNNEY, JR., M.D.

There have been major advances in the treatment of psychoses in the past decade, particularly in the area of maintenance therapy with lithium, tricyclics, and neuroleptics. Such efforts have been enhanced by methods of relating plasma drug levels to clinical efficacy. Research in genetic factors, the mode of action of pharmacological agents, and hormonal influences on psychopathology has added to our theoretical understanding of the psychoses, and we can hope for further advances from sophisticated new methodological tools. Promising areas of investigation include the search for genetic markers, studies of postsynaptic neuronal receptors, and examination of the effects of polypeptides, including endogenous opioid compounds.

IN THE PAST DECADE significant advances have been made in the pharmacological treatment of the psychoses. Furthermore, new methods and hypotheses in the area of psychobiology provide the promise of rapid expansion of knowledge in the future.

Perhaps the most important therapeutic advance that has occurred in the drug treatment of schizophrenia and affective illness during this period is the accumulation of evidence concerning the value of long-term drug maintenance with lithium and tricyclic compounds in affective illness and with neuroleptics in schizophrenia. A large number of studies have shown that lithium carbonate markedly decreases the intensity of recurrent episodes of depression and mania in at least 60% of patients maintained on the drug (1, 2). Some studies suggest that maintenance therapy with tricyclic compounds is also effective in decreasing the incidence of recurrent depressive episodes in unipolar patients (3). Studies in the past 10 years investigated the untreated relapse rate one year after resolution of an acute depressive episode (3, 4). The mean relapse rate in these studies was 64%, with a range of 36%–92%. Additional studies (4) addressed the issue of how the relapse rate would be affected by continuation of tricyclic antidepressant medication and found a mean decrease in relapse rate of 50% with maintenance treatment. Investigations are currently in progress that will provide further data concerning the value of long-term tricyclic maintenance, the doses required, and whether a combination of tricyclic and lithium is more or less efficacious than either treatment alone.

In schizophrenia, it is equally convincing that the long-term maintenance therapy with neuroleptics can decrease the need for rehospitalization and that rehospitalization rates can be further decreased when drug therapy is combined with psychotherapies. The efficacy of neuroleptics has been demonstrated in 26 uncontrolled nonblind studies and 22 controlled double-blind studies (5). Patients taken off phenothiazines after initial improvement show a high rate of relapse through the third and sixth months. The relapse rate continues to increase until 12 months after discontinuation of phenothiazines, when there is a plateau in the cumulative frequency curve of relapse rates. It has also been reported, although recently questioned, that use of the long-acting depot phenothiazines, fluphenazine enanthate and fluphenazine decanoate, further decreases recurrent hospitalizations, probably as a result of avoiding compliance problems of daily neuroleptic maintenance (5). Long-term maintenance with neuroleptics is associated in some patients with the development of tardive dyskinesia (6). Concern at the present time is due to an increased prevalence of tardive dyskinesia and the fact that some of the dyskinesias are irreversible and treatment is often unsatisfactory. If dyskinesias are recognized early, discontinuation of neuroleptics is often effective. However, the physician may then find himself in a therapeutic dilemma, since the patient's condition may require continued treatment. Thus a major task of current research is understanding the mechanism of movement disorders.

DRUG PLASMA LEVELS

Relating plasma levels of drugs to their effectiveness has provided another possible advance in psychotherapeutics, both in avoiding toxicity and in establishing therapeutic levels. This has allowed the safe administration of lithium in the correct therapeutic dose to hundreds of thousands of individuals.

Methods have now been developed to evaluate vari-

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ous tricyclic compounds and some of their active metabolites in plasma. These determinations are beginning to gain wider clinical use. Different individuals given exactly the same dose of a tricyclic compound can show a 10-fold to 20-fold variation in plasma levels. Thus on the same dose some individuals may be receiving too much of the drug, while others have a plasma drug level below the therapeutic range (4). This results from a variety of considerations, including a number of genetically controlled factors affecting the pharmacokinetics of the drug. Absorption and metabolism are critical variables. It has been reported that the unbound portion of the tricyclic may vary among individuals, unrelated to dose, from 5.4% to 23% (7, 8). Since only the unbound portion of plasma tricyclic is pharmacologically active, two individuals on the same dose may differ markedly in the amount of the drug that is available to the target tissue. An inverted U-shaped curve relationship between nortriptyline plasma concentration and therapeutic response has been reported (9). This has been described as a "therapeutic window," which was reported to be 50–150 ng/mg for nortriptyline. Below or above these values, patients did not have a clinical response. An inverted U-shaped curve relationship has also been suggested for amitriptyline (10). However, a larger collaborative World Health Organization study showed no relationship between plasma level and clinical response (11). A linear relationship has been reported for imipramine in which there apparently is no falloff of therapeutic effects at higher plasma levels (12). Thus it appears that nortriptyline and amitriptyline are compatible with the concept of a therapeutic window, while imipramine does not appear to have an upper limit for therapeutic effect.

Attempts have been made to evaluate neuroleptic levels in patients and to relate them to therapeutic change. The metabolism of many of the neuroleptics, including chlorpromazine, is so complex, including many active and inactive metabolites, that drugs with a simple metabolism such as butaperazine may be more fruitful for pharmacoclinical response correlations (13–15). Butaperazine was reported to have an inverted U-shaped dose-response curve (16). More recently, a method for stereospecific ^{3}H-spiroperidol binding in plasma has been described that measures dopamine receptor binding (17). This method may be important for determining and comparing therapeutic levels of different neuroleptics as well as measuring total dopamine binding when a combination of drugs is administered.

In the future it is to be expected that more effective therapeutic levels can be achieved through the use of plasma values of tricyclic compounds, neuroleptics, and receptor binding assays. The availability of plasma determinations for amphetamine and methylphenidate in the treatment of minimal brain disorder holds similar promise for eventually arriving at more accurate designations of therapeutic drug levels for a given patient (18, 19).

MAJOR INFLUENCES IN THEORETICAL UNDERSTANDING OF THE PSYCHOBIOLOGY OF THE PSYCHOSES

Increasing emphasis has been placed on ur[illegible] ing the genetic influences on schizophrenia [illegible] fective illness. It has been well documented [illegible] concordance rate for manic-depressive illness [illegible] identical twins is approximately 13%, while [illegible] cordance rate in identical twins is over 60% [illegible] ies of biological and adoptive relatives of schizophrenic [illegible] ics have also indicated the existence of a genetic factor in schizophrenia (21–23).

Over the past decade drugs have been identified that activate the symptoms of various psychiatric disorders. For example, 20 mg of *d*-amphetamine can produce psychotic symptoms in a subgroup of schizophrenic patients (24), but the same dose rarely produces psychotogenic effects in normal individuals or depressed patients. Monoamine oxidase inhibitors and tricyclics can precipitate manic symptoms in bipolar patients (25). Conversely, as noted previously, many psychological agents can decrease psychopathology and block the recurrence of future episodes. It is of interest that all of the drugs which activate or alter psychopathology in man have effects on neurotransmitter systems in brain. For example, amphetamine releases and blocks reuptake of dopamine and norepinephrine presynaptically, whereas the neuroleptics that are effective in treating schizophrenic patients block postsynaptic dopamine receptors.

Data derived from animal studies concerning the mode of action of various pharmacological agents that can increase or decrease psychopathology have guided biological theories about the roles of catecholamines and indoleamines in affective illness. It has been suggested that a functional deficit of catecholamines and indoleamines exists during depression and activity may increase during mania (26–28). Furthermore, it has been hypothesized (29) that an increase in functional activity of dopamine may be associated with some of the symptoms of schizophrenia. There are also pharmacological and anatomical data suggesting an interaction between γ-aminobutyric acid (GABA) neurons and dopamine neurons, and it has been proposed that GABA may also be involved in the pathophysiology of schizophrenia (30).

Drugs are now available for clinical investigation that facilitate the study of the relationship of brain mechanisms to behavioral responses. Drugs with relative selectivity may block enzymes in catecholamine neurotransmitter synthetic pathways, effect storage and reuptake, act as postsynaptic receptor agonists or blockers, or block degradative enzymes. For example, as α-methyl-*p*-tyrosine, which inhibits the rate-limiting step in the formation of the neurotransmitters dopamine and norepinephrine, or piribedil, a relatively specific dopamine agonist, provide powerful pharmacological tools for the further investigation of neurotransmitter hypotheses of psychoses. Further [illegible]

compounds are becoming available that may preferentially affect hypothesized presynaptic rather than postsynaptic receptors (i.e., low-dose apomorphine). Other compounds may affect various steps in the postsynaptic amplification of the received signal from the postsynaptic receptor.

Another area that has had a major influence and has gathered increasing momentum during the past few years involves studies of the role of hormones in psychopathology. It has been well known for many years that hyper- or hypofunction of most of the endocrine systems can be associated with psychopathology. It has also been established that in depressive illness there is an altered pattern and increased excretion of cortisol with more frequent peaks and higher nocturnal levels (31). Some of the important pioneering work in the relationship of ACTH and cortisol to psychopathology and mood was conducted by Braceland (32–36). Endocrine releasing factors and other small CNS peptides previously thought to play a role only in the regulation of peripheral acting hormones have been shown to have receptor sites in the CNS and to have behavioral effects.

It has more recently become possible to measure prolactin released from the pituitary both in plasma and cerebrospinal fluid. This release is partially under the inhibitory control of dopamine; therefore, indirect dopamine agonists such as L-dopa tend to decrease prolactin levels, whereas most neuroleptics that block dopamine receptors produce an increase in prolactin.

Another influence in developing theoretical concepts of psychoses involves the realization that parkinsonian illness can be successfully treated with L-dopa. Since chronic schizophrenia may be due to some profound disruption in brain metabolism or physiology, the search continues to identify anatomical areas of dysfunction in the brain and to find compounds that might reverse this dysfunction.

NEW RESEARCH METHODS

New methodological tools have been responsible for some of the progress that has been made and will certainly contribute to future discoveries in the field of the psychoses. The development of the metabolic psychiatric research ward, concomitantly utilizing longitudinal nurses' rating scales, continuous 24-hour urine samples, plasma samples, and periodic cerebrospinal fluid samples, has had an important influence on the field. Physical activity has always been a confounding factor in most of the biochemical studies in man, and it is now possible to monitor activity on a continuous 24-hour basis in an extremely accurate manner (37). It is also possible to monitor sleep and dreaming parameters in a longitudinal fashion in a number of patients simultaneously. The development of gas chromatography and, more recently, mass spectrometry, radioenzymatic techniques, and radioimmunoassays has greatly increased the accuracy with which scientists can estimate levels of neuroendocrines, amines, and their metabolites in urine, plasma, and cerebrospinal fluid.

The probenecid technique of blocking the exit of acid metabolites from the cerebrospinal fluid has allowed investigators to estimate turnover of various neurotransmitters in brain and spinal cord, and more recent isotope techniques using differential labeling of origins of urinary catecholamine metabolites and carbon-labeled dopamine may provide even more accurate methods (38). The development of models of psychoses in rats and in primates has also provided a way of testing various new drugs and theories as they relate to clinical problems.

One of the most recent technical advances for the potential study of brain function in psychopathology involves the use of emission computerized axial tomography (ECAT scan) in combination with infused 2[^{18}F]fluoro-2-deoxy-D-glucose. This procedure would provide three-dimensional information concerning regional alterations in brain energy metabolism, which would theoretically reflect regional differences in functional neuronal activity (39, 40, and unpublished data from M. Reivich, D. Kuhl, and A. Wolf).

NEW AREAS WITH POTENTIALLY IMPORTANT IMPLICATIONS FOR FUTURE INVESTIGATIONS

A good deal of the work has been completed in searching for genetic markers in psychiatry, particularly in bipolar affective illness, using red-green color blindness and the Xg blood antigen. However, there now seems to be some reason to question the validity of both of these as markers of affective illness (41). Other genetic markers currently under investigation include enzymes involved in the synthesis and degradation of neurotransmitters such as catechol-*O*-methyltransferase (COMT), monoamine oxidase (MAO), dopamine-β-hydroxylase (DBH), HLA antigens, blood groups as markers, and polymorphic markers such as red cell enzymes and serum proteins. Possible genetic factors in pharmacological responses are also being studied. What is needed is a genetic marker that can be applied to each individual in a pedigree and then used to successfully predict which individuals are at risk. The marker must first be shown to be heritable. A homogeneous subgroup of patients with the disorder must be identified on a clinical, biological, or pharmacological basis, and the transmission of the disorder among families must be found to be related to the transmission of the marker. To date, no such marker has been definitively identified. However, this strategy obviously holds great promise for the employment of preventive measures, and an intensive search for such markers is currently under way (42).

Previous work has focused on the presynaptic neuron in attempting to understand the role of synthesis, release, and reuptake in various animal systems and their possible implications for psychopathology. More

recently, various techniques, including receptor binding assays, have been developed to study postsynaptic neuronal receptors in animals and man. Furthermore, drugs with specific actions on specific neurotransmitter receptors have been used, such as dopamine and norepinephrine agonists and receptor blockers. It has also been suggested that it would be particularly useful if one could synthesize neuroleptics that blocked dopamine receptors in one area of the brain and not in another. The development of neuroleptics that did not affect the nigrostriatal area might result in a lower incidence of extrapyramidal side effects or tardive dyskinesia and would be of clinical and theoretical importance. Currently, pharmaceutical companies are actively searching for such compounds. Also, it has been clearly demonstrated in animals that pharmacological agents can produce supersensitive receptors, as manifested by either an increase in receptor sites or an increase in affinity for a compound. The application of the concept of super- and subsensitive neurotransmitter receptors to human disease, to psychopathology, and to the action of drugs may have important implications. For example, it has been hypothesized that tardive dyskinesia may relate in part to the development of supersensitivity following many years of neuroleptic treatment. A further understanding of this phenomenon could lead to new treatments for tardive dyskinesia or successful preventive methods.

Another new and highly preliminary area is the use of renal dialysis to remove hypothesized endogenous psychotogens in an attempt to treat schizophrenia (43). Scientists agree that what is needed at this point are double-blind evaluations of this technique. If it proves to be useful in some schizophrenic patients, then an intensive and probably long search will ensue for the critical ingredient of blood that is removed by dialysis.

Finally, specific opiate receptors identified by chemical binding techniques have been discovered in brain and other tissues (44). A number of peptides, methionine and leucine enkephalin, and β-endorphin as well as nonpeptide opiate-like substances that bind to opiate receptors have been discovered. These discoveries have initiated a search for a physiological role of these opioid substances. The pharmacological effects of opioid alkaloids provide the best clues for the effects of endogenous opioid compounds (endorphins). Since morphine and its congeners affect mood, pain appreciation, sleep, respiration, and release of pituitary hormones, these physiological functions are currently under investigation.

A number of strategies are being used to study possible functions of endorphins in man. These involve the direct administration of endorphins, attempts to measure endogenous opioid substances in urine, plasma, and CSF by radioimmune and radioreceptor assays, attempts to increase the release of endorphins through a variety of techniques, including experimental induction of pain and stress, implantation of electrodes in the brain, and, finally, the use of narcotic antagonists to study the effects of blocking the endorphin systems. The latter strategy is the one that [illegible] used most extensively (44). Two relatively [illegible] cotic antagonists in current use are naloxone and naltrexone, which reverse the effects of endorphins. Thus if endorphins act to regulate [illegible] neurophysiological function, a pure narcotic antagonist administered to man should alter such [illegible]

It has been suggested that endorphins [illegible] changed in schizophrenia, although current [illegible] is inconclusive. It has also been suggested that catatonia seen in animals following the administration of endorphins may relate to neuroleptic-induced catalepsy and to human catatonia. To date one study (45) and two double-blind studies (46, 47) suggested that naloxone may temporarily reduce auditory hallucinations and perhaps other symptoms of chronic schizophrenia. However, five other studies have failed to replicate this finding (48–52).

There are also a number of theoretical [illegible] suggesting that endorphins may play a role [illegible] Opioids produce euphoria that is similar [illegible] phoria associated with mania. It has been suggested that the behavior of cats given morphine may be a model for mania (53, 54). Furthermore, schizophrenic patients experience markedly less pain in response to experimental stimuli than do normal [illegible] which would be compatible with a theory [illegible] endorphins in mania (55). It has been reported that naloxone produced a decrease in some of the symptoms of mania in a small percentage of patients. However, another study found no antimanic effect of naloxone (56). No therapeutic effects of naloxone in depression have been reported to date.

It was also reported in one single-blind [illegible] one of the opioid compounds identified in the pituitary, β-endorphin, may have therapeutic [illegible] schizophrenia and other psychiatric disorders [illegible]. These findings require study in other laboratories under double-blind conditions. If any of these findings is confirmed, even in subgroups of psychiatric patients, an entirely new field will be opened in attempt to understand psychopathology.

In addition to the morphine receptor, several investigative groups have recently identified a benzodiazepine receptor (58, 59). Since benzodiazepines are the most frequently used psychoactive drugs, understanding of how they interact with the brain, what neurotransmitters may facilitate or inhibit their interaction, and, ultimately, whether there is an endogenous "anxiety-related" compound that binds to this receptor is of critical importance in our understanding of anxiety.

CONCLUSIONS

Major advances have been made in our [illegible] crease the recurrent suffering of individuals with chronic manic-depressive or schizophrenic illness through the long-term use of pharmacologic [illegible]

alone or in combination with a variety of psychotherapeutic techniques. The application of plasma levels for the establishment of accurate therapeutic windows may also increase the number of patients who respond to drugs. The mode of action on neurotransmitters in the brain of many of the drugs now used has led to some provocative and productive hypotheses concerning the role of the neurotransmitters dopamine, norepinephrine, serotonin, and GABA in these illnesses. Furthermore, it has been suggested that hormones may play a critical role in modulating the effects of these neurotransmitters. Methodologies such as the development of psychobiological research wards, new instrumentation for the analysis of body fluids, and new models of psychosis all promise the further expansion of our knowledge of these fields. New areas such as the role of polypeptides (including the endogenous opioid compounds) in modulating behavior and the intensive study of neurotransmitters and their receptors may lead to alternative hypotheses and treatments for the psychoses.

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Thoughts on Aging

BY ROBERT N. BUTLER, M.D.

Aging has not received attention in proportion to its effect on the life of every individual. Much of society's reluctance to deal with old age results from "ageism," a personal and cultural dread that stems from a view of aging that sees only the negative aspects. If we are to understand human development, we must learn about its outcome, old age. One means of doing this is to listen to the reminiscences of old people. Psychiatrists can apply their special expertise in working to overcome ageism. Current and future legislative and research efforts can help identify and resolve some of the economic and physical problems of the elderly and thereby improve the quality of their lives.

> Every man desires to live long, but no man would be old.
>
> —Jonathan Swift

AGING IS an intrinsic part of the life cycle. Old age is experienced by an increasing number of people, yet it is still feared and denied by many. In part, this fear stems from ignorance, for aging has not received attention in proportion to its effect on the life of every individual. One manifestation of society's reluctance to come to terms with aging is its pervasive prejudice against the old. Moreover, the triumph of a longer life expectancy in this century is regarded as a problem, and the elderly are often more reviled than revered. Although childhood development has been studied intensively and examinations of death and dying are gaining popularity, until recently aging has been all but neglected by scientists. Can we really expect to understand early development fully without looking at the outcome of that process, old age? As interest in aging grows and the fields of gerontology and geriatrics expand, these issues deserve serious thought.

"AGEISM"

"Ageism" is a profound psychosocial disorder characterized by institutionalized and individual prejudice against the elderly, stereotyping, myth-making, distaste, and/or avoidance. It is to be distinguished from gerontophobia, which is a more rare expression of unreasonable fear, often accompanied by a hatred of older people. Ageism, like racism and sexism, is a way of pigeonholing people rather than viewing them as individuals with unique ways of living their lives.

The manifestations and symptoms of ageism pervade our lives. For example, older people are often excluded from admission to mental hospitals or are indiscriminately transferred from mental hospitals to unprepared communities without adequate services. The availability of services in community mental health centers is limited, and contact between the elderly and psychiatrists in private practice is minimal.

Age discrimination in hiring should be a thing of the past in the federal sector with the passage of new legislation (Public Law 95-256, April 6, 1978). Thanks to the efforts of Representative Claude Pepper (D.-Fla.), mandatory retirement no longer exists in the government, although reservations still exist in the private sector. These reservations mean that in some cases retirement is based on age rather than on competence, need, or desire to work.

Cultural prejudice is exemplified by the use of epithets such as "crock," "crone," and "old biddie." The medical profession has epithets of its own: "serum porcelain level," "turkey," and even "dirt ball." Social attitudes are painfully clear when the old are referred to as "out to pasture," "fading fast," "boring," and "garrulous."

The basis for ageism is personal dread and discomfort at the prospect of growing old, with its presumed concomitants of helplessness, pain, disability, and, ultimately, death. This fear is reinforced by our cultural emphasis on youth and productivity rather than experience and service.

Institutionalized efforts to treat or combat ageism include laws against age discrimination, the phasing out of mandatory retirement, and a return to the historically more common functional retirement based on individual capabilities. Self-education is essential. Much can be learned about the rich knowledge that accrues over the entire life cycle by listening to people review their lives from the special perspective of old age. Didactic reading in the biology of aging, human development, geriatric medicine, and geriatric psychiatry can be supplemented by a sampling of literature, for some of our greatest novelists have been attentive to the life cycle and old age. Cervantes (*Don Quixote*), Tolstoy

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(*Resurrection*), and Shaw (*Back to Methuselah*) come immediately to mind.

Practical experience with patients in settings and under circumstances in which the possibility of effective treatment exists—for instance, in reversible depressions and the treatable reversible brain syndromes—can contribute much toward improving physicians' attitudes toward the old. Psychiatrists, who by virtue of their training are so well equipped to understand transference, can exploit their classic "bag of tricks" to examine and better understand themselves so as to come to terms with their own fears and dread of aging. Psychiatrists can, in fact, take leadership in working to overcome the general cultural negativism toward aging.

RESEARCH ON AGING

What can one hope to accomplish through research on aging? Should the ultimate goal be to extend life, or would this only serve to lengthen the period of decrepitude, depression, frailty, and mental, economic, and physical dependency? Remember the Greek legend of Tithonus, who was granted immortality but not eternal youth.

In establishing the National Institute on Aging (NIA) in the Research on Aging Act of 1974 (Public Law 93-296), the U.S. Congress chose to concentrate on the "alleviation of the problems of old age by extending the healthy, productive middle years of life." The NIA was created for the "conduct and support of biomedical, social, and behavioral research and training related to the aging process and the diseases and other special problems and needs of the aged" (Public Law 93-296, S775, May 31, 1974). Thus, in addition to the support of basic biological research, NIA is involved in studies of the body's great integrative systems—the central nervous system, the immune system, and the endocrine system—to understand better the nature and mechanisms of time-dependent processes such as declining immunity over the course of the life cycle. NIA is also interested in the behavioral nd social aspects of aging and supports studies of ma- psychological problems, sensory changes with age, life, and the differential life expectancy between es and among racial and ethnic groups.

The primary goal of NIA's research program is improvement in the quality of life. This research is not exotic or quixotic—it has clearly practical applications. For example, advances in our understanding of organic brain disease or the progressive loss of immune function would contribute to an increasingly healthy and more vigorous older population. (For more details on planned research in this area, see reference 1.)

Biomedical research at the National Institutes of Health (NIH) began in the 1930s with the study of specific diseases; it is only in the past 20 years that broad non-disease-oriented institutes have developed. Growing interest in behavioral medicine—for exa care and prevention—may shape future dev at NIH. The institutes' various approac study of human development, health, a make the possibility of collaboration intrigu stance, an improved understanding of the over time that appears to occur with agin vide a means to delay the onset, alter the tions, or reduce the severity of certain ag time-dependent diseases.

THE TRIUMPH OF AGE

The fact that so much is made of the lem" is itself an expression of a deep unde ambivalence toward growing old. It is true older people have problems, but this is the dramatic increase in the absolute number proportion of older people in this century. S not prepared for this "demographic revol problems evolved, but they are likely to be adaptations are made. Just as the geograph tion of the fifteenth and sixteenth centuries entific achievements of Newton and Darw tated vast changes in human thinking, so tions, education, industry, and the family, we adapt to the changing population struc

The demographic revolution is largely a r reductions in maternal, infant, and childhoo in Eastern and Western industrialized nati ity is increasingly limited to old age; ap 70%–80% of all deaths now occur after ag

At the time of the founding of the Amer lic, only 20% of newborn babies lived to be trast, approximately 80% now live to be hardly more than 4% of American familie three generations, despite the idealized days" version of the three-generation Am ily, complete with cookie-baking grandm pipe-smoking grandfathers. It is in this ca cause of increased survivorship, that the t tional and even multigenerational family more than a rarity.

In 1920, the chance of a 10-year-old's living grandparents was 40%; today, it is only 44% of women who survived beyond were likely to attain adulthood, marry, hav see those children leave home, spend some time with their husbands, be widowed, an old age. Thus, 56% did not experience the course" of the twentieth century (2).

It is certainly a triumph that this centur in which the opportunity to fulfill the lif been generally available. Life expectancy— tion of how many years an individual of a will live, based on existing actuarial da creased from 49 years in 1900 to 73 years span—the maximum inherent longevity, probably between 100 and 120 years

beings—has not been extended; rather, we have had a greater survivorship and a resulting increase in life expectancy.

NIA and other agencies and individuals involved in research on aging have the task of making it still more likely that people will have the opportunity to flesh out the totality of their life cycles. We need to understand better the 8-year discrepancy in life expectancy between men and women so that we can extend the life of men to the advantage of both. Currently, because of the poverty associated with widowhood, the average woman has about a 60% chance of becoming destitute. We must also examine the limited life expectancy of certain racial and ethnic groups.

We must remind ourselves that when we speak of aging we are not dealing with a problem but with the result of scientific and medical advances that allow more and more people to experience the last chapter—the consummation—of the life cycle.

PSYCHOLOGY OF AGING

Human developmental studies have historically focused only on children, although study of the aged provides a valuable counterpoint because the aged embody the outcome of these developmental processes. Biographies describe how certain people have lived their lives, but no one has systematically examined the entire human life cycle. We know too little about the psychology of the last years of life, and such knowledge would have practical applications. What traits increase survival? What features enable an individual to adapt better to the normal challenges and stresses of life, as well as to accidents and disability? The answers to such questions would not only help us improve the quality of life of the aged, they would also change our thinking about human development and provide new questions for those studying this process. Perhaps this would be the most effective answer to ageism: if we view the life cycle as a whole, if we see ourselves as of one cloth, we can begin to resolve the ambivalent and conflicting feelings that now prevail in relation to old age.

One means for psychiatrists to learn about the psychology of aging is to *listen* to older people review their lives. Life review is a natural process of reminiscence, during which the individual deals with past conflicts and tries to resolve problems. Psychiatrists who have avoided treating the elderly, dismissing their reminiscences as aimless or wandering, are missing a valuable opportunity to learn about the totality of life from those best qualified to discuss it. An added benefit is that the life review of an older person can help the psychiatrist confront and overcome his or her personal fears and denial of aging.

OUR FUTURE SELVES

Regrettably, some older people in society feel obliged to behave according to social expectations, i.e., to conform to society's norms or negative stereotypes. However, there are those in society who provide us with powerful models of what aging should be and of what effective aging is, for effective aging is not the same as aging graciously. Francis J. Braceland, M.D., is quite an extraordinary model from whom we can learn if we can permit ourselves to do so. New knowledge obtained through research on aging and the self-knowledge that comes from interactions with the aged could eradicate the social and personal pathology of ageism. One by-product would be a renewed appreciation of the significance of life, including the later years, of which Montaigne wrote so movingly:

> Especially at this moment, when I perceive that my life is so brief in time, I try to increase it in weight; I try to arrest the speed of its flight by the speed with which I grasp it, and to compensate for the haste of its ebb by my vigor in using it. The shorter my possession of life, the deeper and fuller I must make it.

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Private Psychiatric Hospitals: "Excellence Is Their Watchword"

BY ROBERT W. GIBSON, M.D.

Throughout their history private psychiatric hospitals have been a major force in psychiatry, and their humanistic concern for the mentally ill has resulted in numerous innovations in treatment. Over the past two decades the sharp increase in psychiatric patient care episodes has resulted in a greater reliance on private psychiatric hospitals, and they have responded by modifying their role, broadening the spectrum of patients served, and developing alternatives to inpatient treatment. In addition to continuing to provide high-quality inpatient care, private psychiatric hospitals in the future should expand various alternatives to such care, explore opportunities for collaboration with other health care systems, and provide more patient services to the underserved.

ALTHOUGH private psychiatric hospitals accounted for only 3% of the total patient care episodes in 1975 (1) and have probably never provided more than 5% of that care, they have been and continue to be a major force in psychiatry. During the 1800s a small group of psychiatric hospitals, by their espousal of the moral treatment of the insane, were instrumental in bringing patients out of the almshouses, attics, and prisons. John S. Butler, M.D., who served from 1843 to 1873 as the fourth Superintendent of the Hartford Retreat (now the Institute of Living), was a leader in the movement toward humanitarian care of the mentally ill. Describing his visit in 1842, the year before Dr. Butler moved to the Hartford Retreat from the state hospital in Boston, Charles Dickens marveled that

> every patient in this asylum sits down to dinner every day with a knife and fork, and in the midst of them sits the gentleman [Butler] whose manner of dealing with the charges I have just described. . . . At every meal moral influence alone restrains the more violent among them from cutting the throats of the rest: but the effect of that influence is reduced to an absolute certainty and is found even as a measure of restraint to say nothing of it as a means of cure, a hundred times more efficacious than all the strait waistcoats, fetters, and handcuffs that ignorance, prejudice, and cruelty have manufactured since the creation of the world. (2, p. 87)

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In 1948, when Albert Deutsch wrote *The* [illegible] *the States* (3), this same humanistic con[illegible] private psychiatric hospitals provided [illegible] that the mentally ill deserved more than th[illegible] warehousing and neglect to which they were [illegible] gated. Some private psychiatric hospitals [illegible] major programs of research, and virtually [illegible] novators in applying the newly emerging [illegible] their treatment programs. As freestanding [illegible] training centers or in affiliation with acade[illegible] many private psychiatric hospitals offered [illegible] an opportunity to learn from intensive w[illegible] small number of patients under the tutelage [illegible] enced clinicians. As treatment services inc[illegible] patterns of care changed, private psychiatr[illegible] frequently in collaboration with newly dev[illegible] cilities, continued as models of excellence [illegible] to serving the patient. Guided by Francis J. [illegible] M.D., the Institute of Living continued to [illegible] standing example of excellence.

A CHANGING MENTAL HEALTH DEL[illegible] SYSTEM

Action for Mental Health (4), the repo[illegible] Commission on Mental Illness and Healt[illegible] to several important changes in health ca[illegible] during the past two decades: a significant [illegible] the number of psychiatric units in gener[illegible] the development of community mental he[illegible] with federal support, and sharp decreases [illegible] ber of beds in state and county mental ho[illegible] tient care episodes[1] more than quadruple[illegible] from 1.7 million episodes in 1955 to 6.9 mil[illegible] The rate of patient care episodes per 10[illegible] lation tripled, increasing from about 1,000 [illegible] 3,000 in 1975. Inpatient episodes accoun[illegible] of the total in 1955 but only 27% of the to[illegible] with 70% outpatient and 3% day treatment [illegible] episodes (1).

Despite these increases total admissions [illegible] care episodes still fall short of the estimate [illegible] its preliminary report of September 1977 (5, [illegible] dent's Commission on Mental Health est[illegible]

[1]Patient care episodes are defined as the number of [illegible] patient facilities at the beginning of the year (or the [illegible] sons on the roles of non-inpatient facilities) plus the [illegible] missions, readmissions, and returns from long-term [illegible] the year.

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"between 20 and 32 million Americans need some kind of mental health care at any one time" (p. 4). As a consequence,

> The current direct cost of providing mental health services is about $17 billion a year. The social cost, when measured in terms of lost wages and a shortened life span, is estimated to be another $20 billion. (p. 4)

And beyond these statistics,

> There are millions more who seek help for emotional problems elsewhere, especially from their personal physicians or from health care clinics. For example, 15 percent of patients seen in general medical practice are found to have psychiatric or emotional problems. At any given time, 25 percent of the population is under the kind of emotional stress that results in symptoms of depression or anxiety. (pp. 4–5)

The increase in patient care episodes should continue. The need is there, and progress is gradually being made to overcome the stigma that impedes the treatment of mental illness. The trend toward greater reliance on community mental health centers, general hospitals, and private psychiatric hospitals is appropriate. The financing of mental health care is grossly inadequate but is improving slowly. Improved financial resources should reduce the dependence on government institutions, and state mental health programs are making a deliberate effort to shift care toward the private sector. Private psychiatric hospitals are already modifying their role and can be expected to do so without compromising quality.

CHANGES IN PRIVATE PSYCHIATRIC HOSPITALS

Until 1972 the numbers of patients served by private psychiatric hospitals and the distribution between inpatient and outpatient services were relatively stable. Bethel (6) reported that during the period 1968–1971 the number of hospitals stayed at about 150. The number of inpatients in residence at year-end varied only from about 10,200 to 11,000; annual admissions fluctuated from a low of about 87,000 in 1970 to a high of 92,000 in 1969. Changes in the distribution of admission diagnoses were slight, with depressive disorders accounting for approximately 40% of the total and schizophrenic reactions for 20%. However, there was a trend toward the admission of younger patients; patients under age 25 at the time of admission constituted 18% of all admissions in 1968 and 24% in 1971.

Substantial changes began to occur during the period 1972–1975, when the number of hospitals increased to 180, the number of inpatients in residence at year-end rose 10% to about 11,500, and annual admissions rose to 130,000. The growth in the number of hospitals was due largely to new investor-owned hospitals. By 1975 nearly two-thirds of all private psychiatric hospitals were operated by corporations, individuals, or partnerships on a for-profit basis, with the other third operated by not-for-profit groups, including church foundations. The greatest number of hospitals and beds was concentrated in the mid-Atlantic region, with the smallest number in the Northwest. Private psychiatric hospitals in New England had the greatest number of beds per 100,000 U.S. resident population. About 15 states had no private psychiatric hospitals, and 11 states had only 1; California had 27, and New York had 14 (7).

Although private psychiatric hospitals are organized to provide inpatient care, alternatives to inpatient treatment are being developed. Between 1971 and 1975 admissions to private psychiatric hospital outpatient programs increased 80%, day treatment, 67%; and inpatient care, 44% (8). By 1975, of a total of about 180 private psychiatric hospitals, 42% provided day treatment; 35%, outpatient care; 24%, emergency services; and 8%, halfway houses (9).

CHARACTERISTICS OF THE PRIVATE PSYCHIATRIC HOSPITAL—1975

In an effort to clarify the role of the private psychiatric hospital in the mental health delivery system, the National Institute of Mental Health and the National Association of Private Psychiatric Hospitals jointly conducted a survey of one month's admissions to private psychiatric hospitals (10). This study yielded information from three-fourths of the hospitals surveyed, making it reasonably representative of the total. During May 1975, the month surveyed, 7,864 people were admitted to the inpatient services of 139 private psychiatric hospitals.

Of the total number of patients admitted, 42% had depressive disorders; 22% had schizophrenia; 9% had alcohol disorders; 6% had adjustment reactions of adolescence, adulthood, or late life; and 5% had personality disorders. The remaining 16% suffered from drug disorders, organic brain syndrome, childhood disorders, and neuroses excluding depressive states. About 45% of the patients admitted were male and 55% female. The trend toward younger patients continued, with 27% under 25 years of age, 37% aged 25–44, 27% aged 45–64, and 10% 65 years old or older (10).

The survey indicated that the three major referral sources of all inpatient admissions were private psychiatrists (44%), followed by self, family, or friends (24%) and by other physicians (11%). Approximately three-fourths of the patients admitted reported previous psychiatric care. Thirty-nine percent had received previous inpatient care in the same hospital to which they were admitted, and 30% had received inpatient care in another psychiatric hospital (10). About 3 out of 10 patients had seen private psychiatrists, and more than 1 out of 10 had had other types of noninpatient psychiatric care. This suggests that most of the patients admitted posed serious therapeutic prob-

lems and many had been treatment failures.

The types of psychiatric treatment most frequently received by the patients in the survey were individual psychotherapy (90%), drug therapy (83%), group psychotherapy (61%), family therapy (20%), and electroconvulsive therapy (8%). Ninety-four percent of the patients received two or more types of treatment, and about 70% received three or more types. The median number of days spent in the hospital was directly related to the number of treatments, presumably because of the greater severity of illness in those patients receiving multiple therapies. Patients receiving one type of treatment stayed a median of 8 days, two types 17 days, three types 21 days, four types 25 days, and five or more types 31 days (10).

Staff-patient ratios are too gross to be an indicator of the quality or adequacy of treatment programs. Many factors are not taken into account in such an index—for example, the patient mix in terms of age, diagnosis, or severity of condition, and the type and intensity of the program. Nevertheless, comparisons of staff-patient ratios for the various types of mental health facilities do illustrate major differences in the intensity of patient care and give some indication of the level of care in private psychiatric hospitals.

According to Taube and Witkin (11), in January 1974 the staff-patient ratio for all private mental hospitals in full-time equivalents of staff per 100 average resident patients was 204 total staff, with 124 patient-care staff (66 professional and 58 other). Not-for-profit private hospitals had a ratio of 238 total staff compared with 173 total staff in investor-owned hospitals; there were 140 patient-care staff in not-for-profit hospitals compared with 110 in investor-owned hospitals. Within the nonpublic general psychiatric hospital units the total number of staff per 100 patients was 130, with 115 patient-care staff (65 professional and 50 nonprofessional). In state and county mental hospitals the total staff per 100 patients was 88, with 57 patient-care staff (18 professional and 39 other).

Of the May 1975 admissions to private psychiatric hospitals, about 95% of the patients were discharged in 4 months or less. Over 90% of all patients were discharged within 12 weeks, with 23% in 7 days or less, 17% in 8–14 days, 25% in 15–28 days, 14% in 29–42 days, and 13% in 43–84 days. Of the patients discharged, about half had been referred to private psychiatrists for further treatment and one-fourth to outpatient psychiatric clinics or services (10).

In summary, about two-thirds of the patients admitted had depressive disorders or schizophrenia. Over half were referred by psychiatrists or other physicians and one-fourth by self, family, or friends. Most patients had had previous psychiatric treatment, often inpatient care. Psychotherapy (individual, group, and family) and drug therapy were the principal treatment modalities, with the vast majority receiving multiple therapies. Staff-patient ratios were generally higher in private psychiatric hospitals than in other mental health facilities, with the not-for-profit hospitals highest at 238 total staff and 140 patient-care staff [illegible] patients. About two-thirds of the patients [illegible] charged within 4 weeks and 90% within 12 [illegible] three-fourths referred for continued treatment

A PLURALISTIC SYSTEM

At this juncture there is no reason to believe that one type of facility is in all respects superior to another. Each has its strengths depending on the [illegible] and characteristics of the institution. For this [illegible] pluralistic approach is in order and will probably continue indefinitely. However, it is helpful to [illegible] the differences among facilities in order to [illegible] how each can best serve the patient.

In the psychiatric units of nonpublic general hospitals, inpatient services are stressed and about [illegible] the patient care episodes involve this treatment [illegible]. Treatment is usually short-term, with an emphasis on crisis resolution and reliance on somatic therapies. The ready availability of surgical and general medical services gives the psychiatric unit of the general hospital a particular advantage in the treatment of patients with interrelated physical illness, psychiatric complications of medical and surgical conditions, somatic psychosomatic illnesses, substance abuse requiring detoxification, and severe organic disorders. Because the attending psychiatrist can follow a patient through modes, including inpatient, outpatient, and [illegible] ment, continuity of care is enhanced. However, physical space, facilities for activities, and [illegible] treatment services are often limited, particularly in smaller units. Hospital support services designed for the needs of medical and surgical patients are not always well suited to the needs of psychiatric patients, particularly those requiring intermediate or [illegible] care. Daily costs are usually higher than in [illegible] hospitals, but this is offset by generally better third-party coverage. Some patients and families find treatment more acceptable in the psychiatric unit of a general hospital because these units are in the mainstream of the medical care system.

Community mental health centers place emphasis on outpatient and day treatment (about [illegible] 7%, respectively, of patient care episodes) [illegible] as alternatives to hospitalization. As an organized system of care, most provide at least the five services initially required for federal funding (emergency, outpatient, day treatment, emergency, and [illegible] tive services). They are expected to identify [illegible] the needs of the residents, particularly [illegible] served, within a defined catchment area and [illegible] for early and prompt intervention. There [illegible] liance on psychiatrists, with corresponding greater use of other mental health professionals and [illegible] professionals. Matching funds helped community mental health centers get started, but severe fiscal [illegible] have been created by the withdrawal of the funds. Somewhat belatedly, the centers are looking [illegible]

sources of financial support such as Blue Cross, commercial carriers, and personal payment.

State mental health systems, particularly state hospitals, have come under intense pressure as the cost of health care has escalated and budgets have not kept pace. This has caused an erosion of resources available to the hospitals and has provided only limited funds for new outpatient and day treatment services in the community. Deinstitutionalization programs all too often have been imposed by politicians with the intent of cutting costs rather than meeting the needs of patients. The leaders of many state mental health programs have recommended that rather than being direct providers of care, states should assume the initiative in identifying needs, planning, coordinating, determining priorities, setting standards, allocating resources, and evaluating. Despite the problems, many state programs are excellent. As direct providers of care the state systems potentially are in the best position to provide programs that fill needs not currently met by the private sector. In many instances these efforts can be enhanced by collaborative arrangements with private hospitals, general hospitals, and community mental health centers.

ROLE OF THE PRIVATE PSYCHIATRIC HOSPITAL

The main purpose of the private psychiatric hospital is to serve the patient. Many private hospitals, as collateral purposes, engage in teaching, research, and community consultation. Most private psychiatric hospitals strive for excellence that goes beyond simply meeting needs at an acceptable level of quality. As a consequence, they have, over the years, served as models against which to measure other programs.

In private psychiatric hospitals, treating the mentally ill is not simply one of many specialty services as it is in the general hospital. Even though private hospitals are expanding day treatment and outpatient programs, the emphasis continues to be on the inpatient treatment of individuals with severe psychiatric disorders, whereas community health centers stress emergency, crisis intervention, outpatient, and day treatment services. This allows the private psychiatric hospital to maintain programs to meet the needs of patients with more severe psychiatric disorders that require more specialized care.

Because of the emphasis on the treatment of psychiatric patients, the private psychiatric hospital is so organized that the institution becomes a therapeutic instrument in its own right. There has been a natural evolution from the moral treatment of the 1800s to the therapeutic community of the mid-1900s. The private psychiatric hospital provides the setting and resources for a coordinated treatment program in which most patients receive multiple therapeutic modalities. The potential is greater for definitive treatment beyond crisis intervention and symptom removal.

The private psychiatric hospital has an advantage over other types of facilities in preserving a direct relationship with the patient. A higher degree of confidentiality can be maintained despite the encroachment of some third-party payers and government requirements. The greater freedom of choice enhances the patient's capacity to develop a therapeutic alliance. The direct relationship to those they serve has made private psychiatric hospitals more directly accountable to the patient than is usually possible in large public systems dominated by the political process and government bureaucracy.

As part of the private sector, private psychiatric hospitals have relied on revenue from patient care as their major source of funding. Of the total admissions to private psychiatric hospitals in 1975, the principal sources of payment were as follows: Blue Cross, 37%; commercial insurance, 30%; Medicare, 11%; other types of government payment, 10%; and personal payment, only 6% (12). This reliance on revenues from services to patients has made private psychiatric hospitals cost conscious and has stimulated the development and refinement of management services. As part of the private sector, they have retained close ties with the majority of private psychiatrists, some 2,000 of whom serve as staff and resident psychiatrists, with several thousand more on attending or courtesy staffs.

Private psychiatric hospitals have struggled against such external regulation as the growing requirements of health planning, certification of need, and government third-party funding. The constraints are considerably greater than they were 10 years ago, but the private psychiatric hospital still has a greater flexibility and opportunity for self-determination than do most other psychiatric institutions. With its multiple funding sources, the private psychiatric hospital has greater latitude in responding to the dictates of third-party payers. Addressing the National Association of Private Psychiatric Hospitals, Dr. Braceland noted, "Government money is 'soft money,' and when economy programs are in vogue that money disappears just as government training and research money is disappearing now. Your hospitals are immune, for they should be dependent upon themselves" (13). Individually, and through areawide and national associations, private psychiatric hospitals have been vigorous advocates and effective leaders in influencing the funding policies of government and private insurers.

THE FUTURE

In the future, inpatient care of high quality for children, adolescents, and adults should continue as the hallmark of the private psychiatric hospital. Major expansions should be made in outpatient and day treatment programs. More specialized alternatives to inpatient care, such as halfway houses and community services, are needed. To be more accessible and responsive to community needs private psychiatric hospitals should initiate more diagnostic, evaluation,

emergency, and crisis intervention services. The spectrum of patients served should be broadened by greater emphasis on alcoholism programs, active treatment for the elderly, programs for the drug abuser, and special education for the mentally disabled. Greater involvement is needed in preventive programs of 1) consultation to community agencies, 2) public education, especially for those who influence others, such as executives, teachers, labor leaders, judges, and politicians, and 3) counseling, school guidance, and vocational rehabilitation.

Private psychiatric hospitals should participate actively in continuing education for mental health professionals and place greater emphasis on in-service training as a method of maintaining and improving the quality of psychiatric care. In keeping with their emphasis on excellence they should utilize, refine, and assess the new techniques for quality assurance. Private psychiatric hospitals can make significant contributions to research, particularly in the application and evaluation of newer therapeutic approaches. They should explore the opportunities for collaboration with public and private agencies, general hospitals, and state mental health systems, as well as with other organized health care systems such as health maintenance organizations and community mental health centers. As more resources are made available through government and private insurers, private psychiatric hospitals should strive to provide more patient services to the underserved.

As Dr. Braceland has so succinctly put it, "As to the future of the private mental hospitals, it is bright providing excellence is their watchword" (13).

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History and the Psychiatrist

BY DAVID F. MUSTO, M.D.

Historical consciousness is a valuable tool in psychiatry. History can be useful to psychiatrists by expanding their awareness and revealing general errors in the content of psychiatric knowledge, detecting drastic distortions that appear in the ways knowledge is applied to patients, helping to avoid mistakes in public policies that involve psychiatry and result from erroneous impressions of social history, and alerting us to possible future trends. Although history does not give the answers—rather, a complex context within which to seek them—it still has value for the psychiatrist.

> If psychiatry is to take its proper place in the science of man, it must be aware of its limitations and realize that it is only a part of this science, an important but a small part insofar as the general knowledge of man is concerned. To forget this is to run the danger of scientific imperialism . . . the tendency, encountered regularly in the history of knowledge, to credit a special discipline with universal significance. The final result of such enthronement is always the catastrophic dethronement of the apparently supreme branch of knowledge. . . . The same danger faces psychiatry, if its popularizers become too enthusiastic or its enthusiasts become too popular.
>
> —Francis J. Braceland (1)

FRANCIS J. BRACELAND, M.D., exemplifies the observation that "great medical leaders [are] fully aware of the value of historical studies" (2, p. 32). He looks to history as a means for placing psychiatry in perspective and into the context of the rest of society, for overcoming what he called in 1945 "one of our difficulties in psychiatry . . . the tendency at times to operate in a vacuum" (3). He has examined the past with respect and humility. His respect arises from a deep interest in history as history as well as from the knowledge that the materials of history are so fragile and random that a preconceived form can be imposed on them which does not illuminate the present but becomes only one more self-serving element in contemporary controversy.

The understandable temptation to affect present policies and practices is likely to distort a historical account. Another cause of distortion is dogmatic certainty on the part of a psychiatrist or other student of history that he or she knows the answer to contemporary policy questions. For such individuals, the past becomes a storehouse of illuminating incidents, and convenient proofs are not difficult to extract from an otherwise complex collection of opinions and raw data. Theoretical or ideological certainty is a sure guide, and its possessor an assured investigator. Therefore, those who are certain of contemporary injustice—or justice—may search the past and easily believe they have found confirming evidence that they can label with the objective emblem of "history." At the very least, history is filled with usable anecdotes.

In the nineteenth century, history was adduced to prove, when taken with the certainty of clinical and "scientific" judgment, that the Irish or blacks were inherently inferior. In the twentieth century, historical evidence has been chosen to "prove" that psychiatry imprisons people and molds their behavior in order to assure social conformity. Of course, in the latter instance, "psychiatry" suffers from the same holistic interpretation that too often is applied to "history." For better or worse, there is no more a unified entity of "psychiatry" than there is one of "history"; beyond some broadly shared aims within each profession, the practitioners are disparate. Generalizations about either discipline are difficult.

When a psychiatrist approaches history without some sense of psychiatry's current limitations, psychiatry suffers as much as history. The opportunity to question contemporary assumptions, both practical and theoretical, is lost when the past is shaped with a heavy hand and tidy theoretical formulae are imposed on superficially familiar events. The assumption is that truth is to be found in contemporary knowledge—that contemporary beliefs are universally true and can be applied to events that took place in other times and places. With this confidence in ourselves and our times, to study history means merely to harmonize rules of behavior with chaotic bits of information.

A related assumption is that history, whether of psychiatry or not, is not relevant to decisions regarding psychiatric practice or policies today. All of the information and political wisdom required for the best pos-

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sible decisions is present in a narrow band of time extending back no farther than the lifespan of the eldest in a group or bureaucracy. To believe that the present day has the elements for answers to social problems is rarely a conscious rejection of history: it is more often a simple lack of awareness that prevents us from considering the past beyond our own memories.

More than most other medical specialties, psychiatry is closely entwined in the social and cultural mores of our era. Furthermore, the profession has not consistently demonstrated a self-reflective power to see clearly how it is influenced and, at times, severely distorted by forces within society. The failure to study the vicissitudes of psychiatry over time only diminishes awareness of how distortion may occur. Similarly, ignorance about cross-cultural psychiatry tends to overemphasize the importance of our own cultural expressions, leading us to perceive as universal those norms of behavior which are in fact characteristic only of our own place.

THE VALUES OF HISTORICAL PERSPECTIVE

To gain perspective on our own place and time is one of the values of a historical consciousness. Whether there is a more practical application depends on the issue addressed. Sometimes this perspective appears to be its own reward. At other times, history may delay or discourage action, although this is not necessarily an unfortunate result. As the late Richard Hofstadter cautioned, "History forces us to be aware not only of complexity but of defeat and failure: it tends to deny that high sense of expectation, that hope of ultimate and glorious triumph, that sustains good combatants. There may be comfort in it still" (4, p. 466).

In the last two decades there have been many studies that are models of sensitive reconstruction of psychiatric history, although not without application to the present. Prof. Gerald Grob of Rutgers University has published two studies, *The State and the Mentally Ill* (5) and *Mental Institutions in America* (6). Among several major contributions to the history of medicine and psychiatry are Prof. Charles Rosenberg's *The Trial of the Assassin Guiteau* (7), Norman Dain's *Concepts of Insanity in the United States, 1789–1865* (8), and Dain's later *Disordered Minds* (9). These books were written by professional historians, not psychiatrists. Along with other monographs they give evidence that those outside the psychiatric profession are becoming increasingly interested in the history of psychiatry and of the social history that embraces psychiatry. Although they deal primarily with the history of psychiatry, these studies are directed not just to psychiatrists but also to the public, policy makers, and, not infrequently, the most vociferous critics of psychiatry.

Historians' attraction to what not long ago would have been termed the history of psychiatry and the almost undisputed province of historically minded psychiatrists has been a mixed blessing. Just as [illegible] psychiatrists who have examined the past with preconceived ideological positions to attack or defend, so there are historians who have taken the past as a convenient collection of illustrations and "facts" with which to attack practices that they abhor in the present system or in psychiatric practice. Important critiques of psychiatry based on the past and written from psychiatrists include David Rothman's *Discovery of the Asylum* (10) and Michel Foucault's *Madness and Civilization* (11). Rothman, for example, sees a movement during the Jacksonian period to build institutions, such as asylums and prisons, as a means to impose conformity through rehabilitation in a controlled environment. Rothman sees invalid commitment of the deviant and dependent as growing, indeed a dominant, trend in the nineteenth century and into the twentieth century. The connection between this historical analysis and attacks on current procedures for psychiatric and criminal incarceration is obvious. What may at first seem an analysis of a distant period, maybe even a quaintly antiquarian study, becomes the basis for a broad attack on contemporary psychiatric practice and the conscious and unconscious intentions of psychiatrists.

Prof. Barbara Gutmann Rosenkrantz, another social historian, has criticized Rothman for a "failure to distinguish between intent and consequences" that leads to the conclusion that beneath a possibly disingenuous naïve reformist rhetoric lay a conscious repressive motive which, when balked, led to a conspiracy to control" (12). Criticisms such as Rothman's probably do not sound antiquarian to a psychiatrist today and are examples of the increasing importance of historical studies in defining psychiatry's role in our future. Psychiatry as a profession has a vested interest in preserving historical records and encouraging fair and thorough scholars to describe the past in detail and accurately. This desire for the best possible historical research is not just a humanistic altruistic joy in history as a pleasure in itself—it is also practical. Fine studies completed or underway by such psychiatrist-historians as Eric Carlson, George Mora, Henri Ellenberger, Otto Marx, and [illegible] Braceland (a fraction of those who could be named) have become a base for further social and professional histories of psychiatry in the United States. Psychiatric training, although it is not a prerequisite for research in psychiatric history, helps one to understand the problems and nature of psychiatric practice so that documents, articles, and other records can be more fully interpreted. Psychiatrists should make available their professional expertise to historians working in these fields.

The connection between the history of psychiatry and everyday practice and planning for the future of the profession may at times appear tenuous. However, the importance of a historical perspective in making decisions affecting psychiatry and the use of historical studies to define psychiatry's role in society and the

social issues with which psychiatry is associated are reasons that ought to impel any foresighted residency program to include courses in the history of psychiatry and American social history.

USEFULNESS OF HISTORY TO THE PSYCHIATRIST

History does not often provide specific answers to contemporary or future problems. When an answer to a problem has been found, it is not usually forgotten: penicillin is still popular for pneumococcal infections. The more common pattern is for solutions to recur and alternate, particularly when the problems cannot be or have not yet been solved. I suppose most of us will agree that we have a characteristic way of filtering incoming stimuli and limiting interpretations of the stimuli to a relatively few formulations. Within our own culture, explanations given for what has happened appear so reasonable that it does not seem necessary to worry about perceptions from another time or place.

History nudges us out of these adjustments to our surroundings, now and then to a useful extent. The occasions when a psychiatrist might find history useful can be categorized in at least four ways: 1) knowledge of the past can expand our awareness by revealing gross and general errors in the content of psychiatric knowledge, 2) drastic distortion can be detected in the accepted manner in which this knowledge is applied to patients, 3) false impressions of social history can result in errors in public policies involving psychiatrists, and 4) social and intellectual history is especially valuable today in alerting us to possible dangers we may face in the near future as psychiatry moves closer, for example, to biological determinism.

Examining Past Errors

Obvious errors in the content of psychiatric knowledge can be found in descriptions of social questions, minorities, and kinds of "mental illness" that rely for their authority on the clinical judgment and the special language of psychiatry—which is not, of course, to say that the record of psychiatry is one of consistent error. When psychiatry does err, at least when viewed in retrospect, the mistake frequently appears in confirming contemporary attitudes in the spirit of a neighborly social institution. Examples are found in the nineteenth century that are rarely debated by psychiatrists now. In the South, claims of inherent inferiority of blacks and the dangers to them of emancipation could be matched in the North by studies of Irish immigrants who were also claimed to suffer from a "constitutional inferiority" (13).

Dismissing these errors as being so far in the past that they are irrelevant to psychiatrists today would be to lose the value of uncovering these past beliefs. It is not that psychiatrists now have a responsibility for beliefs held in the past but that social conformity is a danger to the scientific role and task of psychiatry today. So far, no drug, treatment, program, or law has exempted the profession from the subtle and potentially dangerous effects of having one foot in science and the other in the marketplace. Although criticism from within and without the psychiatric profession is not currently lacking, sensitivity to distorting forces are better understood when the defensiveness and polemics of the issues have long passed. In this sense, the more temporally remote or the less relevant the subject matter of a historical study, the more useful may be the lesson to be learned for our own debates and motivations.

Dealing with Distortions

Some of the ways in which the consensus of psychiatric opinion has been false are documented by history; even the recent past gives evidence that the profession's usual standards of operation, the "delivery system" as we now term it, can undergo dramatic shifts under extreme political pressures. If there is one canon of psychiatric ethics held in the highest respect, it must be that of preserving confidentiality. Psychiatrists have gone to jail rather than reveal patients' statements. Psychiatrists' offices have been burglarized because people wanting information have recognized the hopelessness of obtaining it in any other way. Some psychiatrists keep almost no record of their patients' statements; others disguise their progress reports in an almost incomprehensible jargon. Yet, in the early 1950s, prominent psychotherapists did provide to an academically operated "Appeals of Communism Project" information on patients who had been members of the Communist Party (14).

The therapists were selected "by reputation, position, or published papers . . . in social science research" (14). Thirty-five cases were studied intensively and were provided by the therapists chosen in this responsible manner. About half the therapists declined to provide cases, but "whether their refusal was based on lack of pertinent case records or on lack of willingness to cooperate" (14) was unclear to the project's directors. There was no statement published indicating that these patients had consented to this use of their histories, although the published checklist of information requested from the therapists includes categories that would easily identify the patients to anyone knowledgeable about their party careers or public accomplishments. This request from competent and respected investigators must have been flattering, for the purpose was nothing less than to determine the family constellation that bred Communists and the defensive role of Communism in the lives of Communists. That the nation appeared to be in grave danger may have justified this apparent breach of confidentiality, which at a later time might seem strange. If we take this incident merely as another example of how we have progressed, we conveniently ignore the humbling continuity we have with the past.

The Influence of Social History

Moving from the history of psychiatric opinion and practice to a broader field that we might name "medical history" or "social history," many examples of inaccurate or inadequate accounts of the past that have affected later generations may be found or at least suspected. One example in drug abuse policy is the famous historical precedent of the British system. According to the most devout American believers in the British system, the British had a serious heroin addiction problem after World War I. The Dangerous Drug Act of 1920 was instituted and interpreted to permit the supply of heroin to those addicted. The contrast is often made with the reactionary United States, where prison sentences were being meted out to addicts, while in humanitarian Great Britain they were supplied with the drug. One of the American proponents of this system, Alfred R. Lindesmith, granted in 1947 that "at first glance it might seem that [the British System] would make opiates more available than they are now and lead to a spread of the habit (15, p. 205)." Professor Lindesmith went on to say that the opposite was the case.

In 1972, Brecher and the editors of *Consumer Reports* (16) supported this account of history in *Licit and Illicit Drugs*, and it acquired even more credibility because it was published as a Consumers' Union report. The results of providing heroin to addicts were described as magnificent: "By 1935, the United Kingdom reported to the League of Nations that there were only 700 addicts left in the entire country" (16, p. 121).

Having been educated in this belief, I found the reality to be a surprise. The first correction to what has become a traditional account in America is that the British did not enact the Dangerous Drug Act because of any heroin problem in the United Kingdom. The 1920 act was in response to Article 295 of the Versailles Treaty, which required enactment of narcotic control laws within 12 months from the coming into force of the Treaty; ratification of the Versailles Treaty would also simultaneously signify ratification of the Hague Opium Convention of 1912 (17). This origin would help explain the complaint made in Parliament that the Dangerous Drug Act was much more appropriate for America, where there was a serious narcotic problem, than for the United Kingdom.

Examining the records of the Rolleston Commission in London, I found no evidence of a heroin addiction epidemic or even recognition of a high level of heroin use. The United States, especially New York City, was seen as the example of serious heroin addiction. The records of the New York City Department of Health reveal that almost 5,000 heroin addicts were registered at the city clinic in 1919–1920. Their mean age was in the early 20s. The Rolleston Commission a few years later searched for heroin addicts in Britain who might be studied (18). The dusty documents and obscure journals did not support what I had been told and what I read in medical school or residency.

The British had accepted maintenance of h... addiction because there was so little addictio... United Kingdom, just as the United States h... until growing numbers of addicts alarmed the p... The details of each "system" and their beginn... velopment, and our memory of them cannot b... ciated or sketched in a short space. All that i... of the reader is to suppose that there were v... heroin addicts, hundreds at most, in Great B... the 1920s, but that the policy of giving some ... addicts legal heroin was believed, in another ... and in a later generation, as being the reaso... low numbers of addicts rather than the re... allowing some maintenance. Then consider ... specious explanation is given as an argument ... ing heroin easily and legally available in th... States for a population of addicts estima... minimum of 250,000.

Having granted the assumptions, the reade... agree, I trust, that whether this is true or not ... significance to American policies regardi... abuse. Yet I have had the impression, after ... quite a few accounts of the history of drug ab... cies, that the issue is so divisive and the propo... convinced of their positions that careful hist... search is bypassed for narratives the author ... must be true because they fit their own pref... answer to the drug problem. The firm conv... the truth is known does simplify historical in... tions; it may also destroy their value.

Using the Past to Deal with the Future

The last of the four ways in which study o... may benefit psychiatrists today is as a guide t... ture. That statement must be quickly qualifie... history does not foretell the future. It can, howe... sitize us to the issues, alternatives, and resu... have occurred in the past. For example, a ... threat to national security like that in the 195... probably again severely strain confidentiality b... physician and patient. A more likely candi... close study from historical analysis is the trend ... chiatry and other social sciences toward a gr... gree of biological determinism in interpreting ... ual and group behavior.

Hereditarian explanations for behavior ha... popular in the United States in the past; they w... dominant mode of explanation for scientists a... cians until well into this century. Research in ... netics and constitutional characteristics shou... prevented on the grounds that the conclusio... be misused, but there is great need to become ... the ways in which hereditarian theories can ... applied and overapplied. In the past, biologi... minism has proven exceptionally attractive t... therapists but, more importantly, to policy m... the public. Complex social questions are set... more easily when approached from either ... sively environmental or hereditarian perspe... add to other worries about the results of this ...

must note that psychiatry and the social sciences in America have been exquisitely susceptible to programs based on research, which indicates that final proof for their validity is just around the corner.

COMMENT

Reviewing the various ways in which history can be recommended to the psychiatrist, I recall again Richard Hofstadter's caution about expecting too much from the study of the past. As the examples I have given suggest, history does not so much provide eagerly sought answers as a more complex context in which to make decisions. There may be comfort in it still.

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The Illusion of Simplicity: The Medical Model Revisited

BY HERBERT WEINER, M.D.

Traditional medical models have been found to be linear, restrictive, and oversimplified. Only a truly biological model, encompassing evolutionary as well as molecular and cellular biology, can account for the complex origins, forms, and effects of disease, which are illustrated by a discussion of hepatitis B and slow virus disease. An updated biological model of disease takes into account predisposition to disease, the timing and route of infection, multiple disease forms, variable adaptive response, and the role of social and cultural factors and views disease as a failure of adaptation in one or more systems. Its application to psychiatry is shown in a discussion of stress, bereavement, and separation.

FOR REASONS that resist explanation, psychiatry has once again asked for admittance into medicine. Over the years, psychiatry has repeatedly wandered off along paths that seem to many doctors as remote as exobiology is from terrestrial biology. These excursions into therapy for societies, the improvement of mental health or of international relations, and the diagnosis from afar of psychopathology in the body politic and politicians, for instance, seem premature, if not innocent, to the medical profession.

Therefore, psychiatrists are being called back to their origins: we must be doctors to our patients' minds and bodies, not to the ailing body politic. We are trained as biologists; we diagnose illness, and we care for the sick. We hope to alleviate suffering; occasionally we cure. These ancient concerns of physicians go far beyond the legal right to prescribe drugs, and many psychiatrists have never broken with this tradition. However, others have wished to discard—or at least go beyond—the traditional roles that society has assigned to them because they are discontented with the medical model.

TRADITIONAL MEDICAL MODELS

Recent discussions about the medical model (1–4) have generally not mentioned that there is no one model or have suggested that a new model is nee[illegible] The model usually referred and subscribed [illegible] infectious disease model of the pathogenes[illegible] ease. It states, in its outmoded form, that a [illegible] type of agent is *the* cause of a disease entity, [illegible] the pneumococcus causes pneumococcal pr[illegible] Recovery of the pneumococcus from a patie[illegible] injection into a laboratory animal reproduce[illegible] ease. We owe this model to Koch. It perva[illegible] medicine and seeks a full explanation of a di[illegible] the basis of a single cause, whether the caus[illegible] rial, genetic, viral, enzymatic, immunologic, [illegible] tritional, or psychological. In its most gene[illegible] states that most diseases can be explained o[illegible] of single causes: "Behind every twisted thr[illegible] twisted molecule."

We owe the second model—the cellular pa[illegible] model—to Virchow. In essence, it states th[illegible] is due to a material defect of the cells or orga[illegible] body. Usually this defect is thought to be [illegible] caused by some external agent or by some [illegible] erration of the intrinsic machinery of the ce[illegible] ample, the absence or structural alteration [illegible] zyme. In formal terms a functional model, it [illegible] that fascinates most physicians.

Most medical explanations of disease are [illegible] in nature. They tell us about the relationsh[illegible] cardiac output to pulmonary edema, but the[illegible] tell us why at a particular point the heart fai[illegible] er words, most explanations of the symp[illegible] signs of a disease (and the functional disturba[illegible] express) do not explain its antecedents. Fu[illegible] the disadvantage of an antecedent, or histo[illegible] planation is that it is descriptive and not pred[illegible] example, a variety of animal or plant spec[illegible] explained in part by a genetic mutation, but t[illegible] does not predict when a new mutation wil[illegible] model of disease should allow us to predict [illegible] risk for a particular disease as well as wh[illegible] what circumstances the predisposed perso[illegible] velop it. Minimally, a complete model of dis[illegible] to be functional, historical, and predictive.

The third traditional model is the diagno[illegible] of disease. Kety (5) has defined it as "a pro[illegible] moves from the recognition and palliation [illegible] toms to the characterization of a specific [illegible] which the etiology and pathogenesis are k[illegible] treatment is rational and specific." How[illegible] ideal situation does not occur in nature. As [illegible] edge increases, we are continually recogn[illegible]

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tiple subforms of every specific disease, although each has an invariant cellular pathology; for example, subforms of Graves' disease, essential hypertension, peptic duodenal ulcer, and gout exist. The distinctive lesions of these diseases, the phenotypes, have different etiological or pathogenetic antecedents. Some patients with gout have normal levels of serum uric acid, and elevations of serum uric acid do not invariably lead to gout. Elevations of serum uric acid are produced by separate and distinctive enzymatic mechanisms that produce increased purine biosynthesis by cells or by the enhanced reabsorption of uric acid by the kidney or by a combination of both. In addition, the successful treatment of a disease does not have to depend on knowledge of its etiology or pathogenesis. Therefore, one must conclude that the diagnostic model of disease is idealistic, not realistic.

The fourth traditional model of disease is the curative one, which derives directly from the infectious disease and the cellular pathological models: once we know the singular pathogenetic cause of a disease or its disturbed pathophysiology, we are in a position to develop and launch the magic, curative bullet. Successes in the treatment of infectious diseases and vitamin deficiencies have reinforced belief in this model, which carries with it the hope that eventually we will cure all diseases. But nature is subtle and ever-changing. Bacteria are capable of adapting to the magic bullet. Changing social mores may cause the gonococcus to acquire plasmid DNA from *Hemophilus* residing in the human mouth, thereby producing penicillinase and becoming resistant to penicillin. Once we have a cure, it may not be permanent. Furthermore, most diseases cannot be cured; at most, all the physician can do is relieve suffering, alleviate symptoms, and avoid harming the patient (50% of all illness today is iatrogenic). Indeed, the physician's relationship with the patient can be a powerful agent in the relief of suffering and of symptoms. In addition, the medical profession has traditionally had a deep resistance to prevention of disease.

For the purposes of this discussion, I shall confine myself to these four models, although there are others (e.g., McKeown's discussion [6] of the consequences of the educational model in medicine).

TRADITIONAL MEDICAL MODELS AND PSYCHIATRY

Each of the four models of disease has had its proponents and opponents in psychiatry. In the nineteenth century, the major psychiatric diseases were considered by many to be diseases of the brain. Early in this century, this belief was strongly reinforced by that most paradigmatic and interesting of all psychiatric disease—paretic neurosyphilis. Here confirmation was found of the infectious disease model when Schaudinn's pale spirochete was discovered in the brain. Specific therapies could be directed at it in the form of fever, the arsenicals, or penicillin. Lost in this oversimplification was the fact that the spirochete in its tertiary phase causes paretic neurosyphilis in some but tabetic neurosyphilis, aortitis, gummata of bone and skin, or no disease at all in others. Furthermore, the variable symptoms of paretic neurosyphilis taught many psychiatrists all of psychopathology. Nissl's criterion for the diagnosis of paretic neurosyphilis was the presence not only of the spirochete but also of lymphocytes in the perivascular spaces of the brain.

We still have no explanation of the various phenotypic expressions of tertiary syphilis. The variable symptomatology of paretic neurosyphilis is usually explained by the interaction of the disease with the host's "personality." Nissl's observation is generally explained by the fact that the spirochete occasions a response by immunocompetent lymphocytes.

This model of a classical psychiatric disease has impelled psychiatry to search continuously for the bacterial, molecular, enzymatic, viral, and metabolic cause of mental illness. In another form and at another level of organization, this model has explained most psychiatric disease and its symptoms on the basis of specific psychological conflicts. Hysterical symptoms, for example, are said to have at their roots unconscious sexual conflicts or memories. This psychoanalytic formulation does not differ conceptually from the concept of the infectious disease model; it is merely phrased in a different language. Freud's initial concept of the hysterical symptom was modeled after the identification of a pathopsychological factor, just as much of medicine seeks the explanation of disease in pathophysiological terms *after* disease onset. Later it was recognized that every symptom is multiply determined, and an important dimension—the developmental one—was added to our understanding of symptoms.

Objections to the Traditional Models

Freud (7) also added to our knowledge of the taxonomy of specific psychiatric disease, and of persons, by studying character structure. Gradually the line between the cluster of symptoms and the character structure of the patient has become obscured; a hysterical patient is said to be at risk for hysterical symptoms, and his or her suggestibility can be used to advantage to remove them. Persons in fact have symptoms and diseases, or, in the extreme extension of this view, no disease at all. Disease is seen by some as a way of "labeling" people pejoratively (8). Besides, the argument goes, people are infinitely variable; psychological polymorphism is limitless, so one cannot label people accurately. Diagnosis is fruitless, classification difficult. A variant of this viewpoint holds that the taxonomic label is irrelevant—did Freud not state that we are all at root motivated by the same biological drives? Individuals are all fundamentally the same, according to this view; their problems are in essence just varia-

tions on the same theme, rather than diseases, so that treatment should focus not on diagnosis but on the drive that produced the conflict.

In addition, the concept that diseases cannot be separated from the personality of their hosts is enshrined in literature and medicine. It reaches its height in the work of Dunbar (9), who extended this concept from psychiatric to many "medical" diseases. More recently, Friedman and Rosenman (10) used this framework to describe the personality type at risk for coronary artery disease. Such an individual, they contend, has a high time-urgency, is impatient and irritable, and has an inner sense of being under pressure. This person may also be characterized by certain physiological features (11) such as a higher blood platelet count in whole blood, a lessened decrease in intensity of platelet aggregation to norepinephrine during exercise, an altered lipoprotein pattern (consisting of an elevated β- to α-lipoprotein ratio), and elevated mean serum cholesterol and triglyceride levels. Underlying this attempt at correlation between personality characteristics and disease is the assumption that certain persons are predisposed to a disease by virtue of both their personality and their physiology. Dunbar's approach to the role of psychological factors in disease did not address itself either to questions about the initiation of a disease or to questions about the mechanisms set into operation at the onset of the disease and those that maintain the disease. Thus, the questions of when and why a hard-driving (and inwardly driven) executive develops an attack of angina pectoris or myocardial infarction remain unanswered by this approach.

In this intellectual debate, medicine and psychiatry have confronted each other. The former states that only cells have diseases, the latter that only persons have diseases. The truth is that persons may have diseased cells or altered physiological functions.

DISEASE AS A BIOLOGICAL PHENOMENON

Medicine and psychiatry have both overlooked the fact that disease is a biological phenomenon. Biology consists of two great complementary bodies of knowledge: molecular and cellular biology and evolutionary biology. The first concerns itself with the origins, components, and structures of living things; the second with the manner in which organisms and populations of organisms behave, interact, and communicate and how they live in and adapt to their natural and social environments. In the process of successful adaptation some organisms survive to reproduce (12, 13). By virtue of their variable genetic composition and adaptive capacities some are selected to survive and reproduce in particular environments. Selective advantage in some environments is afforded even to those whose genes produce a specific molecular alteration; for example, hemoglobin-S in its heterozygous form produces the sickle-cell trait. Individuals with this trait are more likely to resist infection with falci- laria. Therefore, in certain environments endows the phenotype with selective adva genetic disposition to form immunoglobu antibodies confers selective advantage by resistance to parasitic invasion that may ca In other environments that contain othe those endowed with the capacity to form bodies are at risk for bronchial asthma and ot gic diseases (14, 15). These examples sug polymorphism must be viewed from two perspectives—cellular-molecular and evolut

These two perspectives are paradoxical evolutionary vantage point, selective advan en to some by the structural abnormality o The physician tends to focus on the struct tion in hemoglobin-S; the biologist welco cause it endows its owner with a selective a The physician is biased toward viewing pa molecular terms, whereas the evolutionary regards the same phenomenon in terms of nism's fitness to survive and reproduce.

The biologist is also concerned with the cu just the natural, environment and with cult tion, during which information is transferred generation to the next by language and tra by genetic means. By cultural evolution I evolution of an organism or population that about by culture acting as the agent. Durin cess, the organism or population may adapt changes.

The Infectious Disease Model Revisited

Many psychiatrists have rejected the fou models of disease because they have fou ear, restrictive, and oversimplified. The tra fectious disease, cellular pathological, and models have so far been unsuccessful in schizophrenia. Nonetheless, a call has gone chiatrists to return to these traditional medi (1) or to develop a model that incorporates cal and social data in the traditional medica using a systems theoretical approach (2).

A third approach consists of the develop truly biological model of disease, one that in the deep insights of modern genetics, virolo nology, biochemistry, cellular biology, and physiology. However, these insights are n if they do not also incorporate and integra cepts of developmental and evolutionary b

Such integration of these two bodies of makes the classical, linear infectious disease adequate, but we can replace it with a se model that contains genetic, developmenta personal, and social elements and is well by the biology of the hepatitis B virus (16) virus diseases (17).

Hepatitis B Virus

There are millions of carriers of hepatit

The prevalence of the carrier state is high in the Philippines and in parts of Europe and low in the United States. The virus can be transmitted horizontally from one person to another in a variety of ways—by blood transfusion, sputum, fecal contamination, a break in the skin, and probably by a variety of insect vectors, such as mosquitoes and bed bugs. The virus may also be transmitted vertically; women with this form of viral hepatitis just before or during delivery or women who are carriers of the virus may pass it on to their children, who in turn become carriers. However, this mode of transmission occurs only in some populations.

Most people who have hepatitis B virus remain carriers and do not develop any of the variety of diseases associated with it. However, the carrier state sometimes produces subclinical disease and mild biochemical abnormalities of liver function tests. At other times, the carrier state is not characterized by any detectable abnormality, but it is associated with the ability to form an antibody against one component, the core of the virus, and with the persistence of that antibody and of another viral antigen in the bloodstream of the carrier. The ability of carriers to transmit the virus is probably genetically controlled; some carriers are able to form antibodies against a third antigenic component of the virus and so do not transmit the virus. On the other hand, the virus may not even contain this third component, in which case the risk of transmission is reduced.

The hepatitis B virus may cause or be associated with a variety of diseases: 1) acute hepatitis, from which the patient may recover or which may go on to 2) chronic hepatitis; 3) chronic hepatitis not preceded by acute hepatitis; 4) any of a variety of diseases associated with immunological abnormalities or deficiencies that may enhance the presence of the virus in the blood; 5) the depositing in small blood vessels of complexes formed by the antigens of the virus and the antibodies of the host; and 6) primary hepatic carcinoma. Several factors determine which disease a person develops. Blumberg (16) suggested that the age at which the person is infected is one such factor—infection in utero or in infancy may be conducive to the later development of hepatic carcinoma in some but not all persons. The form the clinical disease takes is determined in part by the antigenic properties of the virus, by the capacity of the host's immune system to "recognize," or not, the foreign nature of the antigen, by the host's ability to develop antibodies against two, if not three, antigens of the hepatitis B virus, and by the timing and persistence of the antibody response.

Characteristics of Slow Virus Diseases

In some slow virus diseases—e.g., kuru and scrapie—the host does not respond at all immunologically to the unconventional virus and no inflammatory response is observed. Other slow virus diseases, such as subacute sclerosing panencephalitis, are caused by conventional viruses. In this disease the inciting agent, the measles virus, is defective; in other conventional slow virus diseases the host is immunologically defective.

Diseases produced by viruses also exemplify the oversimplification of the traditional cellular pathological model. The disease lymphocytic choriomeningitis, although incited by a virus, is characterized by the invasion of the meninges by a profusion of lymphocytes, as its name implies. However, no virus can be recovered from the brain or its coverings. The immunological response to the virus, present elsewhere in the body, produces the main lesion. If the virus is first injected into a pregnant animal, offspring later injected with the same virus do not show any invasion of the meninges by lymphocytes.

The developmental principle—that the kind of lesion produced by a virus depends on the age at which the animal is injected with it—is also illustrated by studies of immunological tolerance. If embryos of a particular strain are inoculated with various antigens, such as blood or other cells, from another strain, the adult will be tolerant to these foreign cells. At the time of later challenge, the antigenic substances are accepted as if they belonged to the recipient, a phenomenon that would not occur if the same antigens were injected into the unprepared adult animal (18).

The biology of lymphocytic choriomeningitis and of immune tolerance exemplifies the importance of the age at which the challenge occurs. When newborn mice are infected with the scrapie virus, no disease develops immediately. The virus does not replicate in these mice for 1 year, but when they are 18 months old, high titers of the virus are present in their brains and spleens. When mice are injected at 4 days of age or older, virus replication occurs immediately and kills 70%–100% by the time they are 10 months old (19).

Implications for the Disease Model

These viruses exemplify important biological principles in disease: the role of the host's genetic makeup in determining its adaptive (immunological) response, the host's age at the time of challenge by the infectious agent, and various interactions, from none to excessive, between the virus and the host's immune response to it. The biology of viruses and their interactions with the host have additional, even more interesting properties. Blumberg (16) stated that in "the epidemiology of infectious agents not only the host and virus are factors but also the previous host or hosts of the agent." The previous host may be human or, possibly, an insect.

Virus infections may also have surprising social determinants and consequences. Parental infection with hepatitis B virus may determine the sex ratio of offspring. If either parent is a carrier of hepatitis B virus and its surface antigen, more boys are born (20), but if a carrier parent has developed an antibody against the virus, fewer boys are born. Therefore, the virus may

partially determine the composition of a population and have effects on its economy and social structure and on the availability of marital partners.

Another major insight that modern virology has provided is that the customs of a social group may determine viral infection with a deadly disease. This in turn may determine the male-to-female ratio in a population (17). Kuru is rare in men but common in boys, girls, and women. Because it is a fatal disease, men predominate in villages where kuru is indigenous. Women and children used to mourn the deaths of their kin by engaging in the preparation and later ritual cannibalism of the infected brain of the departed and thus later developed kuru. With the abolition of cannibalism, kuru is declining in New Guinea.

The modern biology of virus infections therefore provides remarkable insights into the nature of disease. The model outlined is complex, nonlinear, and broad compared to the traditional infectious disease model. This new model is realistic rather than idealistic and has implications for our conceptualization of many other diseases. In summary, it states that

1. A pool of persons predisposed to a disease occurs in every population—the carriers.
2. The time of life a person is infected and the route of infection determine in part the course and nature of the disease or carrier state.
3. The same inciting agent can give rise to multiple disease forms, which are determined by the characteristics of the agent in interaction with the host.
4. The adaptation (immune response) to the agent can vary quantitatively or qualitatively.
5. The different adaptive responses determine in part the ultimate form in which the disease is expressed.
6. Social and cultural factors play a role in disease and are in turn influenced by it.
7. The disease can be transmitted horizontally and vertically in several ways.

We can make some generalizations on the basis of these conclusions. For example, the factors that predispose to a disease are not invariantly those which incite or sustain it (21). In addition, in every disease genetic polymorphism plays a complex role. The age and sex of individuals determines their response to disease. The phenotypic expression of disease is a complex product of the interaction of the agent and the host in which qualitative and quantitative variables in the adaptive-defensive responses of the host play significant roles.

THE NEW INFECTIOUS DISEASE MODEL AND THE BIOLOGY OF DISEASE

Our present understanding of the role that social, cultural, and behavioral factors play in disease is startlingly analogous to the generalizations outlined in the previous section. If biology is also viewed in evolutionary and cultural, rather than in only molecular and cellular, terms, then social, cultural, and b[illegible] factors can be incorporated into a biology c[illegible] Stress is no longer regarded in the linear [illegible] infectious disease once was. Stress, as a fac[illegible] inception of disease, produces response[illegible] stressed person that are signaled, if at all, by [illegible] The meaning of the stress may be portentous [illegible] if portentous it generates adaptive-coping de[illegible] person may flee, meet the challenge of the [illegible] overcome it, or be defeated by it. Success [illegible] with the stressful challenge is assured in pa[illegible] sonal factors such as intelligence, proble[illegible] ability, past mastery of the stress, and the [illegible] information available (22).

In some persons failure to overcome the [illegible] lead to helplessness, hopelessness, and giv[illegible] i.e., a state of adaptive failure. In others the [illegible] to stress, change, or challenge may be inapp[illegible] excessive. Some persons cannot cope with [illegible] events without another person's assistance. [illegible] immature, or adaptively inept, the person is [illegible] capable he is of coping alone or appropriatel[illegible] stronger the influence of the social field on [illegible] Other persons cannot cope because they are [illegible] of apperceiving or recognizing distress signa[illegible] ing that they should or must cope (25). [illegible] stress alone does not linearly produce disease [illegible] does so in interaction with the meaning it ha[illegible] stressed person, his manner of coping with [illegible] adaptive capacities. This principle is analog[illegible] homologous, to the interaction of a virus [illegible] adaptive-immune response.

In addition, today we know that the same [illegible] gous stresses or events can place people at [illegible] variety of different diseases. The stress of be[illegible] traffic controller is associated with an inc[illegible] cidence of elevated blood pressure, peptic [illegible] ulcer, and, sometimes, diabetes mellitus [illegible] reavement that is not coped with and pro[illegible] "giving-up, given-up" complex has been es[illegible] precede many different diseases (23). Ber[illegible] plays an important role in many, if not al[illegible] with "psychosomatic" diseases. Real or t[illegible] loss has often been cited as a factor contribu[illegible] initiation of other diseases as well (27): cance[illegible] tuberculosis (31), diabetes mellitus (32), l[illegible] and leukemias (33), juvenile diabetes mellitus [illegible] heart failure (35). Depressive moods, the [illegible] giving-up, and the loss of all hope also adver[illegible] the outcome of surgical operations (36, 37).

Therefore, it is not the bereavement per [illegible] termines the nature of the disease, any mor[illegible] hepatitis B virus is associated with any on[illegible] Bereavement may elicit a disease only in [illegible] disposed to it. In other persons a parting do[illegible] duce any disease but occasions appropriate [illegible] sorrow.

Separation early in life may also place org[illegible] risk for disease in later life. Separation has be[illegible] to produce alterations in the physiology and [illegible]

istry of the brain and in behavior. Every organ system so far studied is affected by the separation of young animals from their mothers; heart and respiratory rates fall (38–40), and levels of brain catecholamines, nucleoproteins, and enzymes such as ornithine decarboxylase diminish (41, 42). The prematurely weaned rat is at inordinate risk for later gastric ulceration when restrained (43), and the intrinsic physiological development of the stomach is altered by premature separation (S.H. Ackerman, personal communication). Premature separation also engenders a latent regulatory disturbance in the animal that is only elicited by later restraint-stress. This regulatory disturbance consists of the animal's inability to maintain its body temperature, which is highly correlated with the production of gastric ulcers (44).

The developmental principle that is exemplified by viral diseases and immune tolerance is also illustrated by these studies on stress. It may be extended even further, for it is entirely possible that some diseases are related to the persistence of, or reversion to, age-inappropriate (immature) regulatory patterns. In anorexia nervosa, age-inappropriate patterns of the luteinizing hormone have been found (45), and Vaillant (46) documented that age-inappropriate psychological coping patterns are significantly correlated with morbidity and mortality.

The principle that a virus disease can be transmitted vertically—that an acquired characteristic is "inherited"—is also illustrated by the observation that the offspring of prematurely weaned animals raised to maturity are at increased risk for gastric ulceration when later restrained, despite the fact that they were not prematurely weaned. The risk is much greater than in the offspring of a normally weaned mother (47). We do not know the nature of this effect; however, genetic transmission is highly unlikely.

SUMMARY

I have tried to illustrate that a broad biological model—not an outdated medical or narrow cellular or molecular model—incorporates observations and experimental data from levels of organization as diverse as the cultural, evolutionary, and molecular. In this framework disease can be viewed as a failure of adaptation that may occur in one or many systems; it is a biological phenomenon. Because it is a biological phenomenon, it deals with organisms in interaction with their natural, social, and cultural environments. Disease is not only a matter of a disturbance of the self-regulatory mechanisms within cells or of any one simple factor such as infection, nutritional deprivation, or the psychology of the diseased person. A modern, biological model of disease contains elements with which psychiatrists have often concerned themselves and is broad enough to accommodate diverse points of view from the molecular to the evolutionary level.

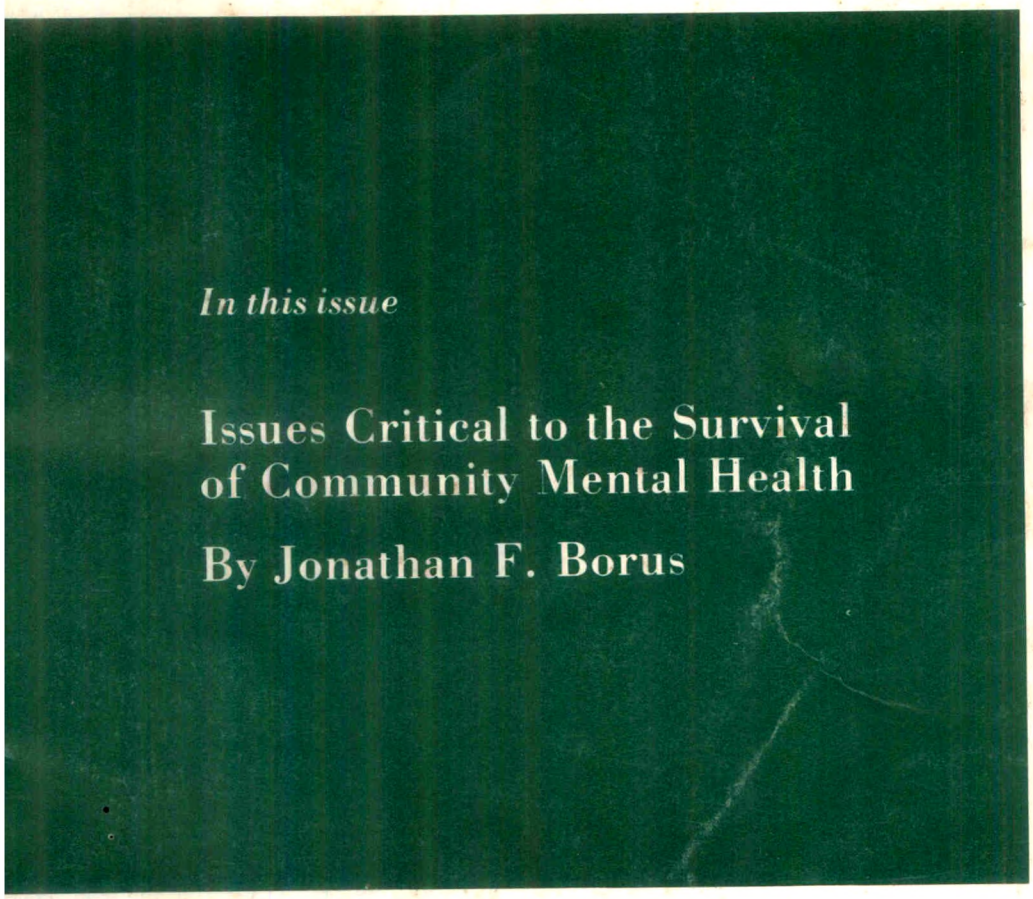
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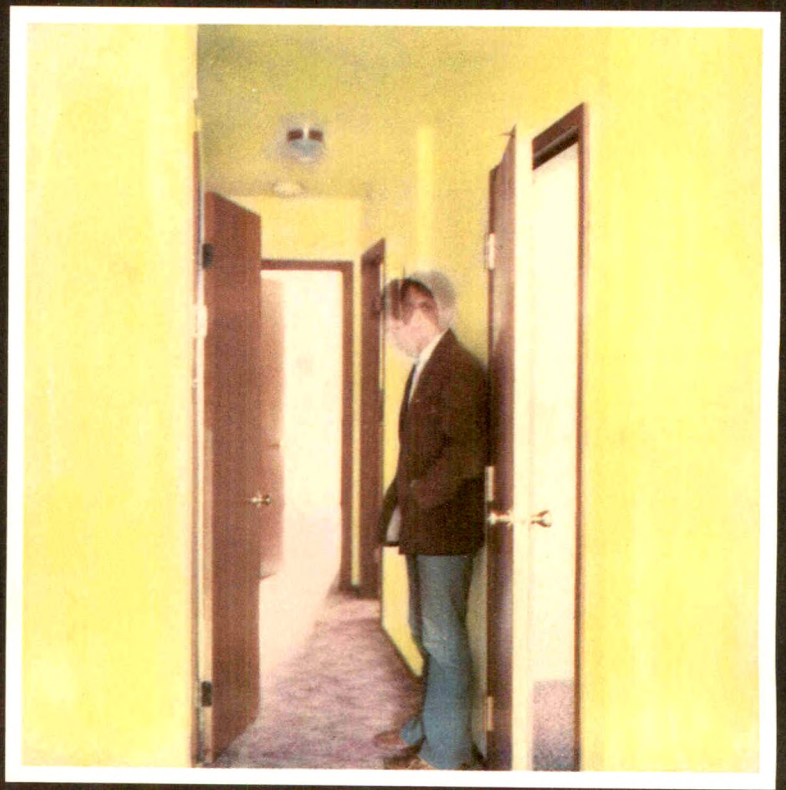
Issues Critical to the Survival of Community Mental Health

By Jonathan F. Borus

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LOST IN A MAZE OF BEWILDERING THOUGHTS



'STELAZINE' HELPS FIND THE WAY BACK

The schizophrenic patient is often lost in a maze of bewildering thoughts, confusing voices and frightening anxiety. For such patients, 'Stelazine' provides effective control of these and other psychotic symptoms.

And 'Stelazine' can help patients participate in your therapeutic programs. Also important, convenient b.i.d. 'Stelazine' usually does not cause excessive sedation.

After 18 years of extensive research and clinical experience, no other antipsychotic agent has demonstrated significantly greater overall effectiveness and significantly fewer side effects than 'Stelazine'. These facts make 'Stelazine' a first choice for therapy.

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Generally avoid using in patients hypersensitive (e.g., blood dyscrasias, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

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Other requirements. The number of words, tables, and figures should appear in the upper right corner and a phone number for the corresponding author in the lower right.

Précis

The précis should be up to 100 words for regular articles and Brief Communications (no précis is used in the Clinical and Research Reports section). The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

Text

Headings and subheadings should be inserted at reasonable intervals in all types of papers. Footnotes to text material should be typed on a separate page at the end of the manuscript. Summaries are rarely desirable, although a final "Comment" or "Conclusions" section may be used. The body of the paper should be written in the active voice and first person where appropriate.

Statistics. Authors should be sure that statistics used are both necessary and appropriate. Statistical tests should be described and a reference citation given if the tests are not generally known. It is not sufficient to say "differences between the groups were significant." In cases of lengthy complicated analyses, the authors may summarize results and indicate that details are available from them on request.

Abbreviations. All abbreviations (other than those for metric units) should be explained the first time they are used. Idiosyncratic abbreviations should be avoided: overuse of abbreviations may hinder rather than facilitate readability.

Drugs. Generic rather than trade names of drugs should be used; trade names may be given parenthetically if necessary.

Units of measurement. All measurements should be in metric units; standard abbreviations are used.

References

References should be restricted to closely pertinent material; a complete review of the literature is rarely desirable. *Accuracy of citation is the author's responsibility.* References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text; no bibliographies can be used.

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26

2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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Navane provides effective, rapid control of the major symptoms of psychosis including hallucinations, delusions, agitation, hostility, and thought disorders.[1-5] Navane promotes rapid progress, usually without the over-sedation or cardiovascular problems that can impede the therapeutic course.

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Long-term improvement

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Capsules 1 mg., 2 mg., 5 mg., 10 mg., 20 mg. Concentrate 5 mg./ml. Intramuscular 2 mg./ml.

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BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg
(thiothixene hydrochloride) Concentrate: 5 mg/ml,
Intramuscular: 2 mg/ml

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, 1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. Denber HCB, Turns D: Double blind comparison of thiothixene and trifluoperazine in acute schizophrenia. *Psychosomatics* 13:100-104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: The place of thiothixene in the treatment of schizophrenic patients. *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, 1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 130th Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlrabe J, Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, et al: Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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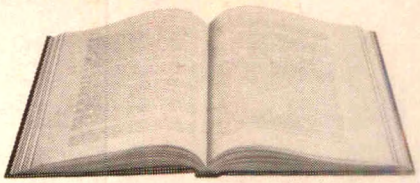
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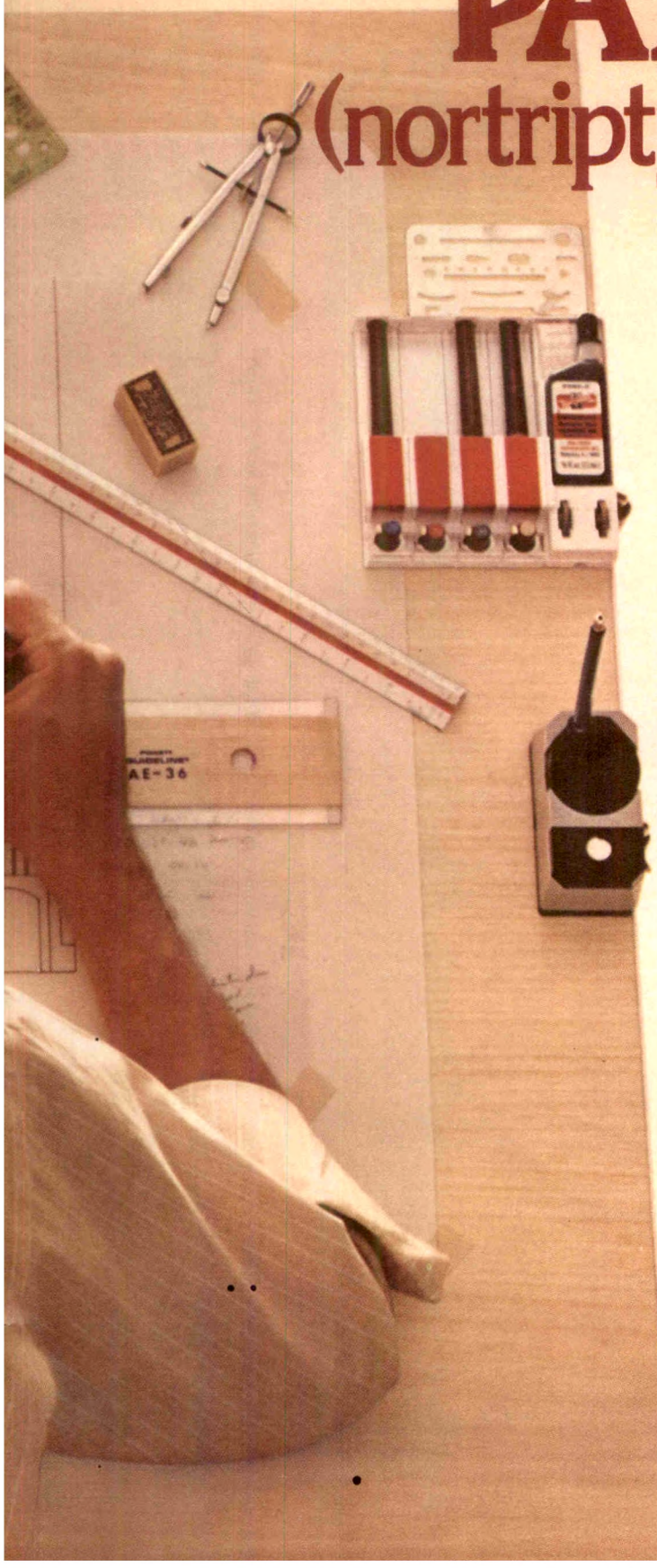
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Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

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Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose* – 25 mg. three or four times daily; dosage should begin at a low level and increase as required. As an alternate regimen, the total daily dosage may be given once-a-day. *Elderly and Adolescent* - 30 to 50 mg. per day, in divided doses, or the total dosage may be given once-a-day. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

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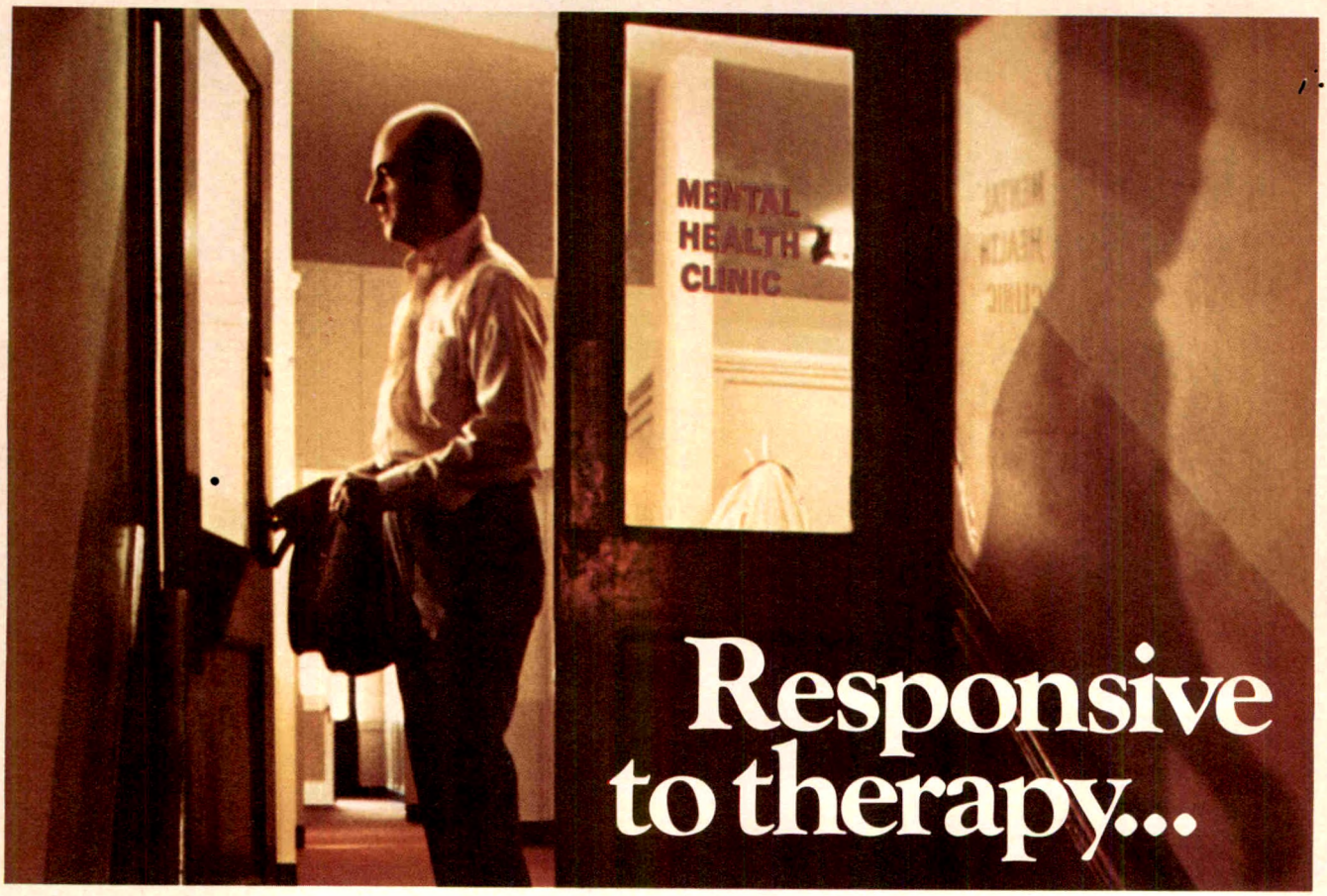
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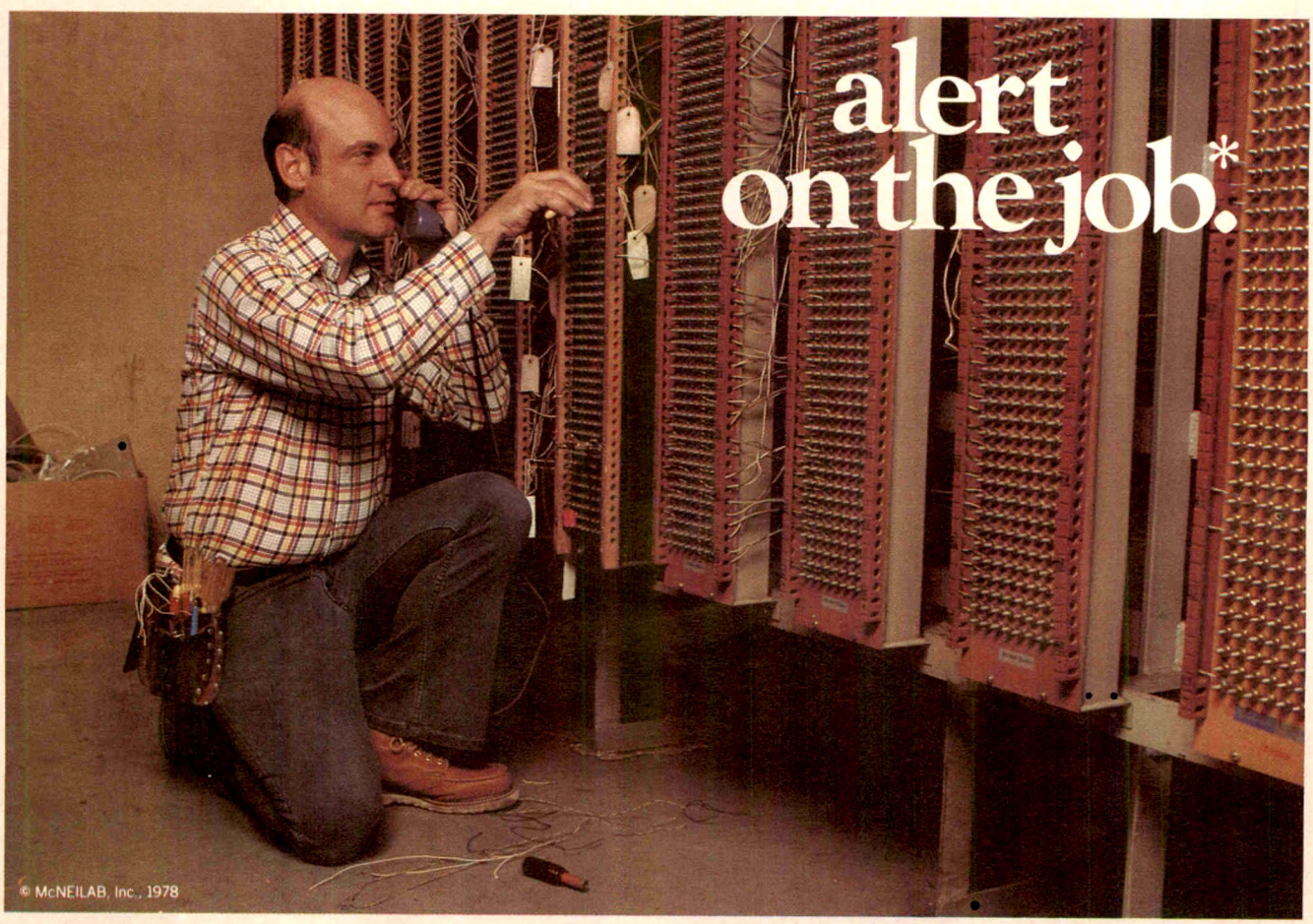
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Highly effective in a wide range of acute and chronic psychotic disorders[1-7]

Permits aggressive titration to effective dosage levels for optimal control[1,4]

Usually leaves patients alert and responsive...
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Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects[3-5,7-9]
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Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Man, P.L.: Dis. Nerv. Syst. 34:113 (Feb.) 1973. 2. Sugerman, A.A., et al.: Am. J. Psychiatry 129:1190 (June) 1964. 3. Ayd, F.J., Jr.: Med. Sci. 18:55 (Oct.) 1967. 4. Howard, J.S.: Dis. Nerv. Syst. 35:458 (Oct.) 1974. 5. Abuzzahab, F.S., Sr.: Psychosomatics 11:188 (May-June) 1970. 6. Darling, H.F.: Dis. Nerv. Syst. 34:364 (Oct.-Nov.) 1973. 7. Gerle, B.: Clin. Trials J. 3:380 (Feb.) 1966. 8. Snyder, S.H., et al: Science 184:1243 (June 21) 1974. 9. Stimmel, C.L.: Dis. Nerv. Syst. 34:219 (Apr.) 1976.

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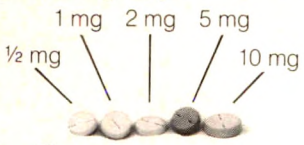
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Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

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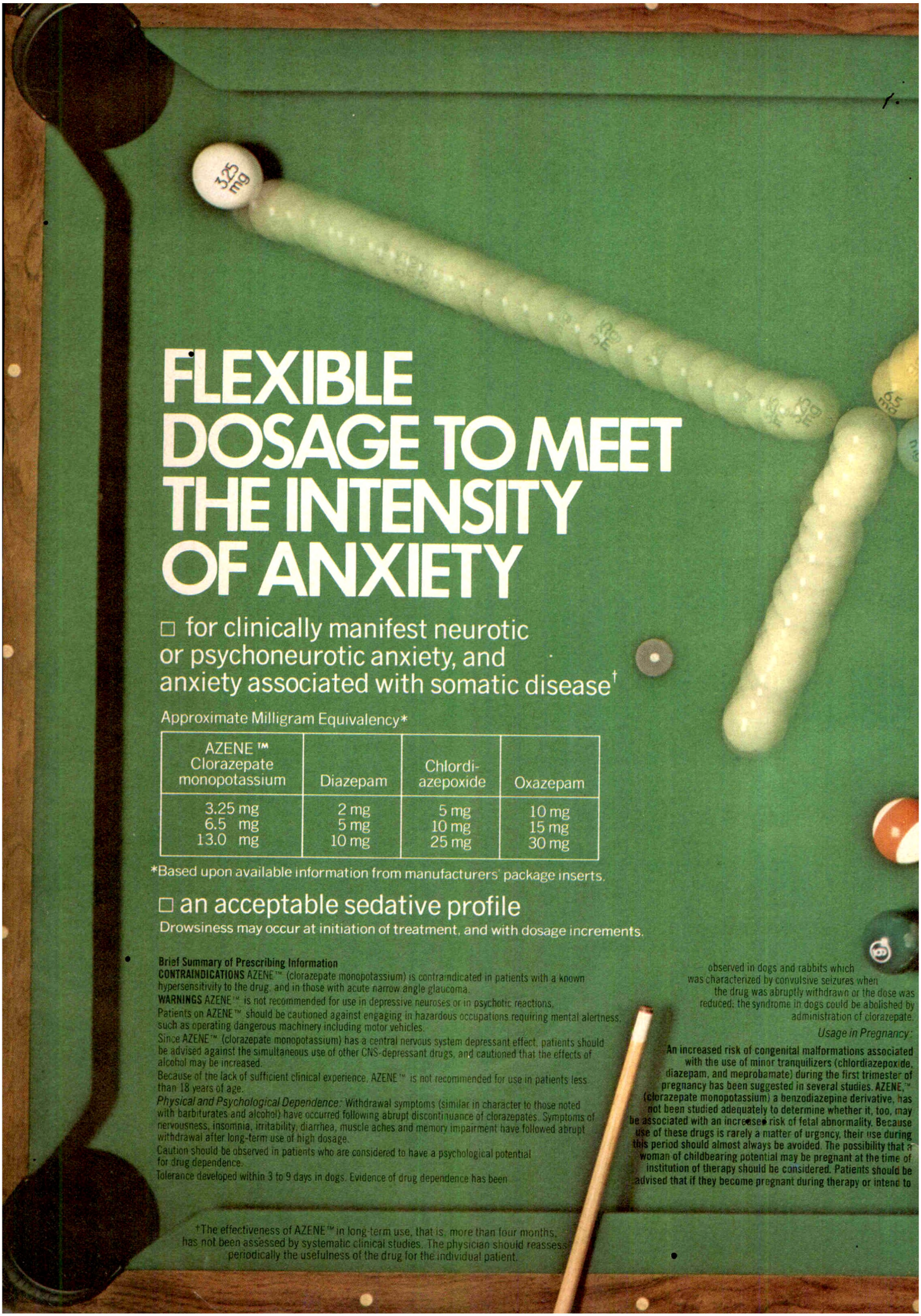
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13.0 mg	10 mg	25 mg	30 mg

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☐ an acceptable sedative profile

Drowsiness may occur at initiation of treatment, and with dosage increments.

Brief Summary of Prescribing Information

CONTRAINDICATIONS AZENE™ (clorazepate monopotassium) is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS AZENE™ is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on AZENE™ should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since AZENE™ (clorazepate monopotassium) has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, AZENE™ is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence: Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepates. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

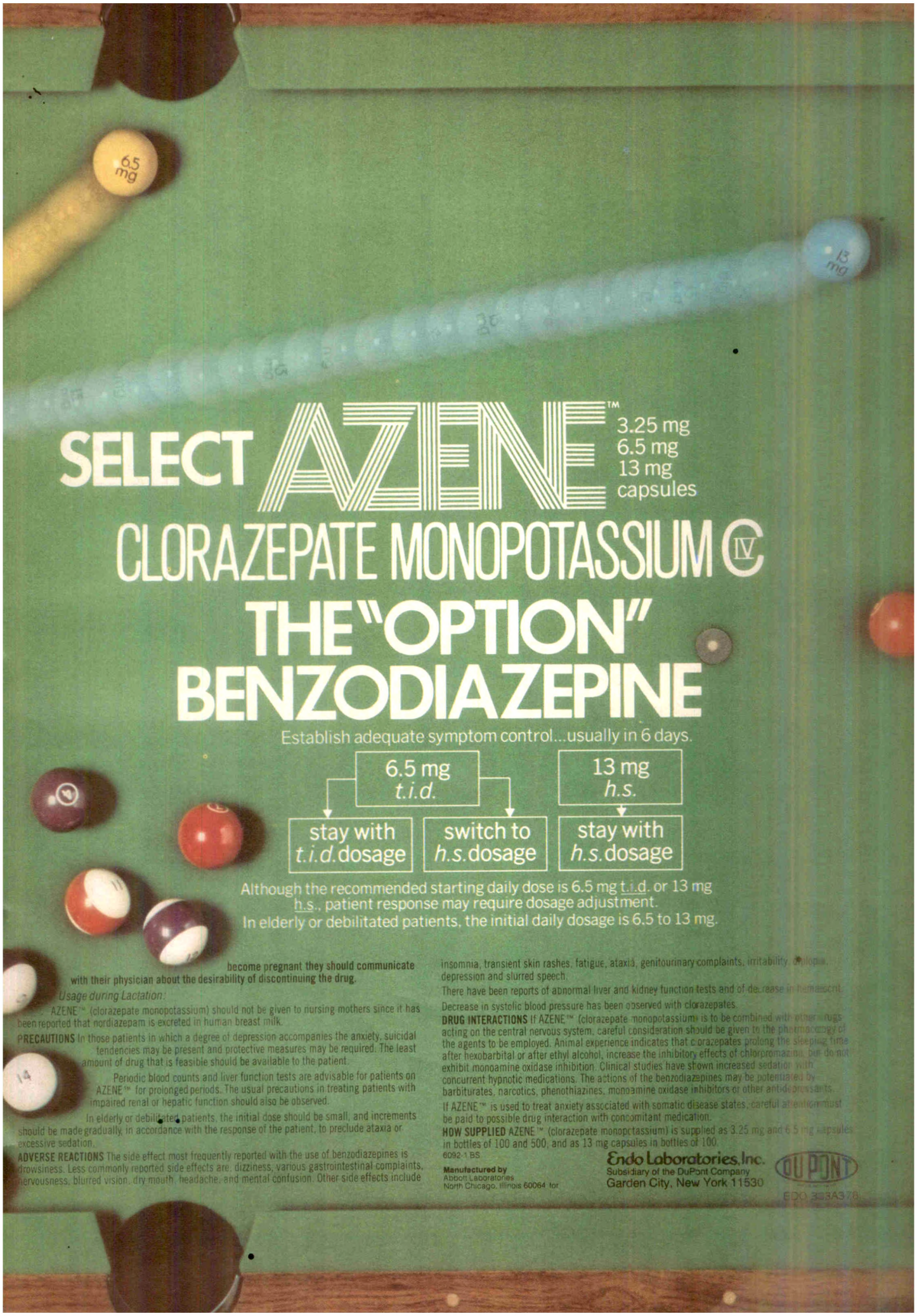
Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Tolerance developed within 3 to 9 days in dogs. Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. AZENE,™ (clorazepate monopotassium) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to

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6.5 mg
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capsules

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THE "OPTION" BENZODIAZEPINE

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- switch to *h.s.* dosage

13 mg *h.s.*

- stay with *h.s.* dosage

Although the recommended starting daily dose is 6.5 mg t.i.d. or 13 mg h.s., patient response may require dosage adjustment.
In elderly or debilitated patients, the initial daily dosage is 6.5 to 13 mg.

become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

AZENE™ (clorazepate monopotassium) should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Periodic blood counts and liver function tests are advisable for patients on AZENE™ for prolonged periods. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS The side effect most frequently reported with the use of benzodiazepines is drowsiness. Less commonly reported side effects are: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects include insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed with clorazepates.

DRUG INTERACTIONS If AZENE™ (clorazepate monopotassium) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that clorazepates prolong the sleeping time after hexobarbital or after ethyl alcohol, increase the inhibitory effects of chlorpromazine, but do not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If AZENE™ is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

HOW SUPPLIED AZENE™ (clorazepate monopotassium) is supplied as 3.25 mg and 6.5 mg capsules in bottles of 100 and 500, and as 13 mg capsules in bottles of 100.

6092-1 BS

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(pemoline)

Prescribing Information

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannibalization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (white) in bottles of 100 (**NDC** 0074-6025-13)
37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)
75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored)
in bottles of 100 (**NDC** 0074-6088-13)

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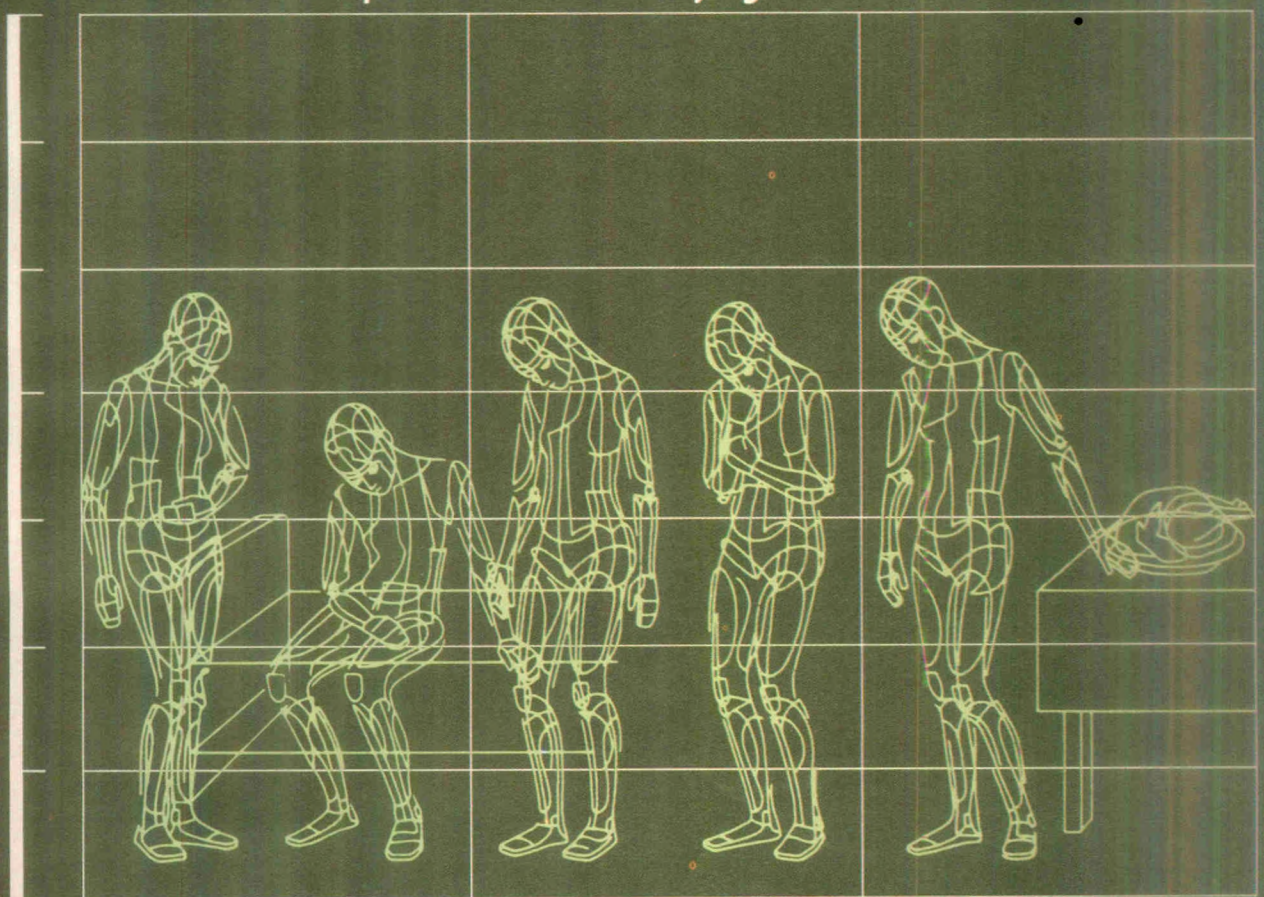
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AJP

Within one week
A better response
For twice as many patients

compared to amitriptyline alone



Limbitrol®

For rapid relief of symptoms in moderate to severe depression and anxiety

ROCHE

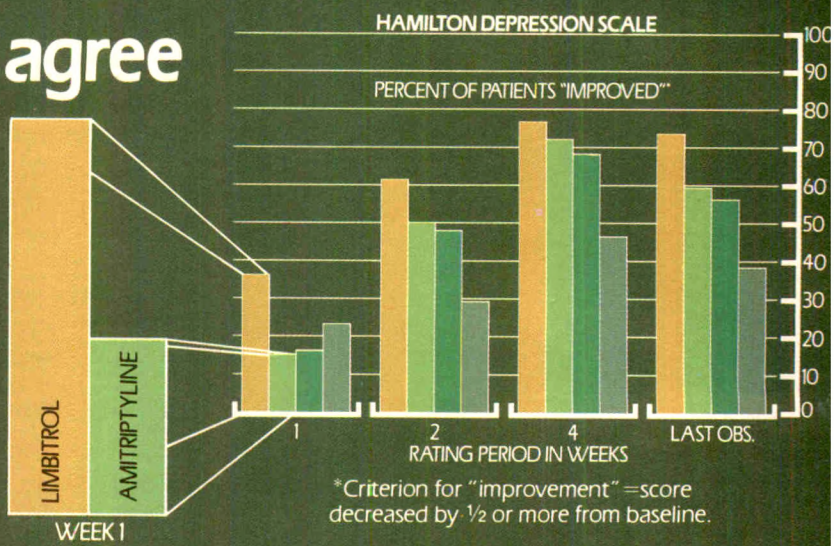
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Within one week
Twice as many patients improved–sooner

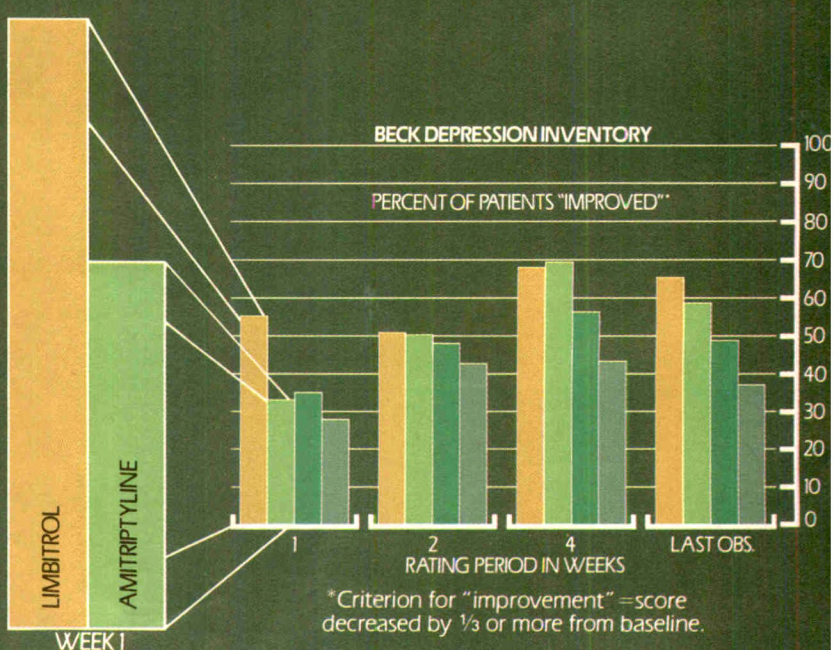
In a rigidly controlled
nationwide multicenter study

The rating scales agree

- ☐ Patients respond faster
  - ☐ Limbitrol—in the first week approximately 2/3 of those who improved had already done so
  - ☐ Amitriptyline—only about 1/3 of those who eventually responded had done so by week one
- ☐ High correlation among all major indices employed including Physician's and Patient's Global Evaluations



*Criterion for "improvement" =score decreased by ½ or more from baseline.



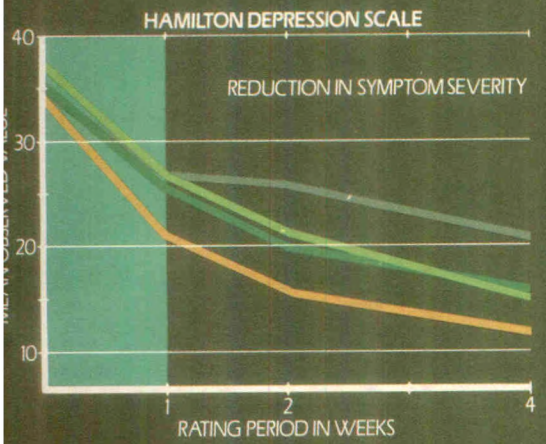
*Criterion for "improvement" =score decreased by ⅓ or more from baseline.

LIMBITROL
AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO

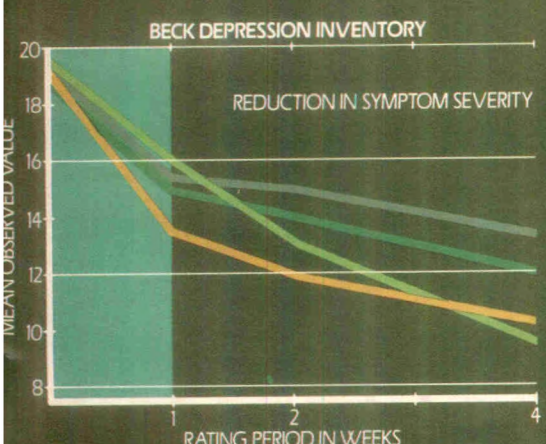
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and better than with amitriptyline alone



Reduction in severity of symptoms based on mean observed value at each rating period.



Reduction in severity of symptoms based on mean observed value at each rating period.

The symptoms subside

Hamilton Depression Scale Items

- ☐ insomnia
- ☐ psychic anxiety
- ☐ somatic anxiety
- ☐ agitation
- ☐ suicidal ideation
- ☐ worthlessness

Beck Depression Inventory Items

- ☐ pessimism
- ☐ dissatisfaction
- ☐ guilt
- ☐ social withdrawal
- ☐ anorexia

By week one, Limbitrol patients' symptoms had, on average, improved to a point not reached by amitriptyline patients until week two.

The data presented are based on a rigidly controlled nationwide multicenter study of 279 patients with a diagnosis of primary depression with symptoms of depression and anxiety of moderate to severe degree.

Provides the well established antidepressant effect of amitriptyline with the proven antianxiety effect of Librium® (chlordiazepoxide HCl)

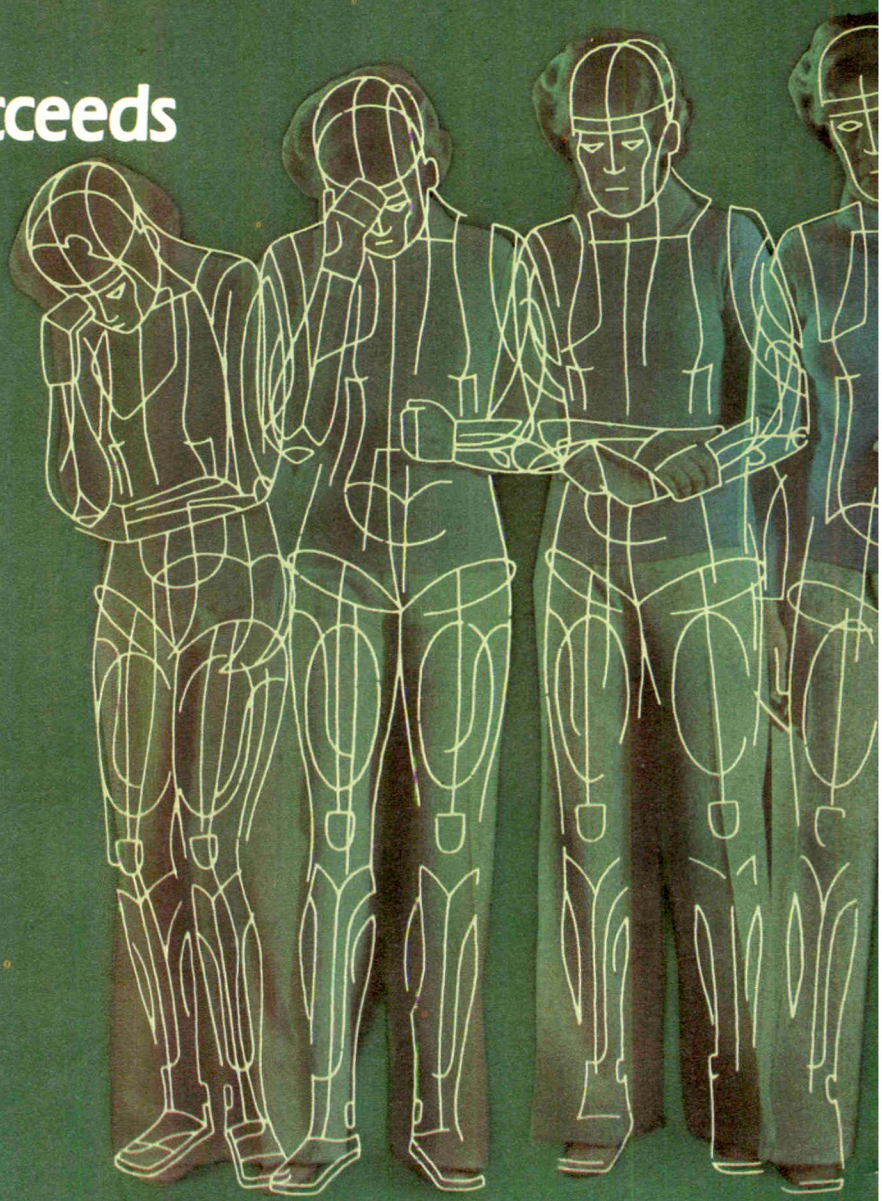
Limbitrol®

For rapid relief of moderate to severe depression and anxiety

A change for the better comes sooner–within one week

In a rigidly controlled nationwide multicenter study

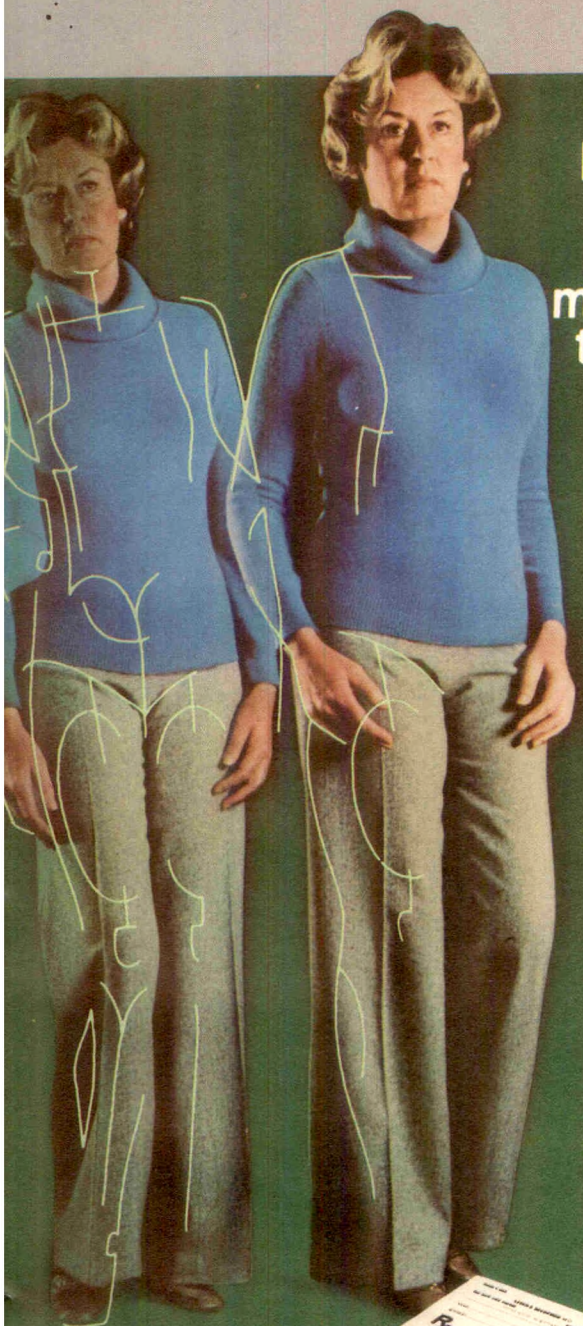
The therapy succeeds



- ☐ Better patient compliance
- ☐ Lower dropout rate
  - ☐ Despite a higher incidence of drowsiness, the dropout rate due to side effects was lower with Limbitrol than with amitriptyline alone (Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)
  - ☐ The dropout rate due to therapeutic failure was lower with Limbitrol (Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)

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Please see last page of this advertisement for summary of product information.



How to initiate and maintain therapy

Select dosage strength appropriate for each patient

☐ Limbitrol 10-25 is recommended for most patients

☐ Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

☐ An initial dosage of three tablets is recommended

☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary

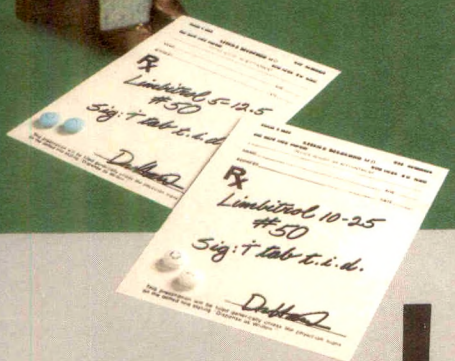
☐ Once a satisfactory response is obtained, evaluate response, and, if necessary, reduce dosage to smallest amount needed to maintain remission

Utilize dosage options to best accommodate individual patient needs

☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness

☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia

☐ Entire dosage *h.s.* to take maximum advantage of the sedative effect



Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

Limbitrol®

For rapid relief of moderate to severe depression and anxiety

Your guide to patient management

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.

Limbitrol®

For rapid relief of moderate depression and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of symptoms of depressive disorders associated with moderate to severe anxiety.

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use. When initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage. Withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (e.g., operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated; sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, overreaction, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with other components alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergics: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male; breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single-dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses for patients who do not tolerate higher doses.

Supplied: Limbitrol 10-25 tablets each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and Limbitrol 5-12.5 tablets each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt) – bottles of 100 and 500, Tel-E-Dose® packages of 100, Prescription Paks of 50.

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UNIQUE-NESS:

In People & at the Lutheran Hospital Psychiatric Care Unit

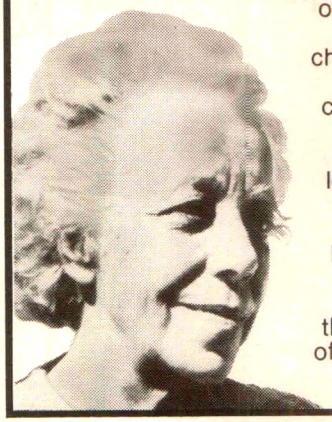
Providing complete psychiatric care in a general hospital setting with special emphasis on disorders of adolescence.

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Karen Horney's theories—particularly in feminine psychology and the role of social and cultural forces in personality formation—have become so widely accepted that her role in formulating them is often overlooked. But at the height of her career, her repeated challenges to Freudian orthodoxy made her a center of controversy on both sides of the Atlantic. Jack Rubins' biography—the first full-length study of Horney's life and work—captures all the drama of that era, showing how her thinking developed and why her boldness and originality place her among the most important pioneers of psychoanalysis. Illustrated with photographs, $9.95



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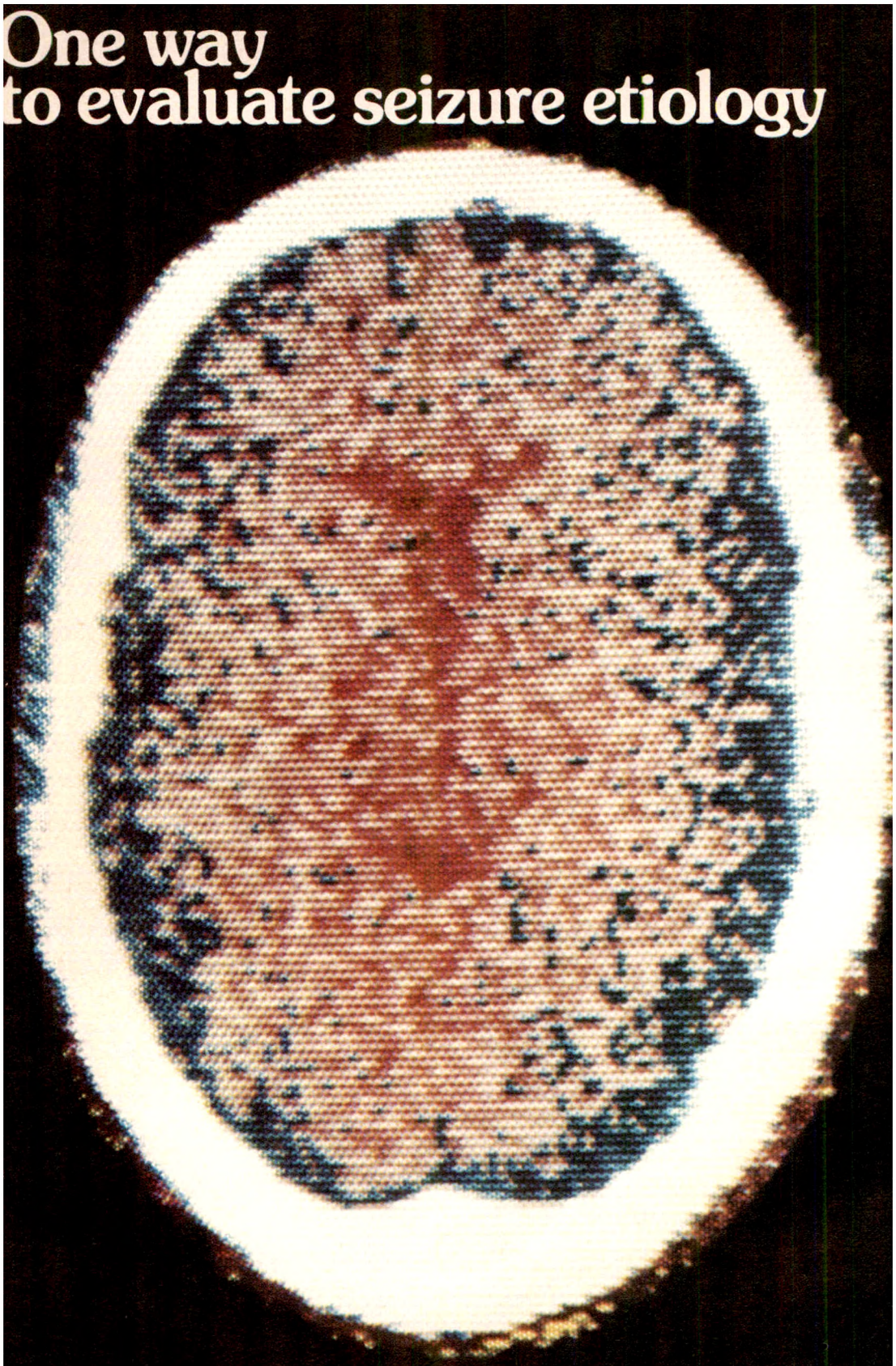
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Accreditation: 14 hours AMA Category I

Tuition: $160

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One way to evaluate seizure etiology



Computerized transaxial tomography (CT scan) is an important new noninvasive techniqu
that can help in determining the etiology of epileptic seizures. Shown here is a single slice
from a color-enhanced CT scan of the brain of a patient with epilepsy. The absence of vasc

A proven way to start seizure control

MYSOLINE®
(primidone)

Tablets 50 mg, 250 mg
Suspension 250 mg/5 ml

A single entity with multiple anticonvulsant properties

Primidone itself has anticonvulsant activity. In addition, its major metabolite, phenylethylmalonamide (PEMA), exerts an anticonvulsant effect of its own and also enhances the anticonvulsant action of the second metabolite, phenobarbital. In patients refractory to phenytoin and phenobarbital, primidone may prove effective as the sole therapy.[1]

Multiple indications—grand mal, psychomotor, and focal epileptic seizures

In grand mal, MYSOLINE is comparable in efficacy to phenytoin and phenobarbital.[2,3] In psychomotor seizures, phenobarbital is rarely effective.[1] In focal seizures, phenytoin used alone is not indicated[4]—and, according to Forster,[3] MYSOLINE is markedly more effective than phenobarbital, which should dispel the misconception that MYSOLINE is "just a phenobarbital." (See chart below.)

Efficacy ratings* of anticonvulsants in epilepsy

Agent	Grand Mal	Psychomotor	Focal
Mysoline	++++	++++	++++
Hydantoinates (phenytoin)	++++	+++	+++
Barbiturates (phenobarbital)	++	++	++

*Relative effectiveness, ++++ being most effective. Adapted from Forster, F. M.[3]

Consider it first for major motor epilepsies

MYSOLINE® (primidone) Ayerst®

See next page for Brief Summary and references.

Start seizure control with MYSOLINE® (primidone)

Initial dose in children over 8 years of age and adults: 250 mg daily at bedtime

BRIEF SUMMARY
(For full prescribing information, see package circular.)
MYSOLINE®
Brand of **PRIMIDONE** *Anticonvulsant*

ACTIONS: MYSOLINE raises electro- or chemoshock seizure thresholds or alters seizure patterns in experimental animals. The mechanism(s) of primidone's antiepileptic action is not known.

Primidone *per se* has anticonvulsant activity as do its two metabolites, phenobarbital and phenylethylmalonamide (PEMA). In addition to its anticonvulsant activity, PEMA potentiates that of phenobarbital in experimental animals.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several weeks before it can be assessed.

Usage in pregnancy: The effects of MYSOLINE in human pregnancy and nursing infants are unknown.

Recent reports suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Data are more extensive with respect to diphenylhydantoin and phenobarbital, but these are also the most commonly prescribed anticonvulsants; less systematic or anecdotal reports suggest a possible similar association with the use of all known anticonvulsant drugs.

The reports suggesting an elevated incidence of birth defects in children of drug-treated epileptic women cannot be regarded as adequate to prove a definite cause and effect relationship. There are intrinsic methodologic problems in obtaining adequate data on drug teratogenicity in humans; the possibility also exists that other factors, *e.g.*, genetic factors or the epileptic condition itself, may be more important than drug therapy in leading to birth defects. The great majority of mothers on anticonvulsant medication deliver normal infants. It is important to note that anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and threat to life. In individual cases where the severity and frequency of the seizure disorder are such that the removal of medication does not pose a serious threat to the patient, discontinuation of the drug may be considered prior to and during pregnancy, although it cannot be said with any confidence that even minor seizures do not pose some hazard to the developing embryo or fetus.

The prescribing physician will wish to weigh these considerations in treating or counseling epileptic women of childbearing potential.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

PRECAUTIONS: The total daily dosage should not exceed 2 g. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 g per day. The initial dose is 250 mg. Increments of 250 mg are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 g. A typical dosage schedule for the introduction of MYSOLINE is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg daily at bedtime	*2nd Week* 250 mg b.i.d.
3rd Week 250 mg t.i.d.	*4th Week* 250 mg q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg, with gradual weekly increases of 125 mg a day, to a daily total usually between 500 mg and 750 mg.

In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances: for initiation of combination therapy; during "transfer" therapy; for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.).

HOW SUPPLIED: MYSOLINE Tablets—No. 430—Each tablet contains 250 mg of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg of primidone (scored), in bottles of 100 and 500.
MYSOLINE Suspension—No. 3850—Each 5 ml (teaspoonful) contains 250 mg of primidone, in bottles of 8 fluidounces.

References: 1. Woodbury, D. M., and Fingl, E., in Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics, ed. 5, New York, Macmillan Publishing Co., Inc., 1975, p. 222. **2.** Rodin, E., in Conn, H. F. (ed.) Current Therapy 1977, Philadelphia, W. B. Saunders Company, 1977, p. 716. **3.** Forster, F. M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. **4.** Physicians' Desk Reference, ed. 31, Oradell, N.J., Medical Economics Co., 1977, p. 1175.

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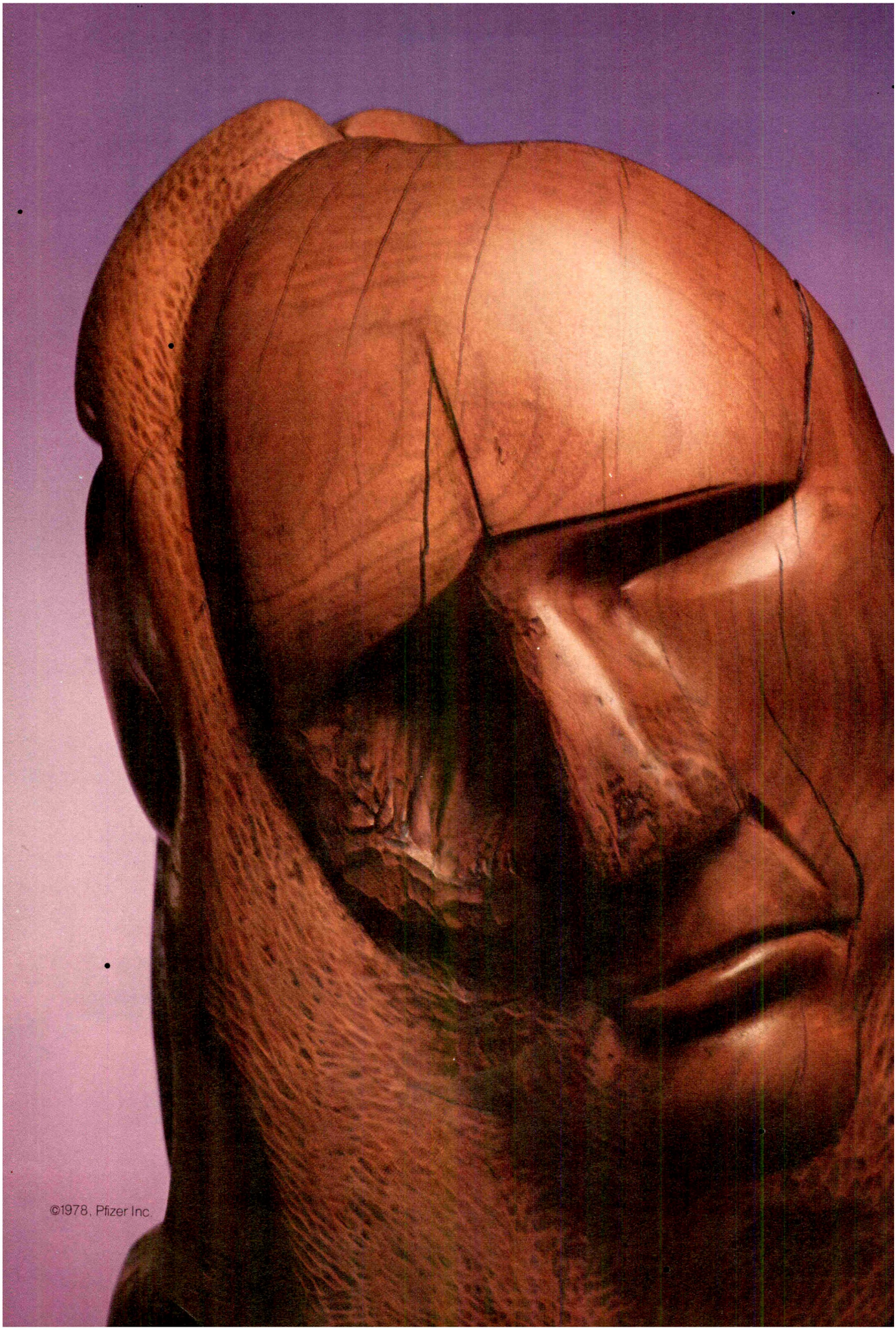
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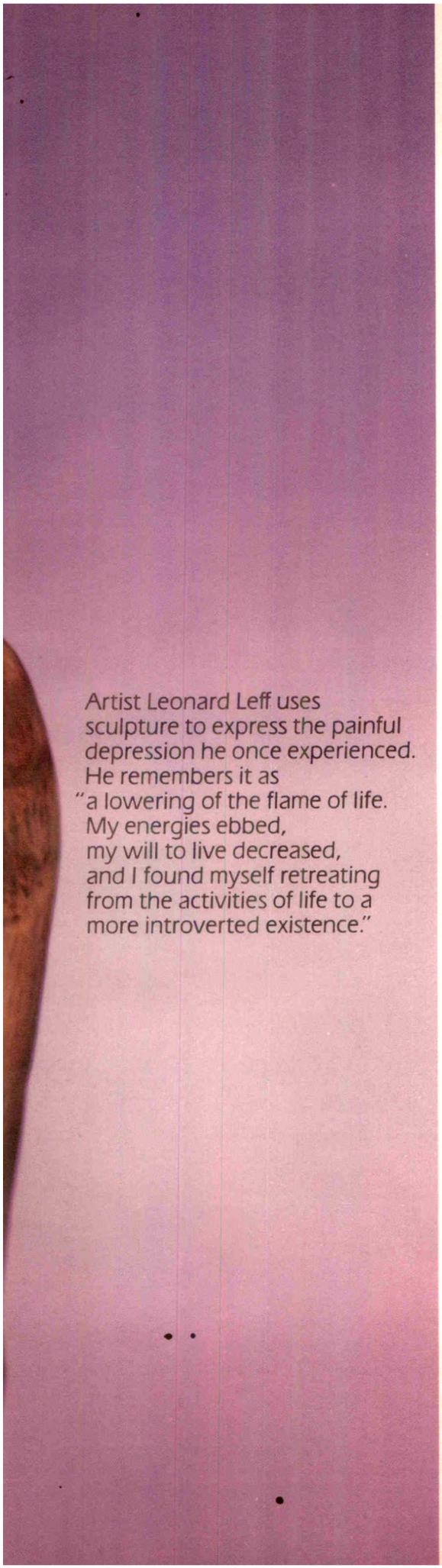
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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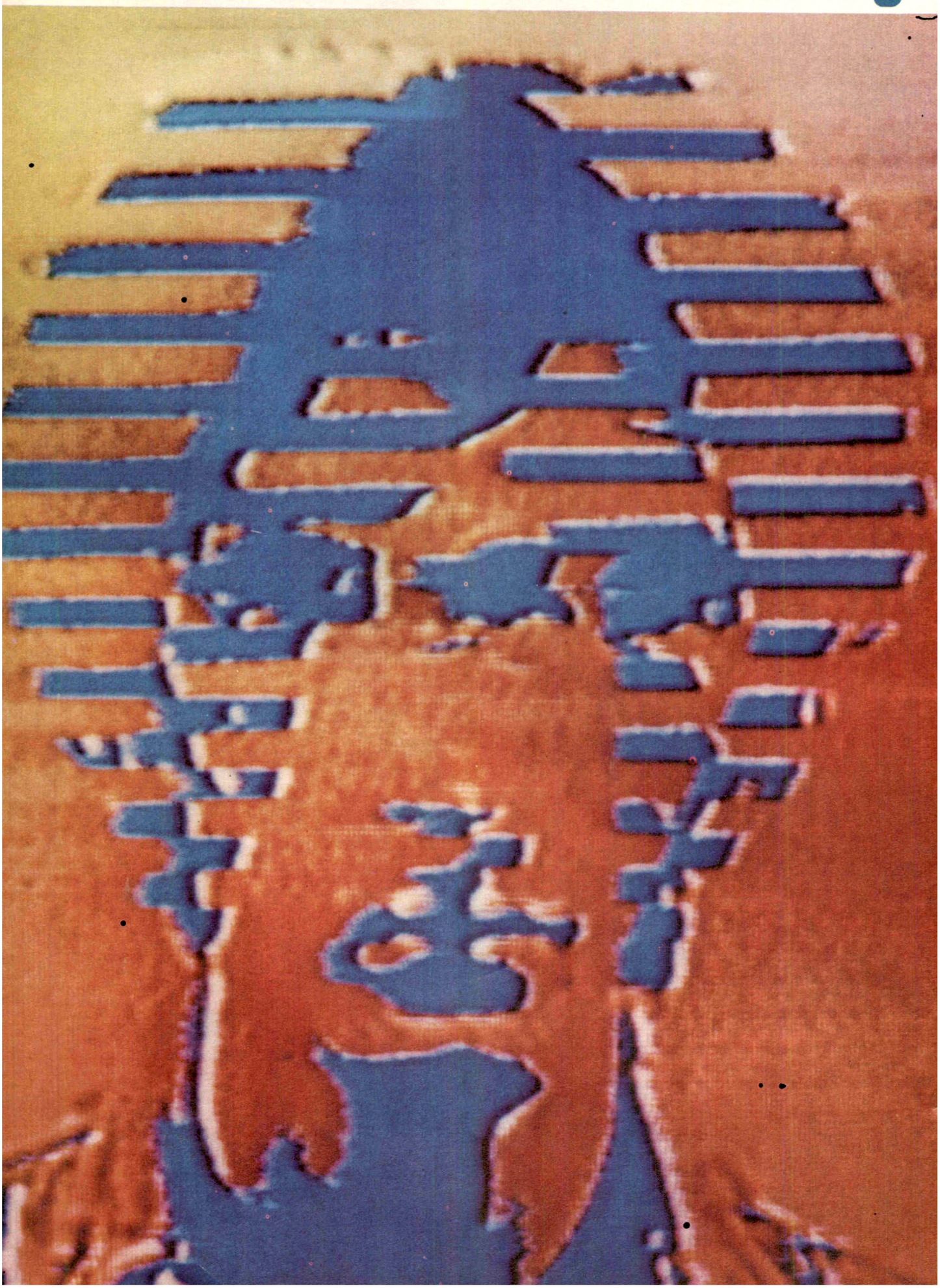
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molindone HCl

- an effective antipsychotic agent
- not a phenothiazine

FAVORABLE BENEFIT/RISK PROFILE, WHICH MAY ENHANCE COMPLIANCE*

NO incidence	Tolerance development Hepatotoxic changes Ophthalmological changes Excessive weight gain Impotence Decreased libido Skin pigmentation Thyroid function changes Persistent tardive dyskinesia
LOW incidence	Sedation Adverse drug interactions Hypotension Hematological changes Lactation Amenorrhea Autonomic changes Skin rash Nonpersistent symptoms of tardive dyskinesia
MODERATE incidence	Extrapyramidal reactions

*This table compiled from clinical and field reports, according to the best available information.

Further advantages with MOBAN®

The pharmacological effect from a single oral dose persists for 24-36 hours, permitting *h.s.* dosage, once the daily dosage level is established.

Several clinicians have reported that obese patients on MOBAN® frequently experienced a marked reduction in weight toward normal weight.

How to use MOBAN®

The usual starting dose is 50-75 mg/day. You can increase to 100 mg/day in 3-4 days. Titrate individually up or down depending on patient response. Some chronic, treatment-resistant patients may require an increase to 400 mg/day. (However, the long-term safety of 400 mg/day has not been established.) Please refer to complete prescribing information for further dosage instructions.

For complete prescribing information, please see next page.

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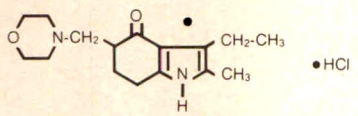
MOBAN®

molindone HCl

5 mg	10 mg	25 mg
Endo	Endo	Endo

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN® (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN® being excreted in urine and feces.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in the pregnant patient have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose—
20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose—
20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose—
5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN® Caution should be exercised where increased activity may be harmful.

MOBAN® does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN® has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological

Extrapyramidal Reactions

Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia

Motor restlessness may occur early.

Parkinson Syndrome

Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome

Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System

Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological

There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN® may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects

Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with MOBAN® therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN®.

Cardiovascular

Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological

Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN® (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN®.

Skin

Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN® usage alone.

MOBAN® (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN® is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia

Although rarely reported with MOBAN® (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50-75 mg/day.

—Increase to 100 mg/day in 3 or 4 days.

—Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.

—An increase to 225 mg/day may be required in patients with severe symptomatology.

—Some chronic, treatment resistant patients, may require up to 400 mg/day (however, the long term safety of 400 mg/day has not been established).

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

Dosages may be administered once a day.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN® in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN® in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles of 100 and 1000 with potencies and colors as follows: 5 mg orange, 10 mg lavender, 25 mg light green.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

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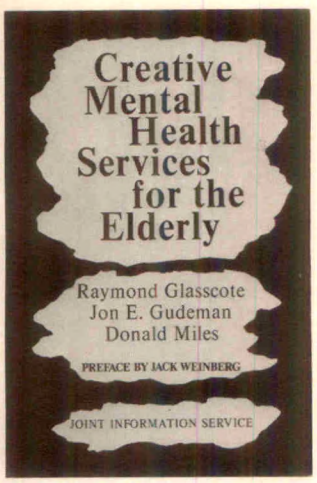
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The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



This carefully documented book reports a field study of ten programs, four in America and six in England and Scandinavia, which serve as practical demonstrations of creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elderly residents of several trailer parks
- a high school for the elderly which sends its students abroad for study trips
- a "Human Development Project" that focuses on responding to the psychological needs of the elderly
- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting document becomes a handbook, and, as well, a forum through which some of the world's most experienced practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Social Gerontology, characterizes this volume as "a masterful job of putting everything in relief . . . the guidelines for action are right here."

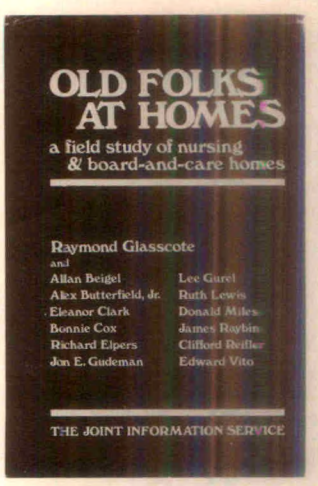
190 pages. Casebound. Price $8.50.

Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

The teams were surpised to learn that care and quality of life in many of the nursing and board-and-care homes they visited were better—sometimes dramatically better—than that in the mental health facilities and often at lower cost.

This unique publication provides an overview of all facilities visited plus vignettes of approximately half. Federal and state rules and regulations are also expertly synthesized into brief and readable style.

148 pages. Casebound. Price $6.50



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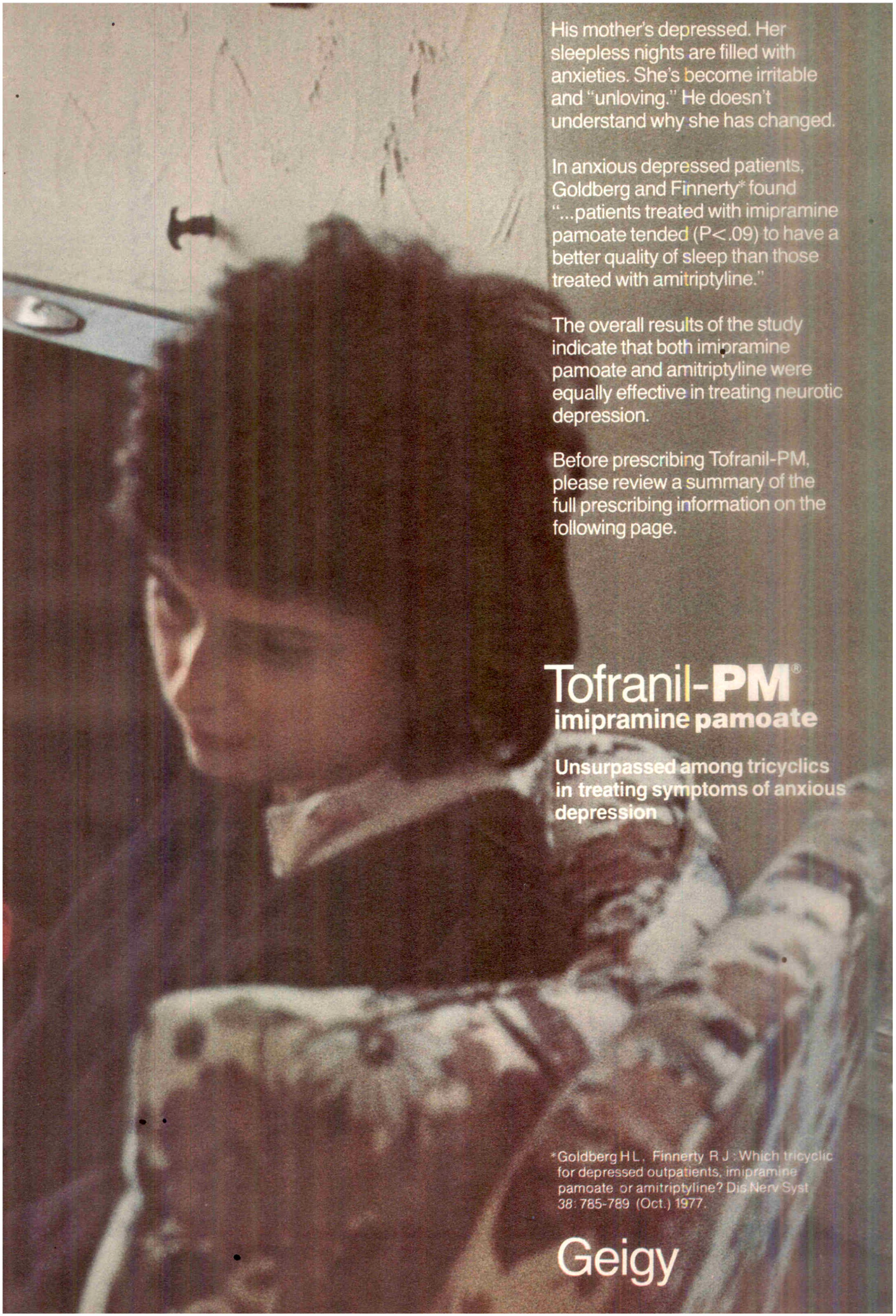
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AJP


"Please don't cry."



His mother's depressed. Her sleepless nights are filled with anxieties. She's become irritable and "unloving." He doesn't understand why she has changed.

In anxious depressed patients, Goldberg and Finnerty* found "...patients treated with imipramine pamoate tended ($P<.09$) to have a better quality of sleep than those treated with amitriptyline."

The overall results of the study indicate that both imipramine pamoate and amitriptyline were equally effective in treating neurotic depression.

Before prescribing Tofranil-PM, please review a summary of the full prescribing information on the following page.

Tofranil-PM®
imipramine pamoate

Unsurpassed among tricyclics in treating symptoms of anxious depression

*Goldberg H L, Finnerty R J: Which tricyclic for depressed outpatients, imipramine pamoate or amitriptyline? Dis Nerv Syst 38: 785-789 (Oct.) 1977.

Geigy

Tofranil-PM®
imipramine **pamoate**

Geigy

As anxiety, agitation, sleep disturbances, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)

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For complete details, including dosage and administration, please refer to the full prescribing information.

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ANXIETY

SURVEY OF 2244 ADULTS SHOWS A CHANGING PICTURE OF PERSONS AT HIGH RISK...

The results of an ongoing Chicago area survey[1] reveal that, contrary to patterns of the past, *day-to-day social stressors such as marriage, parenting and the job account for three to four times more symptoms of anxiety than do demographic factors or early emotional traumas.*

Persons with high symptomatology were defined as the top 13% of the sample. The situations and stresses these people face in their lives are the variables that place them at high risk for anxiety.

The Separated-from-Spouse Person

Persons separated from their spouses showed the highest incidence—40%—of high anxiety symptoms of any group in the survey. Married persons are at lowest risk for any group, with single, widowed and divorced people slightly higher.

The Lowest-Income, Least-Skilled Person

When examined according to socioeconomic variables, the data reveal that persons with the lowest income and occupational status show twice as much symptomatology as the four socioeconomic levels above them, in whom symptomatology varied little. Moreover, high risk demographic variables tend to cluster: considered independently, women and single people show high symptomatology, and 75% of the respondents in the lowest income level were female and 82% were unmarried.

Prevalence of anxiety symptoms by income, education, and occupational status[1]

	HIGH	OTHER	NUMBER OF RESPONDENT
Entire Sample	13%	87%	2,244
*INCOME LEVEL			
highest—1	13%	87%	77
2	7%	93%	267
3	9%	91%	845
4	13%	87%	544
lowest—5	32%	68%	220
*EDUCATIONAL LEVEL			
highest—1	9%	91%	143
2	9%	91%	569
3	11%	89%	790
4	19%	81%	444
lowest—5	16%	84%	294
**OCCUPATIONAL STATUS (presently employed respondents)			
highest—1	5%	95%	62
2	8%	92%	156
3	9%	91%	534
4	7%	93%	391
lowest—5	18%	82%	92

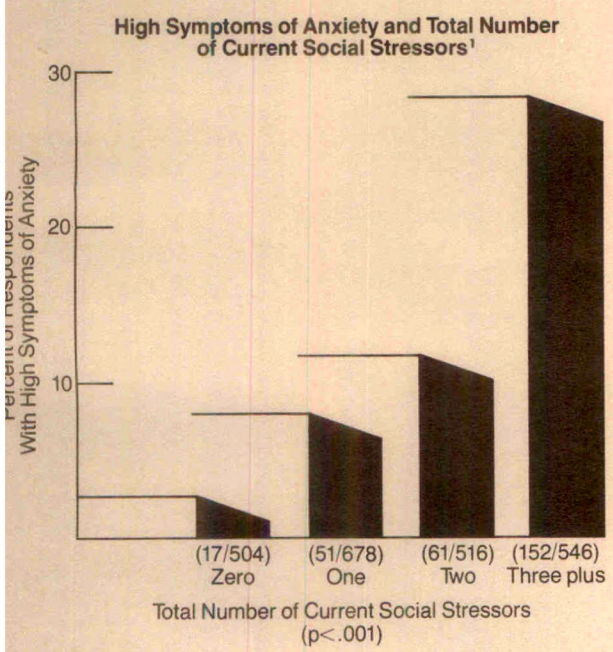
*$p<.001$ **$p<.01$



ROCHE

The Person with 3 or more Concurrent Social Stressors

When subjects are divided into groups based on the number of their current social stressors, it is clear that the percentage of subjects with high symptomatology increases sharply with increasing numbers of stressors. Only 3% of subjects with no current social stressors report high symptoms of anxiety; of those with three or more current stressors, 28% reported high symptoms ($p<.001$).



The Marital Stressor:

Marital/singlehood stressors had the highest and most significant correlation with symptoms of anxiety for all groups except employed single women. For single men and women, differing profiles emerge: the relationship between these stressors and anxiety is much closer for men than for women.

The Job Stressor:

Current stressors related to the job are, after marital stressors, the most closely related to anxiety symptoms for employed married fathers, and are also high for single men. For women, married or single, there was no correlation between job and anxiety. The only significant correlation with anxiety for employed single women was financial stressors.

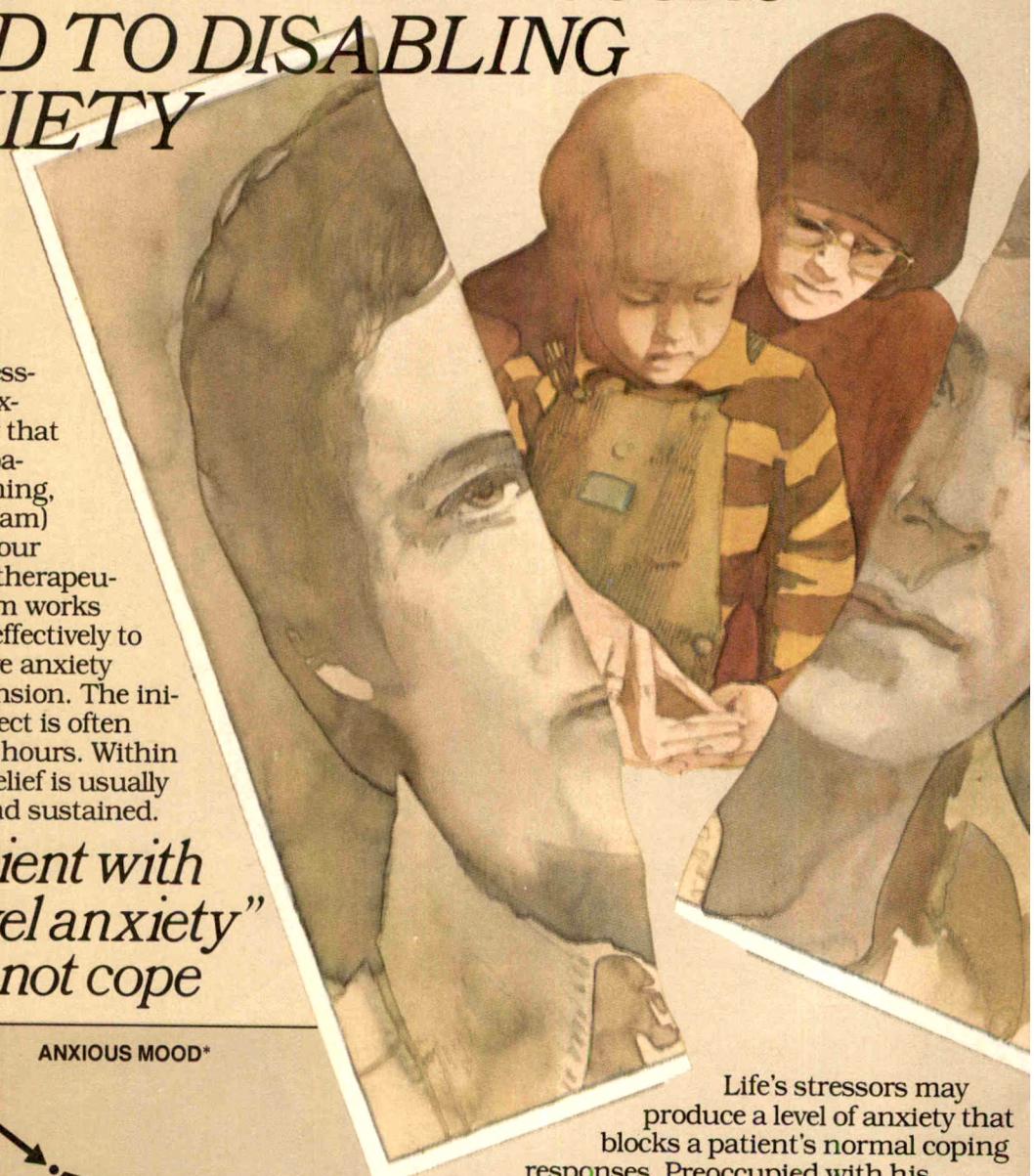
The Parental Stressor:

The interaction of current social stressors with demographic factors can be seen when the parental stressor is examined. This variable is positively correlated with anxiety in all mothers, but to a much more significant degree in unemployed mothers. Parental stressors are also related to anxiety in fathers, but at a lower correlation and with less significance than in employed or unemployed mothers.

1. Ilfeld FW, Jr: Persons at high risk for symptoms of anxiety. Presented at Excerpta Medica symposium "Clinical Anxiety/Tension in Primary Medicine," Washington, D.C., September 1977

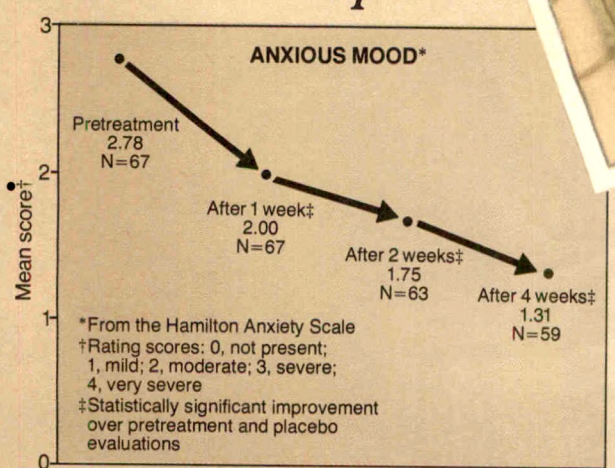


WHEN LIFE'S STRESSORS LEAD TO DISABLING ANXIETY



When the level of stress or the number of stressors produces excessive anxiety that inhibits your patient's functioning, Valium (diazepam) can be one of your most valuable therapeutic allies. Valium works promptly and effectively to relieve excessive anxiety and psychic tension. The initial calming effect is often evident within hours. Within days, anxiety relief is usually pronounced and sustained.

The patient with "high level anxiety" who cannot cope



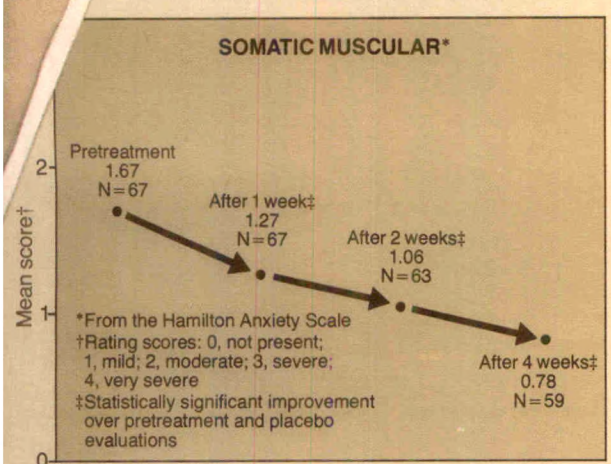
Life's stressors may produce a level of anxiety that blocks a patient's normal coping responses. Preoccupied with his symptoms, the patient may be unable to open up to psychotherapy. In such cases Valium (diazepam) can achieve a rapid response, which helps the patient participate. Reassured by the rapid anxiety relief, the patient becomes more amenable to therapy and better able to help himself. When anxiety has reached manageable levels, Valium should be discontinued.

ROCHE

The patient with anxiety accompanied by somatic symptoms

A recent collaborative study [2] confirmed what physicians throughout the world have found in clinical practice: Valium (diazepam) promptly relieves psychic *and* somatic anxiety symptoms. As anxious mood and tension diminished, the bodily symptoms that so often accompany the anxious state were significantly reduced.

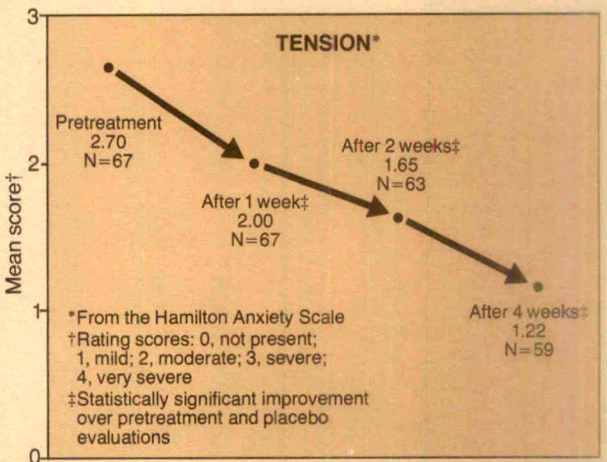
2. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley, New Jersey 07110



The patient with anxiety expressed through functional symptoms

When intense anxiety triggers CNS-mediated defense reactions, patients experience physiological effects they may interpret as organic illness. The prompt response obtained with Valium (diazepam) can successfully counter this anxiety-linked arousal. The rapid initial response to Valium reassures the patient and helps reduce the level of CNS reaction. This helps the patient understand the reasons for his functional disorders and reduces his preoccupation with his physical condition.

Three dosage strengths, all in scored tablets, make it easy to adjust dosage up or down as needed. In patients prone to anxiety at bedtime, adding an *h.s.* dose to the *b.i.d.* or *t.i.d.* regimen reduces anxiety enough to permit sleep.



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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Trifluoperazine	High
Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2-6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170-171.

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It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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Am J Psychiatry 135:9, September 1978

THE AMERICAN JOURNAL OF PSYCHIATRY

Issues Critical to the Survival of Community Mental Health

BY JONATHAN F. BORUS, M.D.

The author delineates some of the critical issues that face community mental health, with the opportunity for either growth and further development or regression into a decentralized type of custodial care. These issues include clarifying boundaries and priorities, caring for chronically ill deinstitutionalized patients, providing differentiated care, collaborating with the community, relating to the rest of psychiatry and medicine, defining the community psychiatrist's role, maintaining psychiatric manpower, undertaking evaluation research, and achieving stable funding. Psychiatry's response to these issues will help determine the resolution of this crisis and the future viability and direction of community mental health.

COMMUNITY mental health (CMH), the population-based, prevention-oriented, primarily publicly funded mental health service delivery system that has grown up in the United States over the last 14 years, faces a crisis in its development. Increasing political and scientific criticism has been coupled with significant funding cuts at a time when this "adolescent" still needs considerable support and resources to develop in a healthy manner (1–5). The national climate of therapeutic optimism, societal demand for distributive justice, and personal presidential interest that created CMH in the early 1960s changed dramatically during 8 years under administrations openly hostile to public mental health services (6). Today we again have a President who has a personal interest in mental health and who has appointed a commission to make recommendations mapping out the directions of mental health delivery for the foreseeable futu[illegible] of these deliberations will determine whe[illegible] ceives the resources necessary to rese[illegible] through further differentiation into a [illegible] and responsive public delivery system. [illegible] will die a slow death of fiscal starvation[illegible] attempts to bring some of the critical issu[illegible] lems facing CMH into sharper focus for [illegible] during this period of reassessment and pl[illegible] ture mental health services.

Dr. Borus is Assistant Professor of Psychiatry, Harvard Medical School, and Director of the Psychiatric Residency Training Program and the Subspecialty Training Program in Social and Community Psychiatry, Massachusetts General Hospital, Boston, Mass. 02114.

This work was supported by Alcohol, Drug Abuse, and Mental Health Administration training grant MH-14124 from the National Institute of Mental Health.

THE BOUNDARY PROBLEM

CMH has been frequently criticized f[illegible] boundaries and priorities, with allegatio[illegible] tried to be all things to all people and has [illegible] the mental health professions by becom[illegible] areas in which it has no expertise [illegible] boundary problems stem in part from th[illegible] mism of the early 1960s in which CM[illegible] marching into the community as the "[illegible] tack" (President Kennedy's words) on th[illegible] mental illness and mental retardation (8[illegible] this crusading atmosphere, federal leg[illegible] dated CMH to do the impossible, that [illegible] serve all citizens with defined mental illne[illegible] for treatment within a large (75,000–200[illegible] population-defined geographic area, but [illegible] mentally ill in the community who do no[illegible] treatment, prevent mental illness in the [illegible] in the community, and improve the me[illegible] the rest of the population. Parenthetical[illegible] prising that society sent psychiatrists t[illegible] the theoretically interesting but clinic[illegible] concepts of catchmented responsibility, [illegible] cus, prevention orientation, and consum[illegible] tion. Psychiatrists, always on the [illegible] fringe," were mysterious enough to be s[illegible] enough to go, and, as shown in recen[illegible] enough to disown if things did not work[illegible]

This march into the community too[illegible]

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some exciting areas of productive consultation and collaboration with such groups as educators, urban planners, and police. However, by the late 1960s some CMH professionals began to lose perspective and, instead of providing needed services, made direct attempts under the banner of prevention to change society by organizing protests against social ills, telling community leaders what to do, and involving themselves in other activities clearly beyond their professional expertise. They had deceived themselves into trying to fulfill the public illusion that psychiatry could solve societal problems; as is usually the case, public disillusionment with CMH followed shortly thereafter when the impossible could not be delivered. Unfortunately, in the minds of many prominent psychiatrists and politicians these misguided ventures of the 1960s persist in overshadowing the majority of CMH efforts aimed at improving the quality and quantity of direct mental health services in the country (9).

In the late 1970s, it is essential that CMH sharpen its boundaries, delimit its priorities, and increase its accountability if it is to survive. As the publicly funded mental health service delivery system, it must recognize its primary obligation to serve first those patients with the most serious and debilitating illnesses whom the public sees as most distressed and distressing. CMH has failed when it has not implemented a network of community-based services for such patients, and it has been most successful when it has increased psychiatry's links to the previously underserved through a realistic delivery system of mental health services (5). Although CMH professionals should contribute their knowledge of normal and pathological behavior to discussions of social issues important to the community (e.g., education, race relations) (10, 11), in the future they must scrupulously decline acceptance of tasks outside their areas of expertise, regardless of societal urgency or importance.

CARE FOR CHRONICALLY ILL PATIENTS

Community mental health centers (CMHCs), originally proposed as alternatives to care in state hospitals, have been criticized for avoiding the difficult task of providing the therapeutic services necessary to maintain severely ill and disabled psychotic patients in the community (1, 2, 12–14). CMHCs have often been unprepared or unwilling to meet the comprehensive service needs of the large number of chronically ill patients returned to the community as a result of the public policy of deinstitutionalization. This failure has resulted in inadequate care for these patients and citizen backlash deleterious to reintegrating patients into the community (15, 16).

If deinstitutionalization is to be more than a regressive shift of patients from the back wards to the back alleys, it will be a very expensive undertaking in both human and monetary costs. As detailed elsewhere (17, 18), meaningful deinstitutionalization necessitates the provision of a broad spectrum of services to reintegrate the patient into the community. These include the predischarge services of discriminate evaluation and individual patient selection for return to the community, assessment of family and community readiness for the patient's return, and preparation of the patient for the transition from hospital to community. Requisite services in the community include provision of basic subsistence needs, provision of sheltered living facilities, maintenance and monitoring of psychoactive medications, provision of appropriate mental health care, provision of meaningful ways to occupy time, and provision of supportive services to the patient's family and community. Recent federal legislation mandates CMHCs to provide posthospital aftercare as an "essential service" and to coordinate the multiple sources of financial, housing, mental health, and medical care needed by these patients (19, 20). CMH professionals should therefore collaborate with citizens' groups to insist that responsible government and political officials make necessary support services available *before* hospitals are closed and large numbers of patients are returned to the community.

PROBLEM DIFFERENTIATION AND SERVICE FIT

CMH has also been criticized as "second-class psychiatry" that substitutes quantity for quality care. Under the pressure of serving the entire population, evaluations are frequently relegated to the least experienced clinicians, and a limited range of therapeutic services are offered in volume. The essence of "first-class psychiatry" is treatment predicated upon the correct evaluation of each patient's problem and application of a differentiated therapeutic response to fit that particular problem (21). CMHCs should therefore use their most experienced clinicians (usually psychiatrists) in the evaluation process to interview or closely review each new patient in sufficient depth to responsibly pinpoint the diagnostic problem.

Accurate evaluation must be followed by fitting the problem to the best possible treatment alternative. This necessitates the availability of a wide variety of treatment and therapist alternatives; a CMHC that offers just support, just medication, just crisis intervention, or even just psychoanalysis to all of its patients is inherently providing second-class treatment. All treatment alternatives do not have to be available in one setting; a coordinated network of services (described below) can be established in which different therapeutic services are provided in different locations. CMH professionals should alert citizens' groups and politicians to the possible conflicts between quality and quantity care, i.e., the provision of a large quantity of inappropriate therapies is not helpful to patients and a smaller volume of service may be necessary to foster a higher quality and broader spectrum of treat-

ment alternatives that promote appropriate problem-treatment fit. Although cognizant of the urgent need to respond better to deinstitutionalized and chronically disabled patients, publicly funded CMHCs should also continue to devote some of their resources to treating patients with less severe disorders, consulting with community care givers, and undertaking prevention/education programs to avoid citizens' stigmatizing perception of them as "community mental illness centers" to be used only as a last resort.

RELATIONSHIP TO THE COMMUNITY

CMH has experienced confusion with the concept of and difficulties in its contacts with the community during its developmental years. The concept of a community as a cohesive group of citizens with common values, a shared sense of destiny, and natural support systems rarely fits the federally defined geographic areas with 75,000–200,000 inhabitants (22). These large "catchmented communities," often created de novo to acquire federal funding, usually include several smaller aggregates of citizens who have little in common with each other and disparate points of view. Many CMHCs have established satellite delivery sites to attempt to respond to the specific needs of these smaller subcatchment areas.

Some CMH programs have been more interested in enlarging the domain of a parent institution (e.g., university, hospital) with new buildings and staff than in working with the community to improve mental health service delivery. Although most centers have some sort of consumer-citizen board, these boards are often controlled by center staff and do not have the power of advice or consent in important policy issues. Community representatives too often are sent the double message that their views are of utmost importance—as long as they are in agreement with those of the professionals. In recognition of this bind, 1975 legislation requires that policy-setting citizens' boards be an integral part of any new CMHC that receives federal funds (19).

Task-oriented collaboration and shared control between consumers and professionals according to their differential expertise is a difficult but critical goal in these fiscally troubled times for CMH. Citizens, not psychiatrists, have the political muscle to let their legislators know that votes depend on continued support of scarce but vital resources for CMH. Citizen support is crucial to any plan to return deinstitutionalized patients to the community; citizens' boards can be key to the success of community education programs and can communicate to politicians the importance of having support services in place for deinstitutionalized patients to avoid community disruption. Since all of the "essential services" dictated by federal legislation cannot be provided to all who need them within any one catchment area, citizen participation is essential in deciding program priorities. CMH professionals should welcome the opportunity to share representatives the dilemma of having sources to meet an unlimited mandate comitant painful task of deciding what ser be provided and which patient needs served.

RELATIONSHIP TO MEDICINE

The growth of CMH has coincided wit psychobiological research, and in recer field of psychiatry has started to return to roots, led by collaborative efforts of psyc other physicians in the general hospita coordinated comprehensive health care psychiatrists have seen CMH moving in direction, away from and outside of me traditions, to form a separate power ba competition between CMH and other se chiatry for extremely scarce research and sources has caused much antagonism, an tings community psychiatrists are in dan their ties to and support from the genera

An evolutionary step for CMH, whic crease its ties to the rest of psychiatry patient care, would be for it to spearhead return to medicine at the primary care lev attention and legislation focus on impro tion's capacity to provide accessible p care for all citizens (24), it is important that CMH systems have been providing tal health care in the community for the (25). Rather than continuing to use freest ties isolated from general health services extend the coordination of medical and care begun in the general hospital to the prehensive primary care in the commun

Attempts at providing such coordina care have been made in neighborhood he and health maintenance organizations in of the country (25–28). Recent studies of programs in neighborhood health center this locus has several advantages for the primary mental health services (25, 29–3 clude greater accessibility and acceptabil sultant increased utilization of mental he when they are offered as part of general a local setting; better case-finding, referra up care when medical and psychiatric ca together under the same roof; and impro cation, consultation, collaboration, and among providers, fostering coordinatio fragmentation of care.

These studies also demonstrate the nee progress beyond the limits of a building-b system. The initial federal legislation fun struction of large CMHC buildings, an grams have attempted to provide all types care *in* this setting (the "edifice com

planners should shift their focus from the self-sufficient CMH *center* to a CMH *network* of services. In the latter system primary mental health services, provided as part of primary health care in the community, would be coordinated with more expensive and specialized secondary and tertiary level interventions (e.g., inpatient care, long-term insight-oriented psychotherapy) offered in freestanding CMHCs, general hospitals, and mental hospitals.

THE PSYCHIATRIST'S ROLE IN CMH

The role of the psychiatrist in CMH is often as ill-defined as the field's boundaries. Some psychiatrists have advanced beyond their area of clinical competence to assume major administrative roles, often with disastrous consequences. Most psychiatrists, as their centers' senior clinicians and only physicians, divide their time between direct service and indirect supervision and teaching of other clinicians. Even this role as clinical leader has been questioned in centers where sharing of expertise and multidisciplinary role overlap have degenerated into false egalitarianism. Some ask why the psychiatrist is paid more than nonpsychiatrist professionals who *could* be the psychotherapists, administrators, supervisors, and teachers; even medical evaluation and psychopharmacological prescription could be done through consultative referrals to primary physicians. False egalitarianism thrives when the psychiatrist's role is not clearly defined, leaving him in an uncomfortable, defensive position within his CMHC.

When such conditions exist, psychiatrists should diplomatically point out that their rigorous medical and psychiatric training, extensive leadership experience in patient management, and ultimate legal responsibility for patient care differentiate them from other clinicians. Although any separate part of the psychiatrist's role might be provided by another professional, the combination of medical, psychological, and management expertise and skills in one person makes him or her an invaluable resource. When the psychiatrist's access to a more lucrative private practice is also considered, the marketplace assigns him or her a higher salary than other CMH professionals; however, as a center's most expensive professional resource, psychiatrists have the obligation to question whether their costly expertise is being used most effectively.

Psychiatry must do a better job in preparing its trainees for the clinical, teaching, and administrative aspects of the community psychiatrist's role. Specific theoretical and practical experiences in community psychiatry should be a core part of residency programs, including opportunities to collaborate with, supervise, and teach other CMH professionals. Medical and neurological skills relevant to the diagnostic needs of the CMHC and consultative-collaborative work with primary physicians must be refined in residency training. In addition, if some psychiatrists are to administratively lead the complex, multimillion-dollar organizations that CMH delivery systems have become, there should also be subspecialty training programs that add specific executive-administrative skills to these psychiatrists' clinical expertise (32).

MAINTENANCE OF PSYCHIATRIC MANPOWER

The lack of adequate and stable rewards has led to a disturbing level of turnover in CMH's psychiatric manpower. Many prominent community psychiatrists have "burned out" and left CMH in recent years. Although the absolute number of psychiatrists working in CMHCs rose by 45% between 1972 and 1976 with the opening of new centers, psychiatrists' proportionate representation has decreased 27%, dropping from 6.4% to 4.7% of all CMHC staff (33, table 20). Many young psychiatrists work in CMHCs after their training (34, 35); often, however, it is a salaried position used as a temporary stepping stone to support them while they establish more rewarding private practices. This frequent turnover of psychiatric leadership has been detrimental to many centers' level of patient care, staff morale, and sense of continuity. Improving community psychiatry training may stimulate more psychiatrists to try CMH practice after residency (36–38). However, to stabilize the psychiatric foundation of CMH and avoid a "drift phenomenon" in which only those psychiatrists least qualified to work in other modes of practice perform its long-term service and leadership roles, CMH's unstable and inadequate reward system must be corrected.

The stability and mix of monetary, academic, developmental, and altruistic rewards often determine a psychiatrist's long-term satisfaction with his or her practice (39). A community psychiatrist usually earns between one-half and two-thirds of the income available for a comparable number of hours' work in private practice, and a recent study found this low fiscal return a definite deterrent to the long-term maintenance of psychiatrists in CMH (40). Without substantial federal help it will be difficult to raise CMH salaries to competitive levels, and many community psychiatrists accept the fact that they must supplement their income by private practice. However, more difficult to tolerate is the frequent intimation by political figures and the media that community psychiatrists who spend extra hours of their own time in private practice to supplement their low public salaries are in some way robbing the public purse, since all of the limitless needs of the citizens in their public catchment area have not yet been (and never can be) fully met. To decrease this distortion, a major public educational effort by the American Psychiatric Association is needed to put the monetary rewards of CMH practice into perspective.

Academic affiliations are extremely helpful in recruiting qualified psychiatrists to CMH by providing university appointments, intellectual stimulation, and

continuing education. However, academia specifically rewards those who conduct and publish research, while rarely recognizing clinical service and teaching in the community with academic promotion. Community psychiatrists usually have to "bootleg" their academic efforts from service time, and medical students and residents, noticing that there are few full or associate professors in CMH, are led to seriously question whether CMH is a respected and valued practice alternative. To keep psychiatrists attracted by the academic tie, CMH programs should designate time for research away from direct service provision to make the evaluation of CMH theory and practice a legitimate part of the psychiatrist's role.

The community psychiatrist's role can provide a variety of opportunities for personal and professional growth. With the nationwide demand, psychiatrists can usually find CMH positions in locations that satisfy the needs of their family and lifestyle. In his or her work the community psychiatrist has opportunities to further develop basic psychotherapeutic, consultative, and clinical leadership skills as well as any teaching, research, or administrative talents he or she may possess. However, if the psychiatrist's multiple skills are not used effectively and he becomes just the "prescription signer" of CMH, he will stagnate and soon begin to consider the possibility of doubling his income by providing such direct services in private practice.

Altruism is a major benefit available in CMH to psychiatrists who enjoy treating patients with the greatest disabilities and needs. However, it is a painful experience for altruistic psychiatrists who work in the public sector serving the chronically ill and debilitated to find themselves the targets of projected anger from a society ambivalent about the costs and difficulties of caring for this patient population. Under the pressure of the public arena, altruism alone is rarely sustaining over extended periods of time; if the other types of rewards are inadequate, altruism wears thin and "burnout" begins as the community psychiatrist discovers that the reverse side of altruism is masochism. In summary, if a more adequate and stable mix of rewards does not become available in CMH, the best and the brightest psychiatrists will not enter or stay in the field.

PAUCITY OF EVALUATION

Insufficient evaluation has contributed significantly to the political and scientific criticism of CMH and has made it extremely vulnerable to cuts in funding (41, 42). Realistic reasons that little careful evaluative research has been conducted to date include CMH's action orientation and constant demand for service, which allow little time for evaluation; the complexity and dynamic state of communities that afford almost none of the laboratory's possibilities for controlling some variables to study others; and frequent public opposition to research that is perceived as exploiting the community. Despite these obstacles it i[illegible] a scientific and a fiscal necessity for CMH [illegible] als to examine the field closely (43, 44).

Evaluation research in CMH should fol[illegible] hierarchy of studies (45). It should first ass[illegible] ment system's effectiveness in decreasing p[illegible] morbidity in the patient, his family, and t[illegible] nity at large; it should then assess the syste[illegible] sibility and utilization; and it should asse[illegible] tem's costs compared with similarly effect[illegible] cessible alternatives (45). Outcome [illegible] investigate the long-term impact of comm[illegible] ment compared with treatment in an insti[illegible] ting are essential to understand the proce[illegible] stitutionalization. Although existing studie[illegible] community treatment may inhibit chronici[illegible] psychotic patients, additional studies are nec[illegible] sess whether community treatment re[illegible] chronic disabilities of patients institutio[illegible] many years. Another outcome too frequ[illegible] sessed is patient satisfaction with the thera[illegible] ices offered by the CMHC. CMHC utiliz[illegible] should be compared with those of other de[illegible] tems and with national rates to assess acce[illegible] care, and patient demography should be s[illegible] determine whether CMHCs treat patient[illegible] lems left unserved by other care provide[illegible] the comparative costs of *quality* care in diff[illegible] ment locations (neighborhood health ce[illegible] standing CMHCs, general hospitals, psych[illegible] tals) should be ascertained, remembering[illegible] cost, poor-quality care is no bargain.

These extensive research needs will requ[illegible] es in the accessibility of research expert[illegible] amount of earmarked evaluation funds fro[illegible] ty, state, and federal sources. Most CMHC[illegible] ther the in-house investigators nor the [illegible] committed funds to undertake these essen[illegible] by themselves and will require both help [illegible] ued government pressure in order to acce[illegible] (46).

UNSTABLE FUNDING

Underlying each of the previous probl[illegible] the need for a more stable economic base [illegible] survive. It has been disheartening to profe[illegible] catastrophic to patients to see program[illegible] years of painstaking work decimated in a [illegible] by withdrawal of government funding [illegible] placed "fiscal restraint" or political whim[illegible] rect service provision must move away fr[illegible] nerability of total dependence on annual[illegible] ated, politically controlled funding from [illegible] sources. President Kennedy's prediction [illegible] support for mental health services would [illegible] essary for a few years until private insura[illegible] nisms were developed to pay for such servi[illegible] not been realized, and we will have to w[illegible]

tional health insurance is enacted to allow everyman to buy his own mental health services.

A fee-for-service, free choice, "voucher" type of national health insurance would provide CMH delivery systems with the ultimate evaluation in which they would either succeed or fail on the open market. The CMH systems that have worked well with their communities to develop services and programs uniquely suited to their citizens' mental health needs will probably continue to be used by a broad spectrum of consumers and become financially stable. Those which have offered nothing unique to their communities will be bypassed in preference to private practitioners by all but the least desirable patients. Since most national health insurance proposals focus primarily on reimbursement for direct services, public funding will continue to be needed through direct subsidy or capitation mechanisms to support the crucial CMH functions of training, evaluation research, consultation and education, and coordination of health and mental health services.

CONCLUSIONS

This paper has delineated some of the critical issues that face CMH with the opportunity for either growth and further development or regression into a decentralized type of custodial care. Psychiatry's response to these issues will help determine the resolution of this crisis and the future viability and direction of CMH.

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Foundations' Fund Prize for Research in Psychiatry

The American Psychiatric Association is inviting submissions for the second annual Foundations' Fund Prize for Research in Psychiatry.

Candidates for the prize should be citizens of the United States or Canada and should be nominated by a sponsor. Sponsors should be members of the American Psychiatric Association. Members of the prize board are excluded from submitting nominations.

The *sponsor* should submit a supporting letter justifying the nomination in detail and summarizing the nominee's research accomplishments.

The *nominee* should submit the following:

1) An up-to-date curriculum vitae,
2) An up-to-date bibliography,
3) A group of representative and thematically linked papers or books published (or accepted for publication) in English and dated within 10 years of the deadline for submission, and
4) A summary statement written by the nominee that emphasizes the principal theme or themes running through the work and its internal cohesiveness and consistency.

Six copies of each entry should be submitted to Joel J. Elkes, M.D., Chairperson, Foundations' Fund Prize Board for Research in Psychiatry, American Psychiatric Association, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but cannot be returned. The award will be presented at the Convocation of Fellows at the APA annual meeting in May 1979.

The deadline for submission is December 1, 1978. Any entry received after that date will automatically be entered for consideration for the 1980 award unless withdrawn by the nominator or candidate.

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Current Medication Use and Symptoms of Depression in a General Population

BY THOMAS J. CRAIG, M.D., M.P.H., AND PEARL A. VAN NATTA, M.A.

In a community survey, 41.8% of 771 men and 60.2% of 1,059 women reported having used one or more medications in the 48 hours before the interview. The use of medications and the number of medications used increased progressively with age among both men and women. Respondents who used four or more medications included significantly more high scorers on a depression checklist than those who used fewer medications. The group of women who used minor tranquilizers and sedatives included significantly more high depression scorers than those who did not. Among both sexes, those scoring in the depressed range who were receiving psychotropic medication tended to be taking minor tranquilizers or sedatives.

STUDIES of medication use in the past decade have used data derived from the prescribing habits of physicians (1–4) and from surveys of general and patient populations (5–9). The time periods for which this information has been collected range from one month to one year (6–8), and analyses have generally been restricted to either a limited number or type of drugs (e.g., psychotropic medication) (1–7) or a relatively limited purpose (e.g., prevalence of adverse reactions) (9).

The present study is unique in several respects: 1) inclusion of all medication use reported by respondents in a community survey, both prescription and nonprescription, 2) focus on current drug use (i.e., within the 48 hours preceding the interview), and 3) examination of the relationship between current drug use and symptoms of depression. The last aspect is particularly important in view of reports indicating that, in populations of medically ill patients, depression is frequently underdiagnosed and is inappropriately treated even when recognized (4, 10–12).

At the time this study was done Dr. Craig was Assistant Professor, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, Baltimore, Md. He is now Director, Admissions and Training Unit, Rockland Psychiatric Center, Orangeburg, N.Y. Ms. Van Natta is Research Associate, Department of Epidemiology, Johns Hopkins University School of Hygiene and Public Health, Baltimore, Md. Address reprint requests to Dr. Craig at 40 Woodbine Rd., New City, N.Y. 10956.

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METHOD

The present data are taken from an interview survey carried out in Washington County, Md., during 1972–1974 in conjunction with the Center for Epidemiologic Studies (CES) of the National Institute of Mental Health (13, 14). The major emphasis of the survey was on depressed mood and related symptomatology. One depression rating scale, the CES-D, was included (15). The CES-D has a potential range of 0–60, obtained by summing the weighted number of days of the week preceding the interview during which the respondent experienced the feeling described by each item (16).

A cutoff score of 16 or above was found to best distinguish between depressed and nondepressed psychiatric patients (17)[1] and was the cutoff score used in the present study to identify individuals with high levels of symptoms of depression.

As an index of recent medication use, the respondent was asked, "In the past 48 hours, what medicine or drugs have you taken?" The specific medications reported were then listed along with the reasons for taking them. Although it was not possible because of the nature of the study to stimulate the respondents' recall as has been done in other studies (e.g., with charts or lists) (6, 7, 9, 18), the interviewers were asked to verify drugs whenever possible by asking to see the bottles.

The questionnaire was administered in two versions. The earlier version was administered to 741 subjects in this study (an additional 930 subjects had been given the first questionnaire before the medication question was included), and the later version was administered to 1,089 subjects. None of the differences in the two versions of the questionnaire directly affected the CES-D or the medication question, and extensive analyses revealed no significant differences between responses on the first and second versions. For this reason, the samples for both versions are combined in the present analysis. The 1,830 interviews represent a response rate of 80.5% for the first version and 75% for the second version.

All of the interviewers were middle-aged women, which may have biased the information obtained. The extent of this bias could not be accurately estimated, but a report on the earlier version of the questionnaire

[1]Weissman MM, Sholomskas D, Pattenger M, et al: Assessing depressive symptoms in five psychiatric populations: a validation study (unpublished paper).

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(14) demonstrated no significant differences among interviewers in respondent reports of total medications used.

RESULTS

For the total population (N=1,830), 41.8% of the men and 60.2% of the women reported the use of one or more medications during the 48 hours preceding the interview. Among men, the proportion using medication ranged from a low of 23.7% among the 18–24-year-olds to 67.3% of those in the 65 years old and older age group. The corresponding figures for women were 52.1% in the 18–24-year-old group and 75.6% in the 65 years old and older category. In the 65 years old and older category for both sexes, approximately 10% of the population reported that they used four or more drugs. In the 55–64-year-old group, however, 7.0% of the men and 4.9% of the women reported using four or more medications. Among respondents reporting the use of one or more drugs, there was no significant difference between men and women either in age distribution or in number of drugs used. The distribution of CES-D scores by number of medications used (see table 1) reveals a consistent trend for men and women. Those respondents who used four or more drugs were significantly more likely to have high scores for depression than those using three or fewer ($p<.05$, chi-square test); more than 30% of the men and 40% of the women scored 16 or greater.

When all of the drug classes were examined, only two medication groups, minor tranquilizers and sedatives, showed significantly greater proportions of high CES-D scorers among users in comparison with nonusers ($p<.01$, chi-square test), both true only among women. Among men, 18.2% of those who used tranquilizers and 21.1% of those who used sedatives had CES-D scores above 16, compared with 11.0% of nonusers. In contrast, among women, 36.7% of those who used tranquilizers and 51.4% of those who used sedatives had high CES-D scores, compared with 17.2% and 17.9%, respectively, of nonusers.

The proportions of respondents with elevated (16 or more) CES-D scores who were receiving psychotropic medication are listed in table 2. Although not all respondents who scored above 16 could be considered clinically depressed and in need of treatment, it is reasonable to assume that a large proportion of them were depressed. An additional cutoff score of 30 or more is included to identify a group considered at extremely high risk of depression. In the group with scores of 16–29, the vast majority of those who were receiving any psychotropic medication were taking either minor tranquilizers or sedatives; only a handful reported using an antidepressant. In the high-risk group (scores of 30 or more), this predominance of tranquilizer-sedative use is even more striking.

Among the total group of respondents with scores of 16 or more, only 1.1% of the men (N=87) and 3.5% of the women (N=201) were using an anti[illegible] the time of the survey.

TABLE 1
Number of Medications Used by 771 Men and 1,[illegible] CES-D Scores

Item	CES-D Sc[illegible] 0–15 N	%	16–29 N	[illegible]
Number of medications used by men				
None (N=449)	401	89.3	44	9[illegible]
1–3 (N=297)	266	89.6	27	9[illegible]
4 or more (N=25)	17	68.0	7	2[illegible]
Number of medications used by women				
None (N=421)	357	84.8	51	12
1–3 (N=590)	474	80.3	89	15
4 or more (N=48)	27	56.2	15	31

DISCUSSION

Several characteristics of the present [illegible] differentiate it from previous studies [illegible] use. The site of the survey is a county [illegible] Maryland whose population is largely wh[illegible] white) and relatively equally divided be[illegible] (40% reside in Hagerstown, a city of [illegible] 40,000) and rural (40%) populations. Its [illegible] sphere might be defined as typical of "[illegible] ca," and the drug use patterns would [illegible] differ to some extent from those found [illegible] and, especially, western areas (6–8).

The relatively short time interval [illegible] which the respondent was queried was [illegible] to maximize recall and to determine, as [illegible] sible, current drug use. In its brevity, [illegible] fers from studies that covered time perio[illegible] month to one year (6–9). Because the p[illegible] the present survey was on depression, [illegible] time could be devoted to detailed inqu[illegible] drug use, as has been the practice in oth[illegible] 9, 18). Several authors have commented [illegible] tive underestimates that may occur in [illegible] (18, 19). Our belief was that the short d[illegible] and the ascertaining of exact informatio[illegible] minimized this bias, but we have no direc[illegible] suring its effect on the present data. He[illegible] parison of the rates obtained in this surve[illegible] data from surveys covering longer time [illegible] to support our assumption.

For example, Parry and associates (7) [illegible] the northeastern United States over a [illegible] 16% of men and 33% of women used p[illegible] nonprescription minor tranquilizers an[illegible] or hypnotics. Our findings were 6.8% [illegible] 12.6% for women in a *48-hour period*. S[illegible] hold true for the other psychotropic [illegible]

TABLE 2
Type of Psychotropic Medication Taken by 771 Men and 1,059 Women, by CES-D Scores

	CES-D Score											
	Men						Women					
	0–15 (N=684)		16–29 (N=78)		30 and Over (N=9)		0–15 (N=858)		16–29 (N=155)		30 and Over (N=46)	
Type of Medication	N	%	N	%	N	%	N	%	N	%	N	%
Minor tranquilizer	27	3.9	4	5.1	2	22.2	62	7.2	28	18.1	8	17.4
Major tranquilizer	2	0.3	2	2.6	0		4	0.5	2	1.3	0	
Tranquilizer-antidepressant	0		0		0		4	0.5	1	0.6	2	4.3
Antidepressant	1	0.1	1	1.3	0		8	0.9	3	1.9	1	2.2
Sedative	15	2.2	4	5.1	0		17	2.0	11	7.1	7	15.2
Stimulant	0		0		0		3	0.3	0		0	

parison of our data with those of Seltzer and associates (8) and Greenblatt and associates (5) further strengthens our assumption that we minimized underestimation. In the former (8), a range of 7.6%–11.4% of men and 14.8%–20.6% of women reported using psychotropic drugs in the previous year, and in the latter (5), 15% of men and 25% of women reported psychotropic drug use in the three months before the study.

Our overall findings of drug use are comparable to those reported in other studies of medication use in patient populations in that drug use tends to increase in both amount and frequency with age in both sexes (4–9). In addition, women of any age tended to be more likely to use drugs than men. As has been pointed out elsewhere (7), the latter may be a relatively deceptive finding, especially as it relates to psychotropic drugs, because we also found that the men tend to use alcohol significantly more often than women (20). A finding that has not been stressed in previous studies, however, is the observation that when only users of medication are examined, the age distribution and distribution of number of drugs used were essentially the same in men and women. This suggests that although more women than men use drugs, there is no significant sex difference among medication users in overall pattern of drug use (by chi-square test).

Another striking finding is the fact that more than 40% of the men and 60% of the women surveyed had used at least one medication during the 48 hours before the interview. These figures exceed those of Seltzer and associates (8), who reported a rate of 24.4%–26.1% among white men and 44.6%–46.3% among white women in the year before the interview. Particularly important is the high level of drug use among the elderly (those 65 years old and older). In this age group, 22.1% of the men and 27.3% of the women reported taking three or more drugs. This very high prevalence of current medication use emphasizes the importance of obtaining complete medication data in clinical medicine to avoid the possibility of introducing adverse drug interactions (9, 21).

Table 1 shows that the percentage of people with symptoms of depression was significantly greater among individuals taking four or more medications ($p<.05$, chi-square test). Only two categories of medication were associated with a significantly higher proportion of high CES-D scorers for users than nonusers (minor tranquilizers and sedatives) ($p<.01$), and this was true only among women. This finding is of particular importance in view of the reports that depression, when recognized, is frequently treated with these categories of drugs, which have been shown to be of little benefit in the treatment of depression per se (although they may assist with such specific symptoms as insomnia) (10, 11, 22). In addition, a 1972 report (23) suggested that high doses of diazepam, the most frequently used minor tranquilizer in this study, may cause depression in nondepressed individuals. Furthermore, antidepressants have been shown to be specifically effective treatment not only for symptoms of depression but for anxiety as well (22, 24).

Perhaps the most disquieting data in the present study relate to the recognition and treatment of depression. Several studies (4, 25) have emphasized the prevalence of symptoms of depression among the medically ill. Further, several studies (26–28) have emphasized the need for early recognition and treatment of depression as a preventive for such negative outcomes as suicide and disability.

Since analysis of drug pairs revealed very little overlap in the use of psychotropic drugs (2 respondents who used antidepressants were also using minor tranquilizers, and 4 who used sedatives were also using minor tranquilizers), the data in table 2 represent virtually mutually exclusive categories. Approximately 14% of the men and 29% of the women whose CES-D scores were in the 16–29 range were using psychotropic medicine. Corresponding figures for CES-D scores of 30 or more were 22.2% for men and 39.1% for women, suggesting that psychological distress was identified and currently being treated in a substantial proportion of these groups. Even allowing for the possibility that some of these individuals may have had medical illnesses that precluded the use of antidepressants, the proportion using antidepressants in these groups was minuscule. Furthermore, the data suggest that high scorers were likely to be disproportionately older and more medically ill than the general popu-

lation, factors that substantially increase the risk of suicide.

In view of these rather startling findings, it is imperative that steps be taken to improve the recognition of depression among physicians and the general public. In addition, it is clearly the task of the medical profession to educate its members, especially those in nonpsychiatric practice, to the specific treatment of depression. At the present time, if depression is recognized at all, it is most likely to be treated with nonspecific remedies (minor tranquilizers and sedatives) that, at best, offer merely palliative symptomatic relief and, at worst, may intensify the distress they are intended to treat.

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The Military Family Syndrome

BY DON M. LAGRONE, M.D.

The author reviewed the case records of 792 children and adolescents seen in a military clinic over a two-year period. His data suggest that the incidence of behavioral disorders was higher in this clinic than in a civilian mental health center. Seven problem areas common to records in which a behavioral disorder was diagnosed were found. The author suggests that the behavioral problems of the group studied represent a process of acting out in the rigid, autocratic system of the military. He also suggests that an approach using systems theory and modified goals may be more helpful in dealing with problems of military families than the traditional individual approach.

FOR TWO YEARS (from 1974 to 1976) I was a member of a military child and adolescent mental health team serving families living on a large military base in the Midwest. I was somewhat surprised and puzzled by the large number of behavioral problems referred to our service and by the frequency with which certain themes appeared in the interaction of these families. In an attempt to understand the reasons for these phenomena, I first reviewed the literature and then reviewed the case material itself. In this paper I will present my findings and conclusions.

LITERATURE REVIEW

Little has been written about military families. Such authors as Cretekos (1), Bey and Lange (2), Gonzalez (3), Pearlman (4), Crumley and Blumenthal (5), and MacIntosh (6) have reported the intrapsychic conflicts of wives and children of military fathers absent from home. As in grief reactions, anger, mourning, and guilt are predominant in these wives and children. Crumley and Blumenthal (5) found that separations from the father substantially altered the development of children, particularly in superego formation and in object relations. Bloom (7) found that 24% of his patients were diagnosed as having behavioral disorders, compared with 16% in a civilian group. Among Bloom's patients, 2 out of 3 had a history of father absenteeism. Bonovich (8) described enforced family separations in the military as crisis situations, called for further study, and recommended crisis intervention techniques to deal with the problem.

CASE REVIEW

The patients whose case records were reviewed for this paper came primarily from the local families of active duty personnel residing on the base. Over the two-year period, 792 new patients were seen in the children's mental health clinic. In any given year, there are about 3,200 families with 7,400 children living on the base. Thus, new evaluations were being done for 12% of about 6,400 families and almost 6% of about 12,800 children.[1]

Of the case reports reviewed (N=792), 4% had the diagnosis of psychosis; 8%, mental retardation; 32%, behavior disorders; 7%, organic brain syndrome; 12%, neurosis; 3%, psychophysiological disorder; and 28%, transient situational disturbances. Six percent had no psychiatric diagnosis.

More than 94% of these patients were from the families of enlisted persons, compared with 6% from officers' families. At the time of referral, 12% of the fathers were absent from home.

The case review revealed seven problem areas common to many of the families and almost universally present in the case records with the diagnosis of behavioral disorders. These were the relationship the family had with the military, father absence, transiency, child-rearing methods, scapegoating, the husband-wife relationship, and resistance to treatment.

The Military-Family Relationship

To understand the military's relationship with the military family, one must understand something of the military itself. Goffman (9) used the term "total institution" to define such organizations, referring to the all-encompassing effect they have on the lives of their "inmates." Like other bureaucracies, the military is characterized by the fact that large groups of people are organized and administered by a smaller group. Ef-

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The author would like to thank Dr. Jon Cargill and Tsgt. Brad Billingslea for their collaboration in preparing this paper.

[1]Because of the rapid turnover of families on the base it is difficult to say that the same families would be on the base two years in a row. Estimates and averages were provided by the base public relations officer from statistics obtained by the base personnel office.

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ficiency is a key factor, and one person's infractions stand out against the compliance of the rest. When this happens, the system responds by concentrating on the individual until he or she is as compliant as the rest or is evicted from the system.

The process the military uses to force compliance is interesting and fairly well known. In total institutions, Goffman called this process "mortification":

> The recruit . . . [enters] with a conception of himself made possible by certain stable social arrangements in his home world. . . . [Stripped of this,] he begins a series of abasements, degradations, humiliations and profanations of self. (9, p. 14)

As a result of this, the "recruit's" life is managed by someone else, so that when he or she considers entering the outside world he or she may feel temporarily incapable of handling his or her affairs.

Greenblatt (10) described the government bureaucracies he worked with as materialistic paranoid systems. The same atmosphere pervades the military. Any special interest in one's job beyond the required minimum makes one suspect of individual aspiration, thus ensuring compliance and a high degree of frustration.

Another source of tension comes from the authoritative hierarchy that exists in the military. The individual's chances for being sanctioned are greatly enhanced by such a hierarchy. When sanctioned, the individual is allowed no face-saving expression; insolence is grounds for further punishment.

One other process that operates is that of the go-between (11). Confrontation is actively discouraged. Conflict between individuals is mediated by their supervisor. Problems with anyone outside the individual's unit are channeled through the chain of command. This relegates the individual to a child's status and discourages communication.

The military system binds its members tightly, and when such outside relationships as those with families are introduced a triangle is formed—with a very strong coalition between the father and the military. The family becomes the third point of the triangle, and all of the pathology that results from such triangles becomes possible. It sooner or later becomes apparent to the family that they are on the "outside." They are classified as "dependents" and have base privileges only as long as the father is on active duty or is alive. They must move from their housing on base if he is transferred or killed. For the military, the family exists only as an extension of the father, and he is expected to enforce compliance within the family. All of this serves to strengthen the father's coalition with the military and to cause the other family members to withdraw into their own systems of support.

Father Absence

Probably the most severe stress a military family faces is enforced separation from the father. In all of the case reports reviewed, the father was [illegible] the home at least once. In 12% of the ca[illegible] sought at the time of his absence. This s[illegible] important factor because in a 20-year ca[illegible] man makes an average of four overseas [illegible] as an indeterminable number of other tri[illegible] home for school, temporary duty, and [illegible] matters. When he leaves, whatever s[illegible] been to the family is gone or reduced. [illegible] lies, one of the members may become [illegible] and make requests for the father's re[illegible] families one member may take over the [illegible] father had.

In interviews with military wive[illegible] Lange (2) found that they became more [illegible] when their husbands were away. Some [illegible] interviewed said they felt little suppor[illegible] and relatives and none from the militar[illegible] supposed to be good waiting wives an[illegible] plain. When the father is transferred from [illegible] family must vacate its military housing, [illegible] and familiar places behind. The mothe[illegible] must depend on themselves, not only [illegible] existence but also for emotional suppo[illegible] the mothers of patients seen in our clinic [illegible] children to sleep with them when the fat[illegible] Others moved back into their parents' [illegible] they often encountered further stress.

Another interesting aspect of the pre[illegible] absence is that the mother quite natural[illegible] role of go-between (11). When the fathe[illegible] mother controls communication betwee[illegible] rest of the family. Some women discov[illegible] powerful position and may exaggerate o[illegible] ments or incidents that may affect the [illegible] tudes toward each other. Some contin[illegible] when the father returns.

When he returns, the father has [illegible] breaking back into the family. Many fat[illegible] cessful, become frustrated, and feel reje[illegible] dency is to repeat the cycle and ave[illegible] which only reinforces the feelings of re[illegible] sides. Many fathers secretly request t[illegible] tours and then act surprised when the[illegible] Over a period of time the family bec[illegible] tered, much like the Puerto Rican fa[illegible] York and San Juan in which the fathe[illegible] ence in the family than the children an[illegible] rary visits are only tolerated (12).

Transiency

Another problem the military family [illegible] that of the many moves it must make [illegible] ther's career—sometimes these moves [illegible] as every two years. Relationships exte[illegible] ily become shallow because there is not [illegible] develop them. The mistrust that the [illegible] feels in his job is carried to the home [illegible] their family never to disclose family [illegible]

lines of communication are down in the family, each member might find himself or herself isolated.

A kind of gypsy phenomenon is also commonly found. Military people are seen by the nearby community as transients and are often targets of mistrust and hostility. Not only does this further isolate the family and cause them to stay within the confines of the base, it presents a difficult situation for the children attending public schools. They have to break into peer groups repeatedly as the "new kid" and are often the school's scapegoats. To the military child, this kind of attention is much better than none, and he or she often begins to act out the image others have of him or her.

Methods of Parenting

The parents who came to our clinic used three methods of dealing with their children—authoritarian, democratic, and a mixed, inconsistent way. Some of the parents who felt they were democratic were actually authoritarian or inconsistent when examined. As one might expect, the greatest number of behavioral disorders, nearly 93%, came from the authoritarian families. The fathers in authoritarian families came primarily from the ranks of the enlisted, were not college educated, and were considering a career in the service. They often came from autocratic homes themselves and found the military comfortable for that reason. Often, the only model of dealing with children they knew was what they learned through their own "mortification" process. When this process is applied to a child, especially one in the adolescent years, there is bound to be difficulty.

Scapegoating

A process that is seen disturbingly often in the military is scapegoating. Since confrontation is practically prohibited and conflict is channeled through the chain of command, scapegoating is inevitable. The supervisor receives a great deal of pressure to keep his men compliant. When problems arise, as they always will in such a system, it is easier to single out someone as the cause than to examine the system. The scapegoat frequently does a great deal to contribute to this process. This model is often carried home to the family. A father who feels worried about his career may focus on one family member as the cause of family conflict.

Husband-Wife Relationship

The military husband and wife, like other couples, begin their married life with fond expectations. The husband is seen as strong and independent, and the wife is often seen as weak and dependent. Women who marry men in the military find their machismo appealing, but they are disappointed to find that their husbands are compliant, that they are tightly bound to the military and unsupportive of their needs. When the husband is assigned to duty away from home, the wife has to take over his family functions. The wife becomes more independent in this process, but the manner in which it occurs causes her problems because she feels rejected. Many times, after even one prolonged separation, an estrangement results. On the other hand, when the man returns home to find an independent woman rather than the dependent wife he left, his need to feel strong is threatened and the family system is shaken. Interestingly, some couples may remember past reunions that were pleasant; during times of family stress they will ask for a separation thinking it will solve their problems. Actually, the problems only worsen.

Treatment and Resistance

The therapist who elects to treat a military family or one of its members should keep several things in mind. The strong coalition the father has with the military probably presented the most difficult problem I had to deal with in the families who came to the clinic. Obviously, this coalition cannot be broken unless the father leaves the military or the military changes its style of relating to its members. In therapy I tried to help other family members understand this coalition and reduce their expectations of the father.

Another problem is the fact that military families are transient. The father might be reassigned in the middle of therapy. Because of this, I modified the therapeutic approach and usually saw a family for a short course of several intensive sessions aimed at uncovering conflict and helping them to understand the system. I found multiple impact therapy (13) to be a useful technique because of its effectiveness in a short period of time.

The military therapist must also be aware of the fact that the military, the local community, and the family are constantly trying to use the scapegoating process. The therapist who agrees to see the identified patient in individual therapy must consider the possibility that he or she might be assisting in scapegoating the patient. Because of this and because of the rather rigid bureaucracy within which the military therapist must operate, I have wondered if military therapists should see these families at all. Military therapists are subject to the same devices the system uses to enforce compliance in other system members, thus they might be in a rather rigid bind if they view themselves as advocates for the family. What often happens, because of pressure from the system, is that the therapist remains an advocate of the military and the scapegoating process continues.

Resistance to seeing a psychiatrist is understandably prevalent among military families. Officers seem to be more reluctant to involve themselves than enlisted men, except for those with more investment in the system. Only 6% of the cases reviewed were from the families of officers on active duty, and fewer actually became involved in the recommended treatment. Almost all of the officers seen expressed a concern for the privacy of their records. Some of their concern reflects the general paranoid atmosphere, but some of it is based in reality; their records are often available for review.

CONCLUSIONS

The data on the 792 patients seen in this military clinic were compared with those from a civilian clinic, the Wichita Falls Mental Health-Mental Retardation Center in Wichita Falls, Tex. The comparison showed that the diagnosis of behavior disorders was made more frequently in the military clinic. Bloom (7) reported a similar finding. Other authors (1–6) have tried to explain the problems that occur in military families by describing intrapsychic defects resulting from such situations as father absenteeism. After working with several hundred cases of behavior disorders, I came to feel that an explanation based on an intrapsychic model is inadequate. Behavior does not have to be a function of individual defect but may express attitudes developed in pathological systems.

It is possible, but unlikely, that the military clinic sees more patients because its facilities are more available. Compared with a population in Great Britain in which the economic availability of services was similar (14), the incidence of behavior disorders in the case records reviewed here was still much higher. In addition, compared with the estimate that 10% to 12% of the general population of children in the United States have emotional problems and need treatment (15), the experience in this military clinic of actually evaluating almost 12% of the families on the base over a two-year period is notable. Other factors, such as geographic availability and educational level of the population, must be considered, but it is questionable how really important they become in a paranoid system with so much resistance toward psychiatric involvement.

When military families seek help, the total system from which they come must be considered, and the therapist must also be aware of the system in which he or she operates. In military families the father is strongly joined with the military and becomes its agent within the family. The rest of the family withdraw from him partly out of necessity and partly out of anger, and there are feelings of rejection on both sides. The father relies more and more on military support and the family on themselves, and the system spirals ever onward. Johnson and Szurek (16) described a process they termed "acting out," and I feel this is a helpful way of viewing these families. In an attempt to get the father back into the family, the mother might exaggerate or distort a child's behavior and might covertly or even overtly encourage delinquent behavior. The children, many of whom feel alienated by the rigid class structure of the military, pressure from the community to act out, resentment of the father's autocratic methods of dealing with them, and the same anger and powerlessness their parents feel, actively respond to the family pressure to act out.

That there are problems within military families is without question. The incidence of alcoholism is high (17). The incidence of child abuse is five times higher than the national rate (18). When confronted with such information, the military becomes defensive and moves slowly, if at all. There have been [illegible] made to humanize the system, but [illegible] change with the leadership or may become [illegible] in the morass of the bureaucracy.

I am pessimistic about change, but this [illegible] done at an administrative level to help [illegible] family. Obviously, at this point the autocratic [illegible] of the system will probably not change [illegible] will continue to be tightly joined with the [illegible] the dismay of their families. Educating them [illegible] to what to expect from the system and from [illegible] early detection and referral of problems [illegible] classes, confidentiality of records, stability [illegible] duty, and reintegrating family sessions when [illegible] returns from an isolated tour would all [illegible].

The military has certainly had precedents [illegible] ideas; it has race relations sessions, civilian [illegible] mittees, and drug and alcohol abuse programs. [illegible] programs like these would not only help [illegible] many of the sources of family pathology [illegible] good long-run economic sense for the military.

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Developmental Issues in the Psychiatric Hospitalization of Children

BY IRVING N. BERLIN, M.D.

Rather than becoming repositories for the community's seriously disturbed children, children's psychiatric hospitals need to develop milieu programs that involve trained child care staff and developmentally oriented child mental health professionals. Treatment programs should address the developmental needs and abilities of the various age groups and the particular developmental deficits reflected in their psychopathologies and should include a variety of treatment elements.

MANY CHILDREN and young adolescents are hospitalized for brief or extended periods because the severity of their illness makes it difficult for community agencies or the children's parents to contain them at home. The most frequent cause of hospitalization is psychotic withdrawn or aggressive hostile behavior that is not amenable to any other community resource. In many instances, hospital care itself provides little therapeutic intervention; however, it removes the child from the community, reducing stress on parents, schools, etc., and provides chemical and physical restraints for the child's difficult behavior.

A small percentage of youngsters are hospitalized because they are actually dangerous to themselves or others. Many preadolescents and adolescents who attempt suicide are hospitalized because a period of hospital evaluation is necessary, and, rarely, youngsters are so hostile and assaultive without apparent cause that the hospital is the only place for them. Children who have committed murder or arson need to be studied in the hospital.

Another reason for psychiatric hospitalization is a serious psychosomatic disorder such as anorexia nervosa. Often psychiatric hospitalization of youngsters with these disorders is no more effective than previous hospitalization in medical or pediatric services because critical developmental and intrafamilial data are not obtained, analyzed, and used to provide a new approach to the illness (1, 2).

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TREATMENT OF SERIOUSLY DISTURBED CHILDREN

Many psychiatric hospitals are not staffed by developmentally trained child mental health professionals. It is still rare for a mental hospital that treats children to have a well-trained child psychiatrist, child psychiatric nurse, social worker, occupational therapist, or child psychologist. The staff is rarely trained in either child development or the design of milieu programs as part of treatment. Therefore, sedation, antipsychotic drugs, isolation, and inactivity are often used, in addition to a haphazardly designed school program. In a few months the ward staff are conditioned by the child's behavior to interact with the child for the purpose of reducing disturbing behavior. Looking beyond behavior to try to understand its antecedents and causes is difficult with untrained staff. Examination of staff participation in precipitating disturbed behavior and finding methods of helping staff are unlikely on a busy, understaffed ward (3–5).

A few hospitals have used various forms of behavior modification programs to help with behavior problems. At first the reward system for positive behavior seems encouraging, but over time, unless specific target behaviors and specific contingencies to be reinforced are selected and changed as the child progresses, this potentially useful method loses its hold as the novelty wears off and no precise, continued use is implemented.

The patient who is very difficult to manage can now be dealt with by chemical restraints (6, 7). Children who appear less "crazy" but whose behavior is assaultive and destructive, often with histories of antisocial behavior coupled with brain dysfunction early in life, may be more difficult to treat with drugs and may require more individual care even though they are usually isolated (8, 9). The very withdrawn children receive little stimulation from ward personnel. It appears that in many inpatient children's settings the program is designed for staff comfort, since the strategies and techniques to promote patient improvement may not be very well defined (10–13).

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MILIEU THERAPY FOR HOSPITAL CARE OF CHILDREN

In the last decade, the milieu program has become a very important part of the hospital care of children (14, 15). A milieu program involves all of the child care staff and specialists in child psychiatric nursing, a teacher, an occupational therapist, a physical therapist, child therapists (any of the mental health disciplines with special training), and a child psychiatrist (16). They plan each day's activities, which are geared to the developmental needs and abilities of particular age groups of children who have particular developmental deficits in cognitive, sensory-motor, social, or interpersonal areas that are reflected in their psychopathology. Precise developmental needs of specific children should be assessed and addressed in the program (17). One of the critical issues in the milieu program is that the program is paced to provide a variety of treatment elements without any great gaps of time that leave the child isolated and with time for self-absorption and psychotic thinking and behavior. The pacing of the milieu program is critical; program elements that require concentration, such as school or occupational therapy exercises (each of which is geared to the child's developmental level and his or her need to succeed at the next developmental level) are alternated with large-muscle activities such as dance therapy or other physical activities. These activities use the range of sensory-motor modalities and are geared to the child's developmental needs for acquiring small and large motor skills. Learning requires the same developmental planning. Such pacing and alternation of program elements throughout the day insures the child's psychological attention and involvement in each of the scheduled activities. Thus, play therapy on an individual basis should not follow a large-muscle exercise that leaves the child tired and excited and not able to become involved with the therapist (18).

Conversely, group activities may be so planned as to move from large-muscle activities in which the group is involved in cooperative building or cooperative design of a large mural, for instance, to more sedentary talking about feelings or role-modeling, and, later, to playing out of feelings by the child. The milieu efforts are designed not only to help the child with developmental deficits but to enhance his or her new and emerging capacities and promote a sense of mastery in each of the activities in which he or she is involved (19–21).

A milieu program must begin with attention to the elements of self-care, with particular attention to helping with grooming, which may alter the child's self-concept. Thus, the simple tasks of making their beds and becoming responsible for their hygiene, grooming, and the care of their rooms, which are rewarded by special persons in the milieu, are opportunities to help youngsters learn that they are capable of doing things for themselves.

If used and monitored precisely, behavior modification programs, or token reward program[illegible] effectively in many of the elements of [illegible] help deal with a child's emotional and be[illegible] lems. The use of a variety of group the[illegible] ties as well as individual therapy is cr[illegible] ment in the child.

Role of Individual Relationships

The relationship of the child to an in[illegible] pist develops as they work together tw[illegible] week. The relationship helps the child t[illegible] verbalize the feelings that often under[illegible] therapist does not respond to the beh[illegible] adults have, i.e., with retaliation or w[illegible]

The therapist can use the relationship[illegible] ually a shift from assaultive or self-des[illegible] ior to pounding pegboard tools, hitting [illegible] etc. The child's increased capacity to e[illegible] sion without hurting or destroying in[illegible] has begun to learn to express feelings [illegible] As a result of not eliciting retaliatory [illegible] therapist or other adults, the child is [illegible] first time, able to understand his or he[illegible] dent feelings that bring about the ass[illegible] structive behavior. He or she gradually [illegible] them out through the use of fantasy [illegible] (18, 20, 22).

Use of Restraint

Nonpunitive restraint provides the ci[illegible] ence with an adult over a period of time[illegible] child to become attuned to the mus[illegible] which are antecedent to the discharge [illegible] tile, assaultive behavior. From non[illegible] straint, the child discovers that he or s[illegible] able to relax muscularly and to cont[illegible] and relaxation of his or her muscles. [illegible] child to use verbal cues to let the thera[illegible] milieu personnel, know that hostile [illegible] are emerging so that they can be transfe[illegible] destructive angry expression with sub[illegible] jects. Such continued efforts can lead t[illegible] of precipitating feelings and thoughts w[illegible] wise suddenly discharged in assaultive [illegible] havior (12, 18, 23, 24).

Occupational and Physical Therapy

The importance of developmentally [illegible] pational and physical therapy progra[illegible] care cannot be underestimated. A good [illegible] py program is geared toward helping c[illegible] their mastery over their bodies and th[illegible] The increased capacity to use thei[illegible] fectively, to become better coordinated [illegible] and to use their large muscles for run[illegible] and throwing brings a great deal of sat[illegible] pleasure is similar to the gradual, develo[illegible] tuned mastery over academic skills i[illegible]

sense of satisfaction comes not just from the praise of the adult but also from the child's sense of acquisition of skills. The capacity to use these skills in the milieu occurs frequently; thus, each interaction can reflect a new level of competency and a higher level of pleasurable exchange with adults (18, 20, 25, 26).

The occupational therapy program is critical. Since the skills of the occupational therapist in developmental diagnosis of perceptual motor functioning are often highly refined, such a therapist can give a very clear evaluation of each child's perceptual motor functioning and some of his or her cognitive areas. The occupational therapy tasks can be clearly designed to facilitate the next stages in perceptual-motor development. In addition, the use of textures and colors enhances the sensory capacities of the child considerably and enlarges his or her horizons in both sensory and motor areas. The fact that many of the projects in occupational therapy produce tangible results—e.g., clay figures or drawings or the products of weaving, woodwork, etc.—is extremely important to the child's sense of accomplishment (12, 18, 22, 26, 27).

ESSENTIALS OF A DEVELOPMENTAL PROGRAM

The effectiveness of such a program depends on the clarity with which it is applied to the developmental state of the child. That is, in each developmental area the program must not demand more of the child than the child is capable of developmentally. On the other hand, it must not be geared to a level where the child is already successful, lest he or she be bored by the activity.

The educational program is a critical element of all milieu therapy. Such a program depends on a thorough, developmentally oriented educational evaluation that clearly defines the cognitive areas in which the child needs help as well as the areas of the child's successes. Areas of academic achievement can be the entree to areas in which the child needs to acquire competence (18, 25, 26, 28–32).

Group Therapy Programs

Group therapy programs must be geared to the developmental and actual age of the child. Activity groups are most effective for young children because they are learning social skills and developing coping capacities for play with a variety of age-mates. Children are involved in a variety of activities that include first those of parallel play and, later, planned activities of collaboration and competition which provide opportunities for interaction with other children. The exploration of feelings first noted and verbalized by adults leads to greater expression of angry or depressed feelings, and, as collaboration in projects occurs, increased social competence develops (16).

With the older child, the activity programs may be more sophisticated in that they may be collaborative projects which lend themselves to the expression of feelings. For example, the painting of murals may have various subjective meanings to each child as he or she depicts a family, family outings, and various other activities, as well as pictures of the child in school and in various phases of living. In such an activity in one milieu setting, murals that described "the way I get in trouble" produced in painting some of the most vivid and meaningful expressions of feelings, which provided subject matter for many discussions (6, 11, 26, 33–35).

Work with Adolescents

Whereas most younger and older adolescents will not talk freely about their own problems, they will, in the course of learning about child development and actually working with younger children in the hospital or nearby day care or mental retardation programs, begin to talk about parenting. As they talk about the problems they see in younger children, they can be helped to speculate about how these problems may result from certain parent-child interactions. Later they may reflect about similar situations in their own home and how they may have been affected.

We have found that dramatic play initiated by talented group therapists is one way of getting young adolescents to play out their feelings about some of their problems. Usually they are asked to write a scenario about the problems of another child and play those out, rather than their own problems, as a basis for discussion.

It must also be stressed that individual psychotherapy with adolescents provides an opportunity for them to form a relationship with one person over time. Such a relationship may become a critical factor in the socialization process, in the reduction of conflict, and in the move toward egosynthesis (12, 18, 23, 24, 28, 33).

Developmental Orientation in Individual Therapy

Individual therapy must be developmentally oriented and include, especially, a developmental diagnosis of ego functioning. It is necessary to understand the psychopathology of the child and the deficits in nurturance that the child has already experienced. The child's specific problems in interacting with meaningful adults must be understood so the therapeutic contacts can be restitutive. Although interpretive psychotherapy may not occur, what may develop is a relationship that permits the child to identify with an adult who behaves therapeutically toward him or her. The adolescent finds an adult who is willing to understand his or her behavior and to help look at that behavior in the context of the immediate relationship. The goal with children and adolescents is to help them alter their previous modes of relating to adults and peers, which can be noted in interactions with other staff members and fellow patients. It becomes clear that unless such interpersonal relationships are altered, the child will never be able to relate meaningfully to other human beings. Thus, all relationships may remain sterile and manipulative without warmth or intimacy. For

more behaviorally disorganized children, as with other disturbed children, the intensity of the one-to-one interpersonal relationship provides a vehicle for focusing on specific conflicts, which are beginning to be reduced in the milieu therapy program, and working through them in the context of play, mutual activities, and talking, as well as in collaborative work with parents.

Collaborative Work with Parents

Most inpatient settings do not include parents in therapeutic work. Unless parents are involved in some collaborative efforts the inpatient treatment may not be effective in returning the child to the community and home. The work of Szurek and Johnson (4, 18, 26, 41) and Schopler and Reichler (36) indicated that parents' involvement is critical to the improvement of very disturbed children (37, 38). I have observed that it is possible to elicit collaboration with even the most disturbed parents on behalf of the child. Members of the treatment team must be willing to visit the home, to see the child in his or her natural habitat, and to help the parents with those behaviors which make living with the child difficult. Gradually, parents who are helped to understand and to work on family problems will learn to manage the behavior of the child at home and make it likely that day hospital or outpatient treatment will become possible.

In my experience, parents' attendance and participation in the inpatient setting several times a week is important. Parents may work first with other children who have similar problems but with whom they do not have mutual problematic emotional involvement. Parents find they can be effective, then gradually learn how to work with their own child. We have used videotape and a "bug in the ear," an electronic hearing aid that permits the treatment team to tell the parent what kinds of interactions are likely to be most effective with the child during a particular interaction or specific project. All of these methods have been extremely helpful in collaborative work with parents. It has been my experience that one does not leave the parent and child alone simply to visit. The staff is most helpful to both the child and parent if the parent is engaged with the child in a specific cognitive or sensorymotor learning task, especially using games. These tasks can then be broken down into the elements necessary for effective performance and facilitated by example and by helping the parent learn what kinds of behavior on their part seem to encourage the child to perform a task or learn more effectively.

As parents learn to work with their children more effectively in the hospital setting, they also become better able to deal with them more effectively when they take them home. It is critical for fathers as well as mothers to be involved in such a program. Fathers who cannot come during the day are encouraged to come in the early or middle of the evening to participate in evening programs and work with their children at specific projects.

IMPLICATIONS OF DEVELOPMENTAL ASSESSMENT

It is almost impossible to make a pro[illegible] ment and do any outcome studies if the [illegible] tion has not been done developmentall[illegible] cognitive, social inter- and intrapersonal[illegible] motor areas have been evaluated, the [illegible] data help one to make a better psychop[illegible] agnosis (8, 21, 26, 30, 31, 35, 39, 40). [illegible] one can make prognostic statements fo[illegible] child. One can then develop the next st[illegible] gram that should help the child's total [illegible] Statements about prognosis, treatmen[illegible] possible, treatment outcomes within a t[illegible] mit one to evaluate the evaluation proce[illegible] to refine these steps with each new pa[illegible] comes possible to evaluate particula[illegible] milieu program with regard to a partic[illegible] velopment to determine which parts o[illegible] seem to be most effective in helping t[illegible] program can also be evaluated in the co[illegible] staff and settings are best able to fa[illegible] development. Individual treatment goa[illegible] assessed and evaluated (13).

The critical issues in a developmen[illegible] the spelling out of the incremental st[illegible] each developmental area to help the c[illegible] now have sufficient information and c[illegible] opment to attempt such a developm[illegible] and therapeutic approach. Thus, both [illegible] outcome can be evaluated more clear[illegible] very seriously disturbed children, and [illegible] ually learn to refine our assessment [illegible] treatment skills.

SUMMARY AND CONCLUSIONS

As inpatient settings become more [illegible] velopmental efforts, they can more clea[illegible] areas of expertise and their capacity fo[illegible] dren, rather than becoming repositori[illegible] munity's difficult children, staffed by i[illegible] feel hopeless about understanding th[illegible] culties and helping them therapeutically[illegible] velopmental approach one must hire o[illegible] can train others in developmental dia[illegible] ment. Where this approach is used [illegible] slowly, it is a potentially effective wa[illegible] seriously disturbed children.

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OPINION AND COMMENT

The American Psychiatric Association and the Food and Drug Administration: An Analysis and Proposal for Action

BY RICHARD DORSEY, M.D., M.B.A.

The relationship between medicine and society is currently undergoing substantial change. While the Food and Drug Administration has no legal authority over the practicing physician, its decisions already influence medical practice. The Drug Regulation Reform Act of 1978, if passed in its present form, would grant FDA broad new powers over patient care and clinical research. In order to retain a high degree of control of its future and activities, the American Psychiatric Association must develop an effective mechanism for meeting this challenge and for serving the interests of its members and their patients. Dr. Alexander M. Schmidt comments on the author's presentation.

THE AMERICAN Psychiatric Association (APA) has recently taken several significant steps toward more effectively representing psychiatrists and their patients. Government regulation of the prescribing of medication, which is likely to be the next focus for attempted control over medical practice, was addressed by Dr. Daniel X. Freedman's testimony on H.R. 12,391 (1).

In addition, *Psychiatric News* obtained and published excerpts from a transcript of the Psychopharmacological Agents Advisory Committee review of labeling for lithium carbonate (2), which, one hopes, presages direct coverage of committee m[illegible] ly all of which are now open to the p[illegible] these steps, there is a critical need for a[illegible] tation of our views on the availability, a[illegible] motion, and proper use of psychotropic [illegible] major problem areas offer immediate cl[illegible]

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THE FDA'S PROBLEMS

Role

As with many regulatory agencies, the [illegible] of the Food and Drug Administration (F[illegible] what ambiguous. This leads to internal [illegible] decision making and external criticism [illegible] constituencies, each of which believes [illegible] priorities should be the same as its own. [illegible] protection" sounds clear until one ques[illegible] consumers are to be protected from drug[illegible] Many healthy Washington health officia[illegible] cates lean toward the former. However, [illegible] controversy suggests that the general pu[illegible] larly those individuals suffering from ser[illegible] are more concerned with the availability [illegible] than with the documentation of safety a[illegible] the satisfaction of "guardians of the co[illegible] est" (3, 4).

In addition, FDA deals with broad [illegible] questions in deciding on the overall risks [illegible] of the availability or promotion of part[illegible] However, the physician who prescribes [illegible] whose marketing FDA must approve treat[illegible] dividually, rather than in the aggregate. [illegible] may seem an inordinate risk for a large pop[illegible]

Papers in the Opinion and Comment section present pro and con statements and discussion of issue[illegible] psychiatry.

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be highly appropriate for a particular individual or subgroup. Further, practitioners, academicians, and medical administrators may differ in their assessment of risk-benefit ratios even for identified populations or individual patients.

Finally, FDA is not a monolithic entity. Some staff members advocate expansion beyond the regulation of the pharmaceutical industry into the provision of drug information to patients (through the "patient package insert" [5]), greater control of drug use as well as drug labeling, and greater agency discretion in evaluating the relative merits of drugs in a particular class. Other FDA personnel feel current agency responsibilities provide more than sufficient challenge and workload and would prefer to concentrate on improving their capacity to deal with the problems at hand.

Complexity of Decisions

Few physicians outside industry and government appreciate the number and complexity of considerations involved in decisions regarding drug regulation. The scientific issues are difficult enough, since data are often incomplete and commonly subject to varying interpretations even by unbiased experts. In addition, the law requires the application of different standards in decision making for different purposes, being most stringent in requirements for the approval of new drugs or new indications and most lax (frequently to the point of scientific invalidity) with respect to adverse effects,

Although good science constitutes the beginning of the drug regulatory process, it is by no means the end. All decisions must be made within the FDA administrative framework; this fact alone imposes a certain amount of delay and costs both the government and the manufacturer time and money. Beyond this point, FDA's decisions are subject to judicial and political review, both of which consider scientific evidence among a number of factors. However, in neither instance do scientific and clinical considerations assume the centrality to which physicians are accustomed in patient care and professional journals.

THE AGENCY'S STAFF

In part because of real recruiting difficulties and in part because of conflicts over agency goals and performance, the quality of FDA staff has frequently been criticized. For example, Senator Edward Kennedy observed in a speech at Tulane University in November 1975,

> It is a fact of life that only top-notch scientists can correctly judge the scientific work of other scientists. By and large, although there are many able scientists in FDA, it has failed to attract and keep the top level scientific talent that it needs.
>
> The consequences are serious. Too often, FDA yields to the temptation to use precaution and delay as substitutes for expertise and scientific judgment. Again, the public pays the price. Badly needed drugs are delayed from joining the fight against disease—not because they are dangerous; not because they are unsafe; but because of the FDA's well-deserved inferiority complex about its scientific judgment.

While this statement reflects some political hyperbole, it is true that the FDA Division of Neuropharmacological Drug Products has not been headed by a psychiatrist since 1974, in contrast to the National Institute of Mental Health, whose staff includes a number of world-famous psychopharmacologists. During most of 1976 the directorship of the division remained vacant, and a neurologist currently serves as the director.

The nature of regulation, with its emphasis on reviewing the work of others, adversary relationship with various groups, and vulnerability to political and legal considerations, rarely appeals to outstanding scientists. The intellectual freedom, professional prestige, and insulation from worldly pressures characteristic of academia or the National Institutes of Health constitute incomparably more agreeable working conditions for most creative researchers.

However, scientific creativity may be less important in the discharge of current FDA responsibilities than such attributes as fairness, diligence, appreciation of the regulatory procedure, and the ability to live with persistent criticism. The staff of the Neuropharmacology Division are generally experienced, fair, and familiar with scientific and regulatory issues. In recent years they have made considerable progress toward facilitating the review of submissions and clarifying the scientific issues presented to their advisory committees.

In the current political climate only scientists of high professional stature, adventuresome temperament, and assured alternative employment are likely to take the calculated risks involved in substantially shortening the time required to review new drugs. Without such individuals in FDA, the outlook is for continued refinement of the basic approach of cautious fairness. Such an approach may be adequate, although not optimal, to discharge present agency responsibilities. However, it will fall far short of the pace and flexibility required if FDA's authority is expanded in ways now under active consideration.

ADVISORY COMMITTEES

The approach frequently proposed to strengthen FDA's internal scientific talent is reliance on advisory committees composed of leading outside experts, usually from academia or the National Institutes of Health. The immediate problem is that agency staff are still legally responsible for all decisions, even those made on the advice of outside consultants. Thus the conservative bias of the system remains, although it is leavened by external views and support.

Advisors (some of whom are nonphysicians) are selected on the basis of their expertise in research, rather than their skill as clinicians or their knowledge of usual medical practice throughout the country. Accordingly, they must sometimes venture beyond their depth in deciding, often in an atmosphere of apparent political urgency, what labeling changes to recommend to appropriately inform and influence the practicing physician. For guidance on regulatory and administrative affairs, the advisors must generally defer to FDA staff, some of whom may also have limited clinical knowledge and experience.

Another major problem for advisory committees is time. Most advisors have numerous other commitments and are not able or expected to devote the time necessary to achieve mastery of the regulatory issues to be resolved or to work continuously with FDA to assure that satisfactory decisions are reached and implemented.

THE PHARMACEUTICAL INDUSTRY

Unfortunately, many of FDA's staffing problems are not unique to government but appear in the pharmaceutical industry as well. The need for initiative and scientific creativity is probably greater in industry than at FDA, since drug companies generally conceive, finance, and monitor the research studies forming the basis of submission that FDA reviews.

Relatively few of industry's psychopharmacology research programs are directed by psychiatrists, and a number of corporations with significant research commitments to the central nervous system area have no psychiatrists at all on their research staffs. Assignment of monitoring responsibility for psychotropic drug trials to physicians who lack formal psychiatric training often leads to limitations in study design and management, difficulties in communication between industry physicians and investigators, and the estrangement of industry from academic psychiatry. This reality reinforces FDA's emphasis on documentation and its skeptical attitude toward industry submissions. The net result is the expenditure on both sides of substantial amounts of time, energy, and resources that could otherwise be devoted to more socially productive research. It also means that the introduction of effective new compounds may be delayed or even abandoned because they do not meet the high U.S. standard of scientific evidence.

ISSUES OF DRUG REGULATION

Several major issues surrounding drug regulation require clarification. Despite court decisions, professional testimony, scientific publications, and statements by FDA commissioners, widespread misunderstanding on these points still unnecessarily clouds public debate and medical practice.

The FDA's Authority

The FDA's control over the pharm[illegible]try derives from the federal govern[illegible]tional power to regulate interstate co[illegible] agency currently has no statutory a[illegible] practicing physician (6, 7). Although [illegible]gally unfettered by FDA in prescribi[illegible] his or her individual patients, FDA h[illegible] direct influence over the practice of [illegible]

First, drug labeling, particularly as [illegible] *Physicians' Desk Reference* (8), cons[illegible] source of prescribing information. [illegible] which must be approved by FDA, a[illegible] pharmaceutical companies may say in t[illegible] and detailing, with the goal of assuring [illegible] sonable scientific accuracy and fairness [illegible] promotional communications.

Unfortunately, many physician[illegible] estimate the scientific validity of [illegible] feel obliged to prescribe in accordan[illegible] mendations in the package insert. [illegible] courts may mistake labeling for a def[illegible] treatise rather than a regulatory docu[illegible] undue weight in defining a standard of [illegible] fessional liability cases. Thus even [illegible] has had considerable experience wi[illegible] inclined to conform to the recomm[illegible] package insert in prescribing rather [illegible] her own professional judgment.

Finally, many public programs h[illegible] beling to directly constrain presc[illegible] reimbursement to drugs given for [illegible] dications." For example, a military d[illegible] ceive methylphenidate at no charge f[illegible] of childhood hyperkinesis, but not [illegible] the effects of tricyclic antidepressa[illegible] Similarly, the question of Medicare [illegible] treatment of multiple sclerosis with A[illegible] the absence of agency "approval" c[illegible] was formally raised at a recent m[illegible] Neurological Drugs Advisory Commi[illegible] all, some state mental health commis[illegible] their own initiative or under court o[illegible] their staff physicians to prescribe [illegible] doses of psychotropic drugs despite [illegible] are often inadequate, particularly [illegible] in public hospitals.

Labeling

Drug labeling constitutes "all labe[illegible] ten, printed, or graphic matter (1) u[illegible] any of its containers or wrappers, or [illegible] such article" (10); this definition has [illegible] judicial and regulatory decisions to re[illegible] tising and detailing. As such, it cons[illegible] cense to promote" and a constraint [illegible] said by the manufacturer. On the the[illegible] turers will tend to overstate the benefi[illegible] the risks of their products, the regula[illegible]

intentionally biased in a strongly conservative direction in the hope that on balance a reasonably accurate presentation will emerge.

Changes in labeling to expand indications or increase dosage must generally be initiated by the manufacturer's submitting a request and supporting documentation. Since such submissions are quite expensive, particularly if original research is required, manufacturers are reluctant to commit the necessary time and money unless a commercial benefit is anticipated. For example, labeled dosage ceilings for most tricyclic antidepressant and antipsychotic drugs are inappropriately low in light of current information, not because psychiatrists, manufacturers, or the FDA do not know any better, but because there is no real incentive for manufacturers or the FDA to change the status quo.

Thus labeling is at least as often obsolescent as authoritative. For example, labeling for diazepam (Valium) included a warning against its use with epileptic patients at a time when it was generally regarded as the drug of choice for status epilepticus. The value of imipramine hydrochloride (Trofranil) for childhood enuresis was recognized for nearly a decade before the labeling was changed. With the exception of haloperidol (Haldol), the maximum recommended doses for most of the antipsychotic drugs are far below those generally recognized as appropriate for significant numbers of patients. Labeling for most tricyclic antidepressants contains no information regarding the correlation between plasma levels and clinical response or the need for daily doses well above 300 mg for selected patients who are rapid metabolizers of these drugs (11).

Introduction of New Drugs

Lasagna and Wardell have been among the leaders in stressing the decline in the introduction of new chemical entities since the Food, Drug, and Cosmetic Act was amended in 1962 to require proof of efficacy as well as of safety. They have also pointed out the delay in the introduction of new drugs to the U.S. market compared with Western European countries having a high standard of medical practice; they coined the term "drug lag" for this phenomenon (12, 13).

Not surprisingly, one result of the stringent U.S. regulations has been a shift of basic drug research to other countries by large, multinational pharmaceutical companies (14). Such a development threatens American leadership in medicine and science, reduces the opportunity to study promising new compounds in their early phases, and weakens this country's basic research structure by exporting jobs and contracts.

NEW LEGISLATION

Background

The Drug Regulation Reform Act of 1978 (S. 2755, H.R. 11611), which is currently moving through Congress with the support of the Administration and key legislative leaders, will provide FDA with breathtaking new powers over the medical profession. Despite the fact that a number of the Act's major provisions, including those summarized below, will be intensely objectionable to most psychiatrists, APA has not yet taken a position on this legislation, thus leaving the burden of opposition to AMA and a few individual physicians.

Approval of New Drugs

The Administration has, in essence, taken the position that although there is no "drug lag," new legislation is needed to eliminate it. The basic rationale for the Act derives from its sponsors' contention that it will accelerate the development and introduction of new drugs. However, AMA, academic pharmacologists, the pharmaceutical industry, and even FDA's own career staff have testified that the actual result is more likely to be further delay.

Medical Practice

Under certain circumstances the offices of all physicians prescribing drugs released into "controlled distribution" would be transformed into "regulated premises," which FDA inspectors would be authorized to enter without a warrant on the suspicion that the physician might be prescribing these drugs in ways not approved by FDA. Inspectors would have wide discretion to inspect and copy the medical records of individual patients. FDA could also issue its own subpoena to compel a physician to appear in person anywhere in the United States and to produce whatever records and documents might be demanded. Hearings on alleged prescribing violations would be conducted, not by a court but by FDA, who officials would act as prosecutor, judge, and jury, with the power to assess fines up to $10,000. Only after the issuance of a final order by the Department of Health, Education, and Welfare would the physician have the right of appeal to the courts.

Thus even the most innocent physician, acting in good faith and according to sound professional judgment, would be exposed to the wholesale violation of his or her patients' confidentiality and to the complete disruption of his or her practice or other professional work. The economic cost of productive time lost and legal fees paid will be burdensome even for large corporations; for the individual physician they will be nothing short of ruinous.

The Administration acknowledges all of these possibilities but seeks to soothe opponents by claiming that these powers would be used only in exceptional circumstances. However, nothing in the history of FDA and of its parent agency, the Department of Health, Education, and Welfare, provides a rational basis for reliance on the self-restraint of government officials.

Patient Information

Despite the limited scientific evidence on the effects

of providing drug information to patients, the Act mandates the development of patient package inserts for most major classes of drugs and requires that the pharmacist dispense them with the prescribed medication. In most cases the physician can direct that a particular patient not receive the package insert, but as a practical matter the patients for whom this information may do more harm than good are likely to be people who will be even more upset by knowing that something is being withheld from them. Worse still, FDA will have the power to override the judgment of a physician for an individual patient and to require that *all* patients receive package inserts for certain drugs.

FDA will also have the power to require that physicians obtain written informed consent, in the precise form and manner dictated by government, as a condition of prescribing certain drugs. In addition, FDA will be able to mandate that physicians notify all patients of specific major hazards of particular drugs even if the physicians disagree with FDA's assessment of the risk, believe that notification would do individual patients more harm than good, or find that identifying and contacting large numbers of these patients would be extremely difficult and burdensome.

Research

At present FDA's authority over medical research is limited to studies sponsored by pharmaceutical companies. However, this Act would bring *all* drug research in humans under FDA control and would require prior FDA approval for the conduct of drug studies, under pain of heavy civil penalties. In addition to the obvious threat to academic freedom, this provision would have the bizarre result of requiring the most distinguished clinical investigators in the country to seek the approval of obscure FDA staffers whose principal qualification to act as arbiters of research consists of desks flanked by an American flag.

PROPOSED APA POLICY

Latitude for Physicians

In dealing with regulatory agencies generally, APA should adopt an explicit policy favoring maximum latitude for the individual physician, subject to appropriate review and controls by organized medicine and psychiatry. As applied to drug regulation, such a policy would have several specific implications.

First, APA should press for earlier availability of new drugs so that larger numbers of psychiatrists can use them and explore their role in the treatment of mental disorders. Prompt reporting of new drug uses and adverse effects in the *American Journal of Psychiatry* should be explicitly encouraged. APA should also provide vigorous public support when FDA does approve new psychotropic drugs or appropriate labeling revisions and not leave the agency to face hostile political and journalistic fire alone on these occasions.

APA should work with the American M ciation (AMA) and other medical specialty resolutely oppose any federal constraints marketed drugs by individual physicians vidual patients. Such a policy has adopted by AMA, whose House of Dele in 1975 that

> the American Medical Association contin and seek to modify any restrictive regulat ened to interfere with the exercise of a ph sional prerogatives in selecting the ther choice for his patients . . .[and] that the A Association continue to work for extensiv by practicing physicians in the review of e and development of FDA policy. (15)

APA should testify and lobby vigorous new legislation that would impose feder restrictions and should be prepared to challenging any such laws. It should AMA's example in providing formal FDA proposals published in the *Federa* nally, APA should press on the national els for the abandonment of any administ requiring physicians to prescribe in acc labeling or limiting reimbursement to d for "approved indications."

In exchange for the earlier introductio and continued broad prescribing freedo cooperate in an effective surveillanc mechanism. Lack of the former has b reason for delaying the introduction of the possibility of providing reasonable timely reports of adverse effects sho FDA's apprehensions in this regard dubious prescribing clearly does exist need to take corrective action to avoid would have a pernicious effect on the ov patient care in order to constrain poo small minority.

For both purposes, Professional St Organizations (PSROs) or other local ganizations should be advocated as mechanism. The PSRO program, to w government is already committed, was provide public accountability for m while assuring that "decisions on m made by other physicians rather thar tors or bureaucrats"(16).

Part of the quality assurance focus on prescribing, and adding a surveilla adverse effects of new or existing dru system should not be unduly difficult establishing such a system, the coope and the specialty societies might reason as in the AMA-Health, Education, and Model Screening Criteria Project (1 ment of Model Psychopharmacological teria by APA on contract to the Natio

Mental Health represents one significant step in this direction.[1]

In addition, APA should take the initiative to enhance psychiatrists' knowledge of psychopharmacology at all levels, from residency training through continuing education in practice. Increasing emphasis should be placed on the proper use of psychotropic drugs, their common interactions and adverse effects, and the appropriate assessment of therapeutic risks and benefits.

APA should also explicitly address the consumer protection issue as it relates to psychotropic drugs. Psychiatrists understand the need for proven efficacy, reasonable safety, and full disclosure of the effects of drugs, as well as the desirability of properly protecting research patients. However, many other groups emphasize these issues strongly and, on occasion, to excess (18). APA's greatest contribution to the drug regulatory dialogue may lie in emphasizing the need for greater balance between the risks of illness and the risks of treatment. The importance of preserving maximum latitude for the patient and his or her physician to select treatment on an individual basis should be stressed. Emphasis should also be placed on reviewing the substantive benefits and hazards faced by research patients, rather than on the precise composition of human research committees or the attempt to explicate and guard against every conceivable risk.

Similarly, while remaining alert to the high financial stakes involved in the pharmaceutical industry, psychiatrists should remind the public that profit in the private sector has no greater capacity to induce misrepresentation and deception than does power or ideology in the public sector. From our experience with state hospital systems and community mental health centers, we should, of all specialties, be most keenly aware that government control assures neither quality nor equity.

Finally, APA should press FDA to include a full disclosure statement on drug labeling informing the physician and other interested parties that the labeling is primarily a regulatory rather than a scientific or medical document. With particular regard to "indications," I have recommended elsewhere (19) that the following statement be included in the package insert in order to put "FDA approval" into proper perspective: "Based on evidence submitted to the Food and Drug Administration, the manufacturer has been authorized to recommend the following uses of this drug." Similarly, the "adverse effects" section should disclose that this list includes many phenomena that have been reported to occur in patients while taking the drug but for which a cause-and-effect relationship has not been established.

The AMA House of Delegates has made a similar recommendation, resolving in 1975

> that the AMA petition the FDA to adopt a standard paragraph in all package inserts to read substantially as follows: Most package inserts of necessity represent a summary of the information available about the drug upon which approval for sale was based. This should be regarded as a guide for the physician who must combine the best knowledge from all sources with individual patient considerations in deciding upon appropriate therapy. This should not be considered as a legal controlling influence over drug use by a given physician in the management of an individual patient. The package insert gives the parameters within which the pharmaceutical manufacturer may advertise or publicize this product. (15)

Benefits of Drugs

The second key element of APA policy should consist of emphasizing the proven value of drugs for the treatment of mental illness. APA also needs to assure wide dissemination of the evidence of the biochemical basis and genetic transmission of the more severe disorders (particularly schizophrenia and affective illness), of which even sophisticated laymen are often unaware, and to point out the limitations of alternative forms of treatment. Further, psychiatrists should note the additive effects of pharmacotherapy and psychotherapy for several major mental disorders (20–22) and emphasize the importance of the psychiatrist's medical knowledge in properly applying either or both modalities.

For maximum public policy appeal, APA should stress the cost-effectiveness of pharmacotherapy and its broad applicability and availability to most segments of the population. In addition, evidence of the beneficial effects of drug treatment for the more severe disorders, which are most common among the poor, elderly, and minorities, should be well-received by Congress.

APA ACTION

Commitment

The first step in addressing the above issues must be a decision by the APA Board of Trustees to pursue an aggressive policy concerning drug regulation. In essence, APA must make a commitment to work with FDA and its congressional oversight committees similar to that already undertaken to deal with insurers. The latter arrangement probably represents a good general model for APA's relationships with the financial, regulatory, and consumer groups likely to have substantial influence on the practice of psychiatry in the next decade.

To assure adequate coverage for psychiatric services under existing third-party mechanisms and under national health insurance, APA has been working for a number of years to develop good relations with government, Blue Cross and Blue Shield, and commercial insurers. Those involved have attempted to address the concerns of these groups and to educate them on

[1]These criteria will be published later this year. APA's role in developing these criteria is noted in the present context and in light of Dr. Schmidt's comments.

our view of clinical reality and public necessity. The Commission on Standards of Practice and Third-Party Payment was established to negotiate with insurers and to coordinate the work of other APA components, including the Council on Mental Health Services and the Committee on Financing Mental Health Care and the Committee on Peer Review. APA also established a field consultant program in peer review to strengthen this activity at the district branch level and to gather information about developments across the country to share with insurers and government at the national level.

Perhaps most importantly, the Association has established a clear and consistent policy of seeking parity in insurance coverage for mental illness with other medical disorders and in exchange offering equal or better accountability through peer review programs and cooperation with third parties. A similarly explicit commitment to goals for drug regulation will be required if APA is to be effective in this area.

Liaison

The Joint Commission on Government Relations and/or the Council on Research and Development should establish liaison with a number of organizations including FDA, AMA, the American College of Neuropharmacology (ACNP), and the Pharmaceutical Manufacturers' Association (PMA). With FDA we should articulate and press for the regulatory actions outlined above. With AMA and ACNP we should coordinate our positions and lobbying efforts. With PMA we should seek mutual understanding and cooperation, exchange of information, and support for mutually acceptable positions.

Representation

Current FDA regulations (23) provide

> the mechanism for nominating and selecting nonvoting members of standing technical advisory committees. In the past three years, the Commissioner has increasingly included such nonvoting members to represent consumers and industry on standing technical advisory committees. These members serve a liaison function with those whom they represent.

Because of their lack of a vote and their well-understood liaison role with a particular interest, these individuals meet the statutory requirement that "any financial interest they may have in the particular class which they represent does not constitute a disqualifying conflict of interest"(23).

The FDA Commissioner has formally noted that

> nonvoting consumer and industry liaison representatives have served on all of the OTC [over-the-counter] drug review advisory committees, the biologics review advisory committees, and the medical device classification panels, with enormous success. With rare exception, their conduct has been entirely dignified and proper, and they have made major contributions to the issues pe... these advisory committees. (23)

When the OTC panels were originally es... 1972, the Proprietary Association, repre... manufacturers of this class of medicat... initiative to arrange a meeting with the Ph... sioner to seek nonvoting representation on... The manufacturers were well received a... and over the ensuing six years have becom... that their involvement has contributed ... the public interest and to their own.[2]

The OTC advisory panels function in mu... way as do their prescription-drug cou... which decisions are usually reached by ... Under these circumstances a voice is wort... much as a vote. Thus APA should proc... delay to nominate a nonvoting represent... FDA Psychopharmacological Agents Adv... mittee. Technically, this individual would ... psychiatrists rather than APA as such (... views presented would be those of psychi... termined by the Association. The re... would have to be sophisticated in psy... cology and regulatory affairs and famil... sentiments and structure of APA. He o... be responsible for advocating APA's vi... matters coming before the advisory comm... advising APA of actions taken and c... Drawing on the expertise of the Council ... and Development when necessary, the re... should also be able to formulate a judgm... issues independently of both the agency ... visors.

Testimony

The APA Office of Government Relati... assigned the responsibility to closely mon... legislation affecting drug research or pres... notify the Council on Research and Deve... the Joint Commission on Government Rel... scheduled hearings on these subjects. ... groups should then work together to prep... ate testimony and secure witnesses of h... represent APA before Congress. Clear ge... as outlined above need to be establishe... vance so that testimony can focus on the ... particular legislation as they relate to ou... cerns.

Reporting

Psychiatric News should increase its ... congressional, FDA, and APA actions a... development and prescribing. At least one ... er should focus on this area in order to ... ficient sophistication to report and interp... ly. APA's representative to FDA should ... paring reports and analyses of agency a...

[2]Mersill A., personal communication, 1977.

committee actions. In the case of any rejections of new drug applications, our representative and the Council on Research and Development should report in the *Journal* whether they believe the rejection was due to intrinsic deficiencies in the drug, shortcomings of research design or conduct by the manufacturer, or undue stringency on the part of FDA.

CONCLUSIONS

We are passing through an era in which the relationship between medicine and society is undergoing substantial change. Whether our profession will retain a high degree of control over its own destiny and activities, subject to negotiation with government and other major societal groups, or will suffer regulatory smothering and financial starvation depends in large part on how effectively we organize and negotiate over the next few years.

Pharmacotherapy has revolutionized psychiatry, shedding light on the pathophysiology of several of the major mental disorders and offering treatment of demonstrable efficacy, cost effectiveness, and wide applicability. Control over the entry of new psychotropic drugs into the U.S. market rests with the Food and Drug Administration, which also must approve the labeling for existing medications. While the agency currently has no legal authority over the practicing physician, its decisions do influence medical practice. In addition, the Drug Regulation Reform Act of 1978 would revolutionize the relationship between the federal government and the medical profession by extending FDA's authority directly into patient care and independent drug research.

Accordingly, APA must develop an effective mechanism for meeting this regulatory threat and for serving the interests of its members and their patients. The Association's commitments and programs in the insurance and peer review fields constitute the best model for such an undertaking in pharmacotherapy.

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Am J Psychiatry 135:9, September 1978

Comment

BY ALEXANDER M. SCHMIDT, M.D.

AFTER I HAD been beaten, in my turn, by the grade-school bully, my father asked me whether my two best friends and I, fighting together, could give our assailant a thrashing. I said, "Sure," whereupon my father suggested that the three of us visit the bully and quietly and politely describe in detail what would happen if he so much as laid a finger on any one of the three of us in the future. It was thus that I learned the important social principle that organized individuals working together in common purpose can do what the same people cannot do alone.

It is this principle that Dr. Dorsey is talking about; his words are as important to psychiatrists as my father's were to me, and for the same reason. Dr. Dorsey is concerned that the medical profession in general, and psychiatrists in particular, are being bullied by regulations, regulators, and Congress. More important, he clearly, concisely, and, in my view, accurately describes practical corrective measures that APA could take. Dr. Dorsey's paper is an important and worthy challenge to the APA and one that ought to be accepted.

I agree with Dr. Dorsey's analysis of the many complex and controversial issues surrounding FDA and the drug industry. He is careful to present a balanced and fair view of the problems presented by, and suffered by, FDA and its staff; and he takes note of FDA's efforts to resolve at least some of those problems. His major thesis is that FDA could use some help; and again, he is correct.

The following scenario is being repeated all too often in Washington: a scientist, somewhere in the world, in a speech, paper, or press release, presents his or her conclusion that a particular drug is a carcinogen, a teratogen, or whatever. The press picks this up, recognizes the sensational news value of the story, checks only whether the scientist actually released it, and then reports the story widely. A congressional staff member, sensing the story's sensational news value to his or her employer, arranges a hearing into the matter. At the hearing carefully selected witnesses testify to the overuse and misuse of the drug, which FDA is called upon to explain. FDA is urged strongly to do something about the problem; there is another round of sensational press repor[illegible] tually, regulations are proposed.

During my tenure as Commissioner [illegible] Drugs, this sequence of events occurred [illegible] ening regularity and with distressing resu[illegible] seldom able to defend itself very well at [illegible] ing, and it should not have had to try. C[illegible] case of misuse of drugs, it should not ev[illegible] FDA that was called to account! But [illegible] again, while violence was being done to [illegible] and often to good scientists, FDA was a[illegible] ness table.

The problem in this scenario is not the [illegible] by a scientist of data suggesting a here[illegible] pected drug danger. Any scientist is o[illegible] port his or her results. The error is tha[illegible] port and the ensuing events were usua[illegible] those who should have been paying t[illegible] tion—the professional organizations c[illegible] the proper use of the drug in question.

At times the initial report resulted i[illegible] study, the data were improperly handle[illegible] could be countered by existing data fr[illegible] ies. Usually, unless FDA already knew [illegible] they went unreported in the press o[illegible] This was in large part true because whe[illegible] hearings on important drugs were held [illegible] professional organizations were unawa[illegible] ings—or if they were aware, they did n[illegible] to monitor the hearing, let alone testify [illegible] inaccurate reports from congressiona[illegible] rected by a properly indignant public s[illegible] prestigious professional organizatio[illegible] organization insist on testifying, even [illegible] advance of a hearing. Later, when FD[illegible] sions discussed with its advisory com[illegible] in question, professional organization[illegible] rarely be in attendance or contribute.

It is important to realize that Cor[illegible] FDA's role. In the long run FDA can [illegible] less than Congress tells it to do. And [illegible] process is conducted in hearings, com[illegible] and mark-up sessions, most of which a[illegible] bad legislation results from that proce[illegible] because those who could have made it [illegible] aloof or uncaring. Thus in a real sense [illegible] ting the regulation it deserves because [illegible] the legislative process.

FDA's present role is not ambiguou[illegible]

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tect the public from unsafe and/or ineffective drugs. In judging whether drugs are safe and effective, FDA needs all the help it can get, and the advisory committee system now in effect is the best way for FDA to get that help. The advisory committees function for the most part in open session, so that interested parties can know what is happening and can influence the proceedings. The proper function of the advisory committees is to answer *scientific* questions, sharply put by the agency; FDA must, and properly should, handle the legal and political questions. It is when the scientific basis of FDA's actions is shaky that the political pressures can become determinative. But even when scientific proof is lacking, a consensus of unbiased and expert scientists can suffice to protect against foolish or even dangerous interpretations of scientific matters by bureaucrats, politicians, and judges.

I can criticize Dr. Dorsey only for passing lightly over some of the problems owned by medicine and psychiatry. For example, he favors "maximum latitude for the individual physician" in prescribing drugs, "subject to appropriate review and controls by . . . medicine and psychiatry." That is all right, but who is going to review and control the prescribing habits of individual physicians—APA?

Dr. Dorsey calls for better reporting of adverse drug reactions. As malpractice insurance rates and court settlements have risen, the reporting of drug misadventures has fallen. No one has yet been able to devise a satisfactory reporting system for adverse drug reactions. If the earlier release of drugs is to depend on practicing physicians' more accurate and timely reporting of adverse drug reactions, then the drug approval process will not accelerate significantly.

However, these minor criticisms do not detract from the need for APA and its members—indeed, all professional associations and their members—to heed Dr. Dorsey's call. Doing what he suggests is the *only* way, short of dissolving Congress, that we can have a beneficial impact on a legislative process that results in oppressive regulatory schemes.

The Manfred S. Guttmacher Award

The American Psychiatric Association invites applications for its annual Manfred S. Guttmacher Award for 1979. The award is given for any outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or other work, including audiovisual presentations (films, etc.), submitted to or presented at any professional meeting or published during the period from November 31, 1977, to December 1, 1978. A plaque and an honorarium of $500 will be presented to the award recipient at the Convocation of Fellows at the 1979 APA annual meeting in Chicago, Ill. Please note that the award recipient is now required to give a lecture at the annual meeting.

Anyone who wishes may apply to receive the award by submitting five copies of the work as well as five abstracts to Carl P. Malmquist, M.D., Chairman, Guttmacher Award Board, 1700 18th Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but will not be returned.

The deadline for submissions is December 1, 1978, and any entry to be considered must be in Washington by that date.

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EDITORIAL

Lithium: Developments in Basic and Clinical Research

LITHIUM IS THE OLDEST of our modern psychotropic drugs. Its efficac treatment of mania was discovered by Cade in 1949, investigated more th by Schou and others in the 1950s and 1960s, and finally accepted for clinic by the Food and Drug Administration in 1970. Like most psychotropic was discovered accidentally or from the wrong hypothesis. Cade's hypothe uric acid was involved in the pathogenesis of mania led him to use lithium blood and tissue levels of this metabolite because lithium urate is sol hence could be excreted. He quickly disproved his own hypothesis, but h ical results about lithium's therapeutic efficacy have been widely co Today, most would agree that lithium is the most specific and, for the c for which it is indicated, the most effective psychotropic drug available. used with the most precision because serum levels are easy to obtain an used to keep the patient in the therapeutic range, avoiding toxic levels. W is done, more than 75% of patients with acute mania respond within 2 Lithium is prophylactic in most patients with bipolar II depression, mi both the manic and depressive phases of the illness. Its role in unipolar d is still uncertain.

There have been reports of lithium's effectiveness in conditions as d hyperactivity in children, premenstrual tension, schizophrenic illness, holism, and in eliminating the euphoria of drugs of abuse, but these stu yet to be done with the rigor and the large populations that have characte work on affective illness. Should they turn out to be positive, we wil reconsider whether there are components of affective illness in those co which clinically have not been considered to have them, or whether the mode of action of lithium transcends specific clinical diagnosis by alter fundamental property like neuronal membrane permeability to endoge ions and thus altering the uptake and release of those organic molecu influx and efflux are associated with ionic flow.

The mechanism of action of the lithium ion (Li^+) is by no means clear it seems certain that the ratio of intracellular to extracellular Li^+ is cruc this ratio is crucial for the endogenous cations sodium and potassium. much research on affective illness implicates disorders of neurotransm tion, a good deal of attention has been paid to the effect of Li^+ on these. decreased release and increased reuptake of norepinephrine, which i ent with its antimanic effects. However, it is less clear how the antidep and prophylactic effects in bipolar patients are mediated. Li^+ also alte lease, uptake, and synthesis of serotonin. It alters the transport of gluco acids, and other cations across cell membranes. Many synthesis and rel tions of the neuron are calcium-dependent and are modulated by magnes competes with these ions to alter these functions. The Li^+ ion at low co tions alters adenyl cyclase activity and cyclic AMP levels, which are bel

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be components of neurotransmitter receptors. These multiple actions of Li^+ are perhaps to be expected because the endogenous cations like Na^+, K^+, Ca^{++}, and Mg^{++} also have multiple actions. Research on the mechanism of action of lithium at this level is in the province of the basic scientist and will undoubtedly be unravelled with time.

Of greater significance to the clinician is the search for even more effective methods for using lithium therapeutically, for more precisely defining the conditions in which it is useful, and for understanding the biological defects of those illnesses in which it is effective. The three papers in this issue of the *Journal* from the laboratories of prominent investigators at the University of Pennsylvania, Columbia University, and the University of Chicago address these questions.

Since intracellular accumulation of Li^+ is clearly essential for its action and is not measured by plasma levels, several years ago the Pennsylvania group suggested that the erythrocyte concentration and the calculated ratio of RBC to plasma lithium (Li^+ ratio) might also be useful. Even more useful would be the intraneuronal Li^+ concentration, but this is not feasible in clinical research. In their early work, they found that the ratio (which is always less than 1 and usually averages about 0.4) showed considerable intersubject variance but is quite constant for any subject over time. This early work suggested that the Li^+ ratio was lower in lithium nonresponders than in responders, that it separated unipolar from bipolar depressives, and that it might be under genetic control. These observations stimulated controversy and considerable research in many laboratories. Typically, the questions asked are basic, clinical, or at the interface. At the basic level the questions relate to the mechanisms by which Li^+ enters the cell, leaves the cell, and by which an equilibrium is established between cell and plasma. At the clinical level, there are the questions of the significance of these cellular mechanisms for the pathogenesis of affective disorder and for the prediction of clinical response. At the interface, there is the question of whether in vitro tests can be developed that would offer better predictors than are now available, as well as more sensitive indications of appropriate dosage.

The Chicago group addresses the basic science problem and elegantly summarizes the evidence for the mechanisms by which an equilibrium is established between intracellular and extracellular Li^+. Li^+ enters the RBC by three mechanisms which can be chemically dissected in vitro. It moves passively from higher concentrations to lower, i.e., from the plasma to the cell. It uses the same energy-consuming Na^+-K^+ dependent ATPase pump that actively transports these ions in directions opposite to their concentrations, i.e., K^+ into the cell and Na^+ from the cell to the extracellular space, and it uses a Na^+-Na^+ exchange pathway that alters membrane permeability to sodium and thereby facilitates a stoichiometric exchange diffusion. The first pathway is chemically insensitive, the second is inhibited by ouabain, and the third by phloretin. Li^+ leaves the RBC by passive diffusion and also by a phloretin-sensitive Li^+-Na^+ facilitated exchange diffusion pathway. The steady-state distribution between RBC and plasma reflects a dynamic balance between the influx via the chemically insensitive diffusion pathway from the high plasma concentration to the lower RBC concentration and the efflux via the Li^+-Na^+ counterflow system which keeps the intracellular concentration of these ions below the extracellular concentration.

Interestingly, these researchers have found a manic-depressive patient in whom the phloretin-sensitive pathway is markedly diminished. Studies of this patient's family show that this seems to be a genetically transmitted autosomal dominant trait. Unfortunately, one cannot arrive at simple answers from this observation because there is not a one-to-one concordance between the trait and the manifestation of clinical manic-depressive illness: the patient's father had an identical defect but no recorded affective disorder, although he appears not quite psychologically normal. The Chicago group also finds some but not all manic-depres-

sive patients with a similar defect, suggesting a subgroup. This defect is not fo
in schizophrenic or in unipolar depressed patients.

The Pennsylvania group reports their continuing work, which is both basic
clinical. They agree with the Chicago group about the mechanisms involve
influx of Li^+ into cells and the method of efflux. They feel that the great ir
subject variance is related to variance in the efflux system. They continue to
the Li^+ ratio to be a predictor of beneficial effects of lithium for treatmen
prophylaxis, but their attempts to differentially diagnose unipolar from bi
patients by the ratio fail to reach statistical significance. They have develope
in vitro system in which the distribution of Li^+ between cells and the mediu
a .90 correlation with that found in vivo.

The Columbia group also agrees that the level of Li^+ in a cell is determ
primarily by its efflux rate and has also developed an in vitro system in v
RBCs are preloaded with Li^+ and the rate of efflux into an artificial mediu
directly measured. With this system they found no differences between pa
with affective disorders who were not on lithium and controls. However, the
find that chronic lithium administration lowers the efflux rate to a stable
significantly below the pretreatment state. Patients with lithium toxicity had l
even markedly lower.

These three papers are similar conceptually and methodologically to
found in the clinical pharmacology of internal medicine. They are sophistic
and difficult to read, but they show maturation in the research needed fo
understanding of the mode of action and optimum use of a very effective ps
tropic drug. Like all good research they raise many questions but offer me
for answering them, and they contribute simultaneously to basic physiology
cal pathogenesis, and therapeutics. It can safely be predicted that studies s
to these will continue with lithium and that the same principles of investig
will soon be applied to other psychotropic drugs and other illnesses.

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TOPICAL PAPERS: Lithium Transport—New Research Findings

Clinical Correlates of the Lithium Pump

BY DAVID L. DUNNER, M.D., HERBERT L. MELTZER, PH.D., AND RONALD R. FIEVE, M.D.

The active efflux of lithium from erythrocytes, expressed as the rate constant k_o, was studied in patients with primary affective disorder and in controls. The authors' data suggest that k_o is stable within individuals over time, is inhibited during treatment with lithium carbonate, and may be a good index of lithium toxicity. k_o correlates significantly with the lithium erythrocyte/plasma ratio but is seemingly not related to age, sex, or affective disease subtype. Preliminary data suggest that k_o may be determined, at least in part, by a genetic factor.

RECENT STUDIES from our and other laboratories suggest that lithium movement across cell membranes is governed by active transport processes—the lithium pump. It can be shown that lithium is actively transported out of human erythrocytes (1, 2). The influx and efflux rates of transport across erythrocyte membranes can be expressed as rate constants (k_i and k_o, respectively), which define the fraction transported per hour. These rate constants can be assessed in subjects who are taking lithium carbonate and in drug-free individuals. The k_i/k_o ratio is fractional, i.e., the active transport process for lithium efflux from erythrocytes is greater than that for influx.

In this paper our purpose is to discuss some of the clinical correlates of the lithium pump. We will focus on the relationship of pump activity to the lithium erythrocyte/plasma ratio (Li^+ ratio), the effect of lithium administration on lithium pump activity, and a possible role of the lithium pump in lithium toxicity and present some preliminary data on lithium pump activity in genetically related individuals.

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

Dr. Dunner is Psychiatrist, New York State Psychiatric Institute, 722 West 168th St., New York, N.Y. 10032, where Dr. Meltzer is Research Scientist V (Neurochemistry) and Dr. Fieve is Chief of Research, Lithium Clinic and Metabolic Unit. The authors are also with Columbia University, where Dr. Dunner is Associate Professor of Clinical Psychiatry, Dr. Meltzer is Assistant Professor of Biochemistry, and Dr. Fieve is Professor of Clinical Psychiatry.

This work was supported in part by Alcohol, Drug Abuse, and Mental Health Administration grant MH-21586 from the National Institute of Mental Health and by the Foundation for Depression and Manic Depression.

METHOD

Subjects in this study were outpatients of the New York State Psychiatric Institute Lithium Clinic or inpatients on the Metabolic Clinical Research Unit of the institute. All patients met the diagnostic criteria of Feighner and associates (3) for primary affective disorder and were further classified into bipolar and unipolar subgroups based on the criteria described by Fieve and Dunner (4). The relatives and spouses of patients were also studied, and psychiatric diagnoses for all individuals were established through a structured interview (5). All subjects gave informed consent to participate in these studies.

Heparanized blood for the study of lithium pump activity was obtained at various times and from patients in varying clinical states. For outpatients, most of whom were euthymic and were being maintained on lithium carbonate therapy, blood was obtained between 12 noon and 1 p.m., which was in most cases approximately 14 hours after the last dose of lithium carbonate. Mood state and other medications were

The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

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TABLE 1
The Lithium Pump: Mean Activity in Affective Disorder Subtypes

	Non-Lithium-Treated Group							Lithium-Treated Group				
		Age (years)		k_o		k_i/k_o			Age (years)		k_o	
Diagnosis	N	Mean	SD	Mean	SD	Mean	SD	N	Mean	SD	Mean	SD
Men												
Bipolar I	5	39.2	10.5	.243	.066	.19	.08	26	47.8	13.7	.181	.064
Bipolar II	3	59.7	17.2	.254	.031	.15	.01	7	54.6	15.3	.136	.051
Bipolar other	0							2	41.5	20.5	.261	.115
Unipolar	0							4	44.0	23.8	.206	.065
Controls	10	51.6	19.2	.264	.091	.18	.06	0				
Women												
Bipolar I	6	36.7	19.6	.254	.062	.16	.03	22	38.5	16.5	.178	.052
Bipolar II	2	34.0	17.0	.242	.125	.22	.09	12	58.3	10.1	.148	.036
Bipolar other	6	37.3	17.9	.214	.033	.21	.06	5	43.8	13.3	.144	.045
Unipolar	5	59.4	12.1	.255	.055	.16	.02	3	57.0	3.5	.198	.046
Controls	12	48.8	16.7	.287	.076	.15	.03	0				
All subjects	49		—	.258	.070	.17	.05	81		—	.173	.06

noted for outpatients (6). Blood from relatives of patients was obtained in the early afternoon, and blood from inpatients was obtained in the morning before breakfast (9–24 hours after the last lithium dose). Some inpatients were studied at weekly intervals.

Laboratory procedures for the study of the lithium pump are described in detail elsewhere (1). Briefly, the kinetics of lithium efflux were measured after suspension of erythrocytes in a modified Ringer's solution and incubation at 37° C. The efflux rate constants were obtained from the increase in lithium concentration in the Ringer's solution over a 2-hour period. Erythrocytes from subjects who had not been taking lithium carbonate were preincubated with a Ringer's solution containing 15–20 mEq/liter of lithium in order to preload the erythrocytes. This preloading was also performed in cells of patients who had been treated with lithium. Data for lithium pump activity are expressed as rate constants: k_o refers to the activity of the process that transports lithium out of erythrocytes, and k_i to lithium influx into erythrocytes. In our laboratory, measurements of k_o are considered more reliable than measurements of k_i because of certain technical problems in the assay for k_i. Units for these rate constants are per hour. For purposes of this report, the k_i/k_o ratio was calculated using uncorrected data for k_i (i.e., no correction for the effect of k_o on lithium accumulation in erythrocytes). In other reports we have applied such a correction factor, which entails multiplying the k_i/k_o ratio by approximately 1.2.

RESULTS

Lithium pump activity was assessed in 49 subjects who were not being treated with lithium carbonate (see table 1). The mean k_o (±SD) in these individuals was .258±.070. There was no significant correlation between k_o and age of subjects (r=.07), and there were no significant differences between mean k_o for men and women controls (t=.61). Similarly, there were no significant differences in mean k_o between groups of patients. The mean k_i/k_o ratio for all subjects was .17±.05.

The mean k_o in 81 subjects who were being treated with lithium (see table 1) was significantly different from that for subjects who were not being treated with lithium (t=7.51, p<.01). The mean k_o in the lithium group was significantly different from that in the non-lithium group (t=6.55, p<.01). There were no significant differences in mean k_o among the groups of lithium-treated patients.

The effect of lithium on k_o is further illustrated in table 2, which presents data from 6 patients who were studied before and during lithium treatment. All 6 showed a reduction in k_o with lithium treatment (paired t=5.32, p<.01), and 5 of the patients showed an increase in k_i/k_o (paired t=3.45, p<.02).

The stability of k_o over time in 25 lithium patients (and 1 non-lithium-treated control) is illustrated by considering data from patients who were treated with lithium for longer than 2 weeks. Preliminary data suggest that k_o will decrease with time. The mean k_o during early stages of treatment was almost identical with the mean 2 weeks later (means=.190±.073 and .189±.071, respectively, range of repeated determinations 2 weeks). The correlation coefficient between initial and later measurements was high (r=.96, p<.01).

The relationship of lithium pump activity to the Li^+/Na^+ ratio could be assessed in 53 subjects. In these subjects, the pump activity and the Li^+/Na^+ ratio were determined from blood samples taken during the same clinic visit. The Pearson's correlation coefficient for the relationship between the k_i/k_o ratio and the Li^+/Na^+ ratio was r=.49 (p<.01). The correlation for the relationship between k_o to the Li^+ ratio was r=−.47 (p<.01). The relationship between k_i and the Li^+ ratio was not significant.

Several relatives of patients volunteered for the research study. To analyze these data, we examined the k_o correlation in 15 pairs of first-degree relatives.

TABLE 2
The Lithium Pump: Intrapatient Effect of Lithium Treatment

	Before Lithium		During Lithium	
Patient	k_o	k_i/k_o	k_o	k_i/k_o
1	.191	.20	.147	.36
2	.221	.14	.149	.28
3	.248	.18	.150	.31
4	.321	.14	.299	.12
5	.302	.12	.193	.21
6	.303	.13	.218	.19
Overall				
Mean	.264*	.15**	.193	.24
SD	.052	.03	.060	.09

*Statistically higher than during lithium, paired $t=5.32$, $p<.01$.
**Statistically lower than during lithium, paired $t=3.45$, $p<.02$.

including probands and first-degree relatives if the proband was not treated with lithium. We also computed the correlation for 4 pairs of subjects who were not genetically related but who were living in the same household—these subjects were parents of probands. A high correlation was found between first-degree relatives ($r=.65$, $p<.01$), but the correlation between nongenetically related subjects was lower ($r=.38$). Furthermore, the correlation for 23 pairs of "random" non-lithium subjects who were not genetically related was $r=.12$ (n.s.).

We were able to study k_o in 2 patients who were admitted during states of lithium toxicity. Both patients showed reduction in k_o during lithium toxicity (values of .088 and .148) compared with later analysis in the Lithium Clinic (values of .176 and .178).

DISCUSSION

Activity of the lithium pump can be determined in individuals regardless of whether they are being treated with lithium carbonate. For this paper, we analyzed data on the lithium pump up to March 1, 1977. It should be noted that our studies of the lithium pump are continuing, and we plan to augment the data presented here with data on more subjects.

The data we have obtained thus far reveal several facts about the lithium pump. In subjects who are not being treated with lithium carbonate, k_o does not seem to be related to age, sex, or diagnosis. k_o appears to be stable over time and may be determined, at least in part, by a genetic factor. During lithium treatment k_o is considerably reduced, and there may be even further inhibition of k_o during lithium toxicity. A significant correlation can be demonstrated between the activity of the lithium pump and the Li^+ ratio.

Many questions regarding the lithium pump remain for further study. Preliminary data suggest that the activity of the lithium pump may correlate with the outcome of lithium prophylaxis (7). Also, there appears to be a critical dose of lithium that will cause inhibition of the lithium pump, and the relationship of this dose to therapeutic plasma lithium concentration requires further study (7). Other issues, such as the relationship of the efflux pump to lithium plasma concentrations, the mechanism of inhibition of the pump by the lithium ion, and the relationship of the lithium pump to other ions (e.g., calcium and magnesium) and to other cation transport mechanisms, await further study.

Our data suggest a possible role of the efflux pump in lithium toxicity. In this speculative model, lithium toxicity could be defined as occurring when the lithium efflux pump is inhibited beyond the usual inhibition that occurs with lithium treatment. Inhibition of the efflux pump would lead to higher intracellular lithium ion concentrations (and abnormally elevated Li^+ ratios). Treatment of lithium toxicity would entail treating the inhibition of the lithium pump. According to this model, changes in the lithium efflux pump activity and/or the lithium erythrocyte/plasma ratio would reflect and parallel clinical changes during lithium toxicity as the high intracellular lithium load is transported to the extracellular fluid compartments.

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Erythrocyte Concentrations of the Lithium Ion: Clinical Correlates and Mechanisms of Action

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When lithium carbonate is administered to individuals, there is considerable intersubject variation in the extent of accumulation of Li^+ by erythrocytes. The primary reason for this is differences in the activity of a Li^+-Na^+ counterflow system, which, under clinical conditions, removes Li^+ from the cell. It appears that some bipolar patients accumulate more Li^+ in their red cells than either unipolar depressives or normal controls. The precise clinical characteristics of the bipolar patients who accumulate relatively large amounts of erythrocyte Li^+ need to be clarified in future research. Finally, the measurement of red cell concentrations of Li^+, in addition to the usual plasma measurement, can be used as an indicator of patient compliance.

EXTENSIVE RESEARCH has been stimulated by our observations (1–4) suggesting that study of the relative accumulation of the lithium ion (Li^+) by erythrocytes (RBCs), expressed as the ratio of lithium in the erythrocyte to its concentration in plasma (Li^+ ratio), might help identify a distinct subgroup of depressed patients and perhaps even a biological abnormality in these patients. This work has included examination of a variety of clinical phenomena that might be associated with variations in the distribution of Li^+ across the erythrocyte membrane, as well as the physiological mechanisms involved in transfer of Li^+ across this membrane. In this paper, we will review the current status of this continuing research.

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Our initial study (1) involved 13 depres[sed] pitalized on a research ward. Several [ob]servations were made when erythrocyte [con]trations were measured simultaneously [with] levels of the cation. Although there was [some] intersubject variation (about twofold) [in] of the Li^+ ratio (a similar variability ha[d] earlier by Maggs [5] in a small group of st[ud]. was good intrasubject stability in the [same] time. All of the subjects had less lithi[um] cytes than in plasma, implying that unde[r] conditions Li^+ is not being distributed [in] with Gibbs-Donnan equilibrium. Two q[ues]from these observations: 1) What is the [basis] intersubject variability? 2) What facto[rs] transfer of Li^+ across the red cell mem[brane]?

Our initial study noted that the depr[essed] who improved with lithium carbonate de[fined] er Li^+ ratios than the nonresponders ([.]39±.02, $p<.005$, Mann-Whitney U te[st]. we were not able to replicate this find[ing] (as yet unpublished) study (see table 1) [of] the initial study, the second study inclu[ded] of outpatients and women. We reviewed [in] the original study to determine whether [any] factor might account for the difference [in] between the two studies. Variables such [as] illness, dosage of lithium carbonate, an[d] centration of Li^+ do not seem to acco[unt] parity in the results.

One factor that might provide an e[xplan]these apparently discrepant results is [the] between unipolar and bipolar affective [ill]the 9 lithium responders in the first stu[dy] affective illness, whereas 50% of the [non]were diagnosed as unipolar depressives [(]tion between a positive antidepressant [re]lithium carbonate and the diagnosis of [bi]has been noted before (see reference (6[)]). study, the 3 bipolar patients who did [not] treatment with lithium carbonate had [low] Li^+ ratios. In the second study, although [the] tion between bipolar illness and a posit[ive] sant response to lithium carbonate pers[ists] the 4 bipolar patients who did not respo[nd] high mean Li^+ ratio (.61±.06). Thus, w[hile] not be an association between the anti[de]fectiveness of lithium and the Li^+ rat[io].

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TABLE 1
Lithium Ratio in Patients Treated with Lithium Carbonate During a Depressive Episode

Patient Group	N	Li^+ Ratio Mean	Li^+ Ratio SEM	Plasma Li^+ (mEq/liter) Mean	Plasma Li^+ (mEq/liter) SEM
All responders	24	.50	.03	.83	.02
All nonresponders	12	.50	.06	.88	.04
Bipolar responders	20	.53	.03	.83	.02
Bipolar nonresponders	4	.61	.06	.86	.05
Unipolar responders	4	.36	.04	.84	.03
Unipolar nonresponders	8	.44	.07	.88	.05

difference in the Li^+ ratio between unipolar and bipolar diagnostic subgroups. It may have been this association between the Li^+ ratio and diagnosis that we misinterpreted as an association with the antidepressant response in our initial report.

We have examined this question in a large sample of patients and have noted a significant difference ($p<.03$) between the Li^+ ratio in 23 unipolar patients (mean=.41, SEM=.03) and 29 bipolar patients (mean=.52, SEM=.02), with the difference being most pronounced for men. In the bipolar group, the clinical state, i.e., depressed, hypomanic, or manic, had no apparent effect on the Li^+ ratio. This suggests that the ratio may be a trait rather than a state variable.

While other investigators have also reported differences in the Li^+ ratio between unipolar and bipolar patients (9, 10), this has not been a consistent observation (11–14). Factors that might contribute to this inconsistency will be discussed later in this paper.

It must be emphasized that the Li^+ ratio cannot be used to make the diagnosis of unipolar or bipolar illness, even in men, because there is clearly substantial overlap between the groups. However, part of the variance in the Li^+ ratio between the subjects we have studied can be accounted for by the unipolar-bipolar distinction.

Comparisons of lithium erythrocyte/plasma ratios for patients and normal volunteers indicate that the ratio tends to be higher in the bipolar patients. Data on normal volunteers are still very limited (2, 8, 15), and many of these subjects have taken lithium for a short time and have relatively low plasma lithium concentrations. We have recently given 21 normal subjects daily doses of lithium carbonate for 21 days. In these subjects, the mean plasma concentration of Li^+, measured between days 7 and 21 of lithium administration, was .76±.02 mEq/liter and the Li^+ ratio was .46±.02. The difference in the mean ratios between this group of normal subjects and the bipolar patients studied earlier just fails to reach statistical significance ($.05<p<.10$). Recently, using an in vitro test to predict the Li^+ ratio (16), we have noted a significant difference in the mean ratios in 79 bipolar patients and in 49 non-psychiatrically-ill control subjects (17).

It thus appears that bipolar patients are more likely to develop high Li^+ ratios than are unipolar or control subjects. It remains unknown whether this is a characteristic of some bipolar patients (it has been suggested that bipolar illness may include at least two genetic subtypes [18]) or an as yet undefined subgroup of depressed patients that overlaps with (or is independent of) the bipolar-unipolar dichotomy.

Because of the importance of long-term maintenance treatment with lithium carbonate in selected patients, we have also examined the relationship between a beneficial "prophylactic" effect from lithium and the Li^+ ratio. A preliminary retrospective study involving 31 patients with recurrent affective disorders was conducted in association with Dr. A. Kukopulos and associates in Rome, Italy (see reference 4). The patients who benefited from lithium prophylaxis had a significantly higher mean Li^+ ratio than the nonresponders (.66±.03 versus .44±.03, $p<.001$, Mann-Whitney U test).

This observation remains controversial. Several investigators (19, 20) have reported that patients who have responded well to lithium treatment for periods ranging from 6 months to 4 years had significantly higher ratios than patients who relapsed. However, others (10, 21) have been unable to replicate these results. Clearly, what is needed is a properly controlled prospective study of the effectiveness of long-term maintenance treatment with the lithium ion as it relates to the Li^+ ratio.

METHODOLOGIC ISSUES IN LI^+ RATIO RESEARCH

There is substantial disagreement among investigators as to the clinical significance of the Li^+ ratio. Although the "positive" claims may be in error because of such factors as small, biased patient samples, it is also possible that important methodologic differences between studies may be responsible for the lack of agreement. It is important to consider what these methodologic factors might be and to control for them in future studies. Similar controversy and inconsistency surrounds almost all biological measures in psychiatry, including reports of biogenic amine and psychoendocrine abnormalities (see reference 22).

Of initial importance are the clinical criteria for treatment response and for diagnosis. These problems have been extensively discussed elsewhere (23–25) and need not be detailed here. However, it is clear that differences in these criteria could be a major source of inconsistencies. The use of standardized diagnostic criteria, e.g., those of Feighner and associates (26) or the modifications embodied in the *Research Diagnostic Criteria* (27), would obviously be of help in reducing this problem.

There are also several important technical problems that have not been approached in a consistent fashion by all investigators; these will be discussed below.

Direct Versus Indirect Measurement of Erythrocyte Lithium Concentration

Several groups of investigators (7, 8, 10) have used an "indirect" method (i.e., assessing the difference between whole blood lithium concentration and plasma lithium concentration) to measure erythrocyte Li^+ concentration. We have recently shown (28) that this indirect method gives much more variable, and presumably less accurate, results than values obtained by measuring the concentration of the cation in a hemolyzate made directly from packed erythrocytes. It appears to be relatively unreliable to calculate a mean value for an individual subject when using the indirect method unless there are a number of separate values for each subject.

Compliance with Lithium Medication Regimens

Medication compliance is a major problem for all studies that include outpatients. Our initial observations in hospitalized patients indicated that the intraindividual Li^+ ratio was relatively stable over time (1), but we have seen considerably more intraindividual variation over time in outpatients. The possible contribution of poor medication compliance to the variability in the Li^+ ratio has been demonstrated as follows: Venous blood was drawn from 6 patients receiving lithium carbonate—first in the morning after no lithium had been ingested for 12–14 hours (as is standard practice) and then 2 hours after a morning dose of 300 mg. As shown in table 2, the mean Li^+ ratio on the second occasion was significantly lower than that on the first occasion (29).

Thus, outpatients who accidentally or deliberately take a morning dose of lithium before having blood drawn may have spuriously low Li^+ ratios. This effect would be exaggerated if doses larger than 300 mg of lithium carbonate were taken before blood was drawn. These data suggest that the Li^+ ratio might be used as an indicator of patient compliance. Measurement of the plasma concentration of the cation alone may be misleading, since apparently therapeutic levels of plasma Li^+ can be achieved even if the patient has missed many doses over the preceding few days. However, significant swings in the Li^+ ratio over repeated visits probably indicate that an individual has not been taking the drug according to the schedule prescribed. Compliance problems may also be partly responsible for the alleged effect of plasma Li^+ on the magnitude of the Li^+ ratio (30).

TABLE 2
Effect of a 300-mg Morning Dose of Lithium Carbonate on Li Ratio

Patient	Plasma Li^+ (mEq/liter)		Erythrocyte Li^+ (mEq/liter cells)		Ba
	Baseline	2 Hours	Baseline	2 Hours	
1	0.91	1.26	0.52	0.52	
2	1.00	1.16	0.57	0.54	
3	0.98	1.07	0.84	0.80	
	0.68	0.91	0.53	0.57	
4	0.55	0.66	0.18	0.18	
5	1.00	1.02	0.42	0.42	
6	0.88	1.17	0.53	0.55	
	0.51	0.92	0.26	0.32	

TRANSFER OF LI^+ ACROSS THE ERYTHROCYTE MEMBRANE

Despite the continued uncertainty about the clinical significance of differences in the Li^+ ratio, considerable attention has been directed to the examination of factors that determine the distribution of Li^+ across the erythrocyte membrane, in an effort to understand the considerable intersubject variation obs investigators. This body of work indicates ium ion can move into the red cell by th mechanisms: 1) diffusion down a concen ent, a "leak" process that appears to be the presence of bicarbonate ion in the me transport of the K^+ site of the Na, K-activ sine triphosphatase (ATPase) (31, 32); an into the cell in exchange for an internal (Na^+), a Li^+-Na^+ counterflow system or e fusion system (33–35).

Only two processes appear to be involv flux of Li^+ from the cell because under cl vant conditions there is no active transpo of the cell on the Na^+ site of the Na, K ATPase (32, 36). These processes are diffu concentration gradient or transfer out by counterflow or exchange system.

Under clinical conditions, the concent in the cell appears to be determined prima ance between its entry by diffusion down tion gradient (higher concentration in pl the cell) and its removal by the Li^+-Na system. The other mechanisms listed abov relatively less important.

We have recently used computer sim nonlinear regression techniques to inves of these factors is responsible primarily subject variation in the magnitude of the an initial study (37), it was shown that interindividual variation is related pri ferent rates of efflux of Li^+ from the cel

Based on the factors cited above, we oped a more sophisticated model of the t esses (see figure 1), which takes into acco tant physiological factor: the effect of se Li^+ transfer. This model generates th equation shown at the bottom of figure 1 calculated the three unknown constants tion, k_{leak}, $k_{exchange}$ and $flux_{active}$, by mea take by red cells in vitro and using the gression techniques mentioned above.

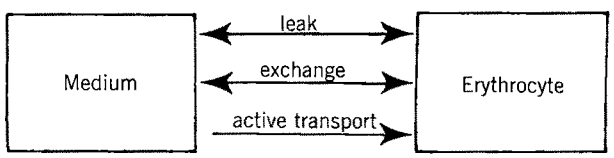
The results obtained for 7 subjects st

TABLE 3
Computer-Derived Parameters for Li^+ Exchange Across the Erythrocyte Membrane In Vitro

Subject	k_{leak} ($hr^{-1} \times 10^3$) Mean	k_{leak} 95% CL*	$k_{exchange}$ $\left(\frac{liter}{mEq/hr} \times 10^3\right)$ Mean	$k_{exchange}$ 95% CL	$Flux_{active}$ $\left(\frac{mEq}{hr} \times 10^3\right)$ Mean	$Flux_{active}$ 95% CL	Observed Li^+ Ratio
A	7.7	4.5	6.0	4.0	1.0	1.5	.053
B	17.5	2.7	3.7	0.6	0.1	2.2	.059
C	15.0	2.5	2.6	0.4	6.6	4.2	.084
D	8.8	1.9	1.7	0.5	7.3	4.4	.090
E	9.9	3.0	1.7	0.5	7.3	5.2	.103
F	13.6	2.1	2.0	0.2	10.9	3.5	.113
G	8.7	1.8	0.8	0.3	5.4	4.5	.148

*CL=confidence limit.

FIGURE 1
Schematic Representation of the Exchange of the Lithium Ion Between the Incubation Medium and Erythrocytes



$$\frac{d[Li_e]}{dt} = k_{leak}([Li_m] - [Li_e]) + k_{exchange}([Li_m][Na_e] - [Li_e][Na_m]) + flux_{active}^*$$

*$Flux_{active}$=0 in the presence of ouabain.

are shown in table 3. Inspection of the data reveals that the values for k_{leak} vary only about twofold between subjects; furthermore, it is obvious that there is no correlation between the magnitude of the rate constant for the leak process and that of the Li^+ ratio actually measured. It may be concluded, therefore, that passive diffusion of Li^+ in vitro shows little variation among individuals and cannot explain interindividual differences in the magnitude of the Li^+ ratio.

Similarly, there is no obvious relationship between the rate constant for Li^+ movement on the K^+ site of the Na, K-activated ATPase (i.e., $flux_{active}$) and the magnitude of the experimentally determined Li^+ ratio. In contrast, values for $k_{exchange}$ are related inversely to the Li^+ ratio, and also vary by a factor of approximately sevenfold among subjects. Therefore, intersubject variability in the magnitude of the Li^+ ratio probably results primarily from differences in the rate of the Li^+-Na^+ counterflow or exchange diffusion process, a system that drives Li^+ out of the cell. Low values of $k_{exchange}$ for an individual may result from either a lesser number of carrier sites or turnover of the sites (i.e., a lower V_{max}), a lesser affinity of the substrate for the sites (K_m), or both. A kinetic analysis by Duhm and Becker (38), using data from 4 control subjects, indicates that it is the number of carrier sites (V_{max}), rather than the affinity constants that varies among individuals.

These results indicated that it was reasonable to assume that if an in vitro system were developed by which Li^+ movement between cells and medium occurred primarily by the activity of the Li^+-Na^+ counterflow system, then the distribution of Li^+ between cells and medium measured in such a system would correlate highly with the distribution observed clinically. We have developed such a system (16) and have noted a very high correlation ($r>.90$) between the Li^+ ratio measured clinically and that measured in RBCs after a 24-hour incubation in an artificial plasma-like medium. Systems similar to ours have also been developed by Greil and associates (39) and by Pandey and associates (40) and have yielded correlations with the in vivo Li^+ ratio very similar to those we have found. Such systems should prove useful for further investigations into the relevance of the erythrocyte accumulation of Li^+ to the pathogenesis and treatment of affective disorders.

CONCLUSIONS

The uncertainties and problems surrounding our understanding of the clinical significance of the variations in the rate of Li^+ accumulation in erythrocytes are not dissimilar to the problems associated with other "systems" in biological psychiatry. Initially intriguing findings, partial replications, numerous methodologic problems, and small sample sizes for most studies seem to be a repetitive pattern. There is an urgent need for more carefully designed studies, adequate numbers of appropriately defined patients, and control data. Some of the issues are now more adequately defined insofar as this system is concerned, and, in particular, the development of a reliable in vitro procedure allows us to study large numbers of control subjects. These data should be crucial in clarifying the clinical significance of this system. In the meantime, we have gained important physiological information about the passage of the lithium ion across cell membranes that has served to focus our attention on the broader issue of possible abnormalities in transport systems and affective illness.

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A Heritable Disorder of Lithium Transport in Erythrocytes of a Subpopulation of Manic-Depressive Patients

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The in vivo RBC/plasma Li^+ ratio is determined by the equilibrium between Li^+ influx (ouabain-sensitive Na^+-K^+ pump and ouabain-insensitive leakage pathways) and Li^+ efflux (phloretin-sensitive Li^+-Na^+ counterflow). A study of RBC Li^+ transport via these pathways showed that a deficiency of Li^+-Na^+ counterflow was responsible for the high in vivo ratio (1:1) observed in a manic patient. This defect was related to an alteration in the membrane Na^+ exchange system and was under genetic control. The level of counterflow before lithium therapy was an excellent predictor ($r=.88$) of the in vivo Li^+ ratio and was deficient in approximately one-fourth of manic-depressive patients but not in controls, schizophrenics, or unipolar depressed patients.

LITHIUM IS EFFECTIVE in the treatment of manic-depressive (bipolar affective) illness (1, 2). The red blood cell/plasma lithium concentration ratio (Li^+ ratio) is relatively constant for each individual (3), a fact that has led to considerable interest in whether or not the in vivo Li^+ ratio in bipolar patients differs from that in normal individuals (4) and/or is related to response to lithium therapy (3, 5, 6). Recent in vivo and in vitro studies in normal subjects have demonstrated that genetic factors contribute to interindividual differences in the ratio (7, 8), and family studies have shown a genetic predisposition to bipolar affective illness (9). The above lines of investigation have led to explorations of the biochemical mechanisms underlying the maintenance of the Li^+ ratio in human RBCs (10–13). It is of interest to examine the question of whether any of those factors that control the Li^+ ratio themselves may be under genetic control.

Although alkali cation transport processes are complex, they can be schematically divided into three major mechanisms. Under normal physiological conditions, the ouabain-sensitive Na^+-K^+ pump actively transports these ions uphill, potassium into the cell and sodium from the cell to the extracellular medium (14). Another mechanism of sodium transport by cell membranes is the sodium-sodium exchange pathway, which allows for the stoichiometric exchange of intracellular for extracellular sodium ions and therefore cannot be responsible for net uphill transport of sodium but rather functions as a facilitated exchange diffusion pathway (15, 16). Finally, there is the downhill leakage of cations along their concentration gradient. We have characterized three distinct pathways of lithium transport, which resemble the three pathways described for sodium-potassium transport (see figure 1) (10–13). These three pathways are operationally distinguishable by virtue of their differential sensitivities to ouabain and phloretin. In the first pathway, lithium is transported by the inward loop of the ouabain-sensitive Na^+-K^+ pump, the loop normally used by K^+ (11). Lithium can also be transported by a Li^+-Na^+ counterflow system, which results in the transport of lithium in the direction opposite to that of the sodium gradient and is inhibited by phloretin (11, 12). Finally, lithium can diffuse through inhibitor insensitive pathway(s) down its chemical concentration gradient. Under normal physiological conditions, namely $[Na]_{rbc}=5$–10 mEq/liter and $[Na]_{plasma}=140$ mEq/liter, the phloretin-sensitive Li^+-Na^+ counterflow system operates to extrude Li^+, and the concentration of Li^+ is maintained at a level 2–3 times higher in the plasma than in the RBC. Thus, the steady-state distribution of lithium between the red cell and plasma in vivo depends primarily on the balance between influx via the insensitive leakage pathway(s) and efflux via the phloretin-sensitive Li^+-Na^+ counterflow system. This picture of lith-

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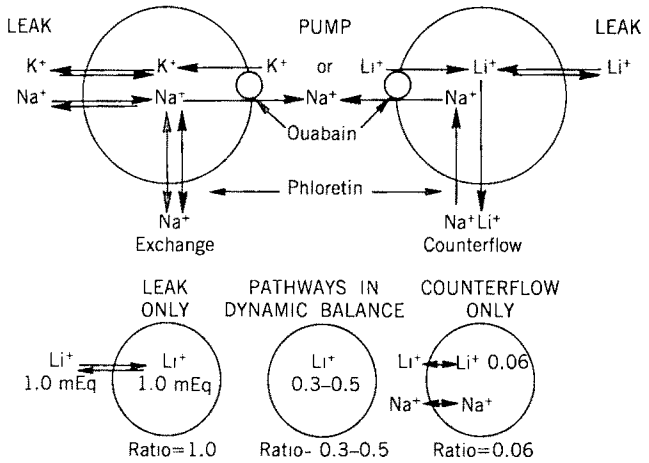
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FIGURE 1
Transport Determinants of the In Vivo Li^+ Ratio



ium transport has been confirmed by several independent groups of investigators (13, 17, 18).

As shown in figure 1, alterations in the balance between these various pathways can result in large changes in the intracellular/extracellular lithium ratio. For example, if the insensitive leakage pathways were negligible and only the Li^+-Na^+ counterflow system were operating, the distribution of Li^+ between the inside and outside of the cell would be identical to the distribution of Na^+, giving a ratio of approximately 0.06 (see figure 1, lower right). In contrast, if the influx pathways were operative and the Li^+-Na^+ counterflow system negligible in RBCs (see figure 1, lower left), the distribution of Li^+ in the steady state would be determined predominantly by the electrochemical potential of the cell, and the ratio would be approximately 0.8–1.0 (15). We already know that most individuals on lithium have a ratio in the range of 0.3–0.5, indicating a dynamic balance between influx of Li^+ via the leakage and ouabain-sensitive pathways and efflux via the Li^+-Na^+ counterflow system.

In the course of our study of Li^+ transport in both normal individuals and patients with affective disorders, we observed a patient with a Li^+ ratio of 1.0. We found that this increased ratio is the result of a deficiency or defect in this individual's RBC phloretin-sensitive Li^+-Na^+ counterflow pathway, which is related to the Na^+ exchange diffusion system of the membrane, and that this defect is genetically transmitted. In addition, we have demonstrated that this decrease in phloretin-sensitive Li^+ transport is found in the RBCs of a subgroup of manic-depressive individuals. Some of these findings have been presented in preliminary reports (19, 20).

METHOD

Subjects

Control subjects were employees of either the Department of Research at Illinois State Psychiatric Institute (ISPI, a state hospital) or the Depart... chiatry, Michael Reese Hospital and Med... (MRH-MC, a private psychiatric hospital) ... subjects were screened for medical or psy... orders and were without history of affecti... first-degree relatives.

Psychiatric patients were either hospit... research wards at ISPI (including the 21-... described below) or were manic-depressiv... or outpatients at MRH-MC. All psychiat... were diagnosed by both *DSM-II* (21) and ... teria and gave written informed consent be... pating in the study. Inpatients were all ... cept for the manic-depressive patients o... the time of transport measurement, the ... patients for a minimum of 2–3 weeks an... MC patients for varying lengths of time. M... sive patients on lithium were on fixed d... steady plasma lithium levels for at leas... in vivo Li^+ ratio determinations and ... redeterminations.

Family studies were performed for se... depressive patients with severe Li^+ tra... malities. Subjects included as many firs... tives (parents, siblings, and offspring) ... able and willing to participate. Relati... formed written consent and were ... evaluated for psychiatric illness unless t... diagnosed previously as having a psychia...

Not all subjects participated in all aspe... search protocol, so the actual number o... patient subjects assayed differs for ea... transport measurement or experiment. ... number of subjects is indicated in each f... with explanatory footnotes where necess... number of subjects by diagnostic catego... any or all phases of the study is given in ...

Protocol

Blood was drawn from both normal con... tients during their drug-free period. W... were prepared and the three sets of stud... below were done on these cells.

1. Twenty-four hour in vitro Li^+ ratio... by incubation of the cells in a plasma-like ... taining 1.5 mM lithium chloride at 37° C ... after which the ratio of intracellular to ... Li^+ was determined (7).

2. Li^+ influx rates were determined ... RBCs in a magnesium chloride medium ... 20 mM lithium chloride and measuring i... crease of intracellular lithium at 37° C o... 1–2 hours. By incubating these cells eithe... ence or absence of 10^{-4} M phloretin and o... bain, linear influx rates were obtained ... sensitive, ouabain-sensitive, and phlore... sensitive lithium influx (19).

3. Phloretin-sensitive Li^+-Na^+ cou... measured by preloading cells to a lithiu... mEq/liter of RBCs by incubating for ap...

TABLE 1
Subjects By Diagnostic Category

Diagnosis	N	Sex		Race			Age (years)	
		Male	Female	White	Black	Oriental	Mean	SD
Normal controls	30	21	9	25	5	0	26.4	3.6
Manic-depressive (bipolar I)	50	19	31	41	8	1	30.8	12.1
Bipolar other (bipolar II, cyclothymic personality)	9	6	3	7	2	0	28.3	4.0
Manic-depressives on lithium*	59	24	35	49	9	1	31.1	11.8
Unipolar depressive	10	4	6	8	2	0	33.0	5.2
Schizophrenic	10	6	4	3	7	0	25.0	5.2

*Includes 47 from drug-free group.

TABLE 2
Li^+ Ratio and Counterflow Measurements*

Time of Measurement	Li^+ Ratio			Phloretin-Sensitive Li^+-Na^+ Counterflow (mEq Li^+ efflux/liter RBCs/4 hours)		
	N**	Mean	SD	N**	Mean	SD
Before Treatment						
Patient	3	0.73	.21	1	.110	
Normal controls	12	0.23	.03	21	.309***	.076
During lithium treatment						
Patient	11	0.96	.03	4	.054	.009
Normal controls†	8	0.39	.03			—
After lithium treatment						
Patient	3	0.65	.02	1	.105	
Normal controls			—			—

*All measurements are in vitro duplicate measurements except Li^+ ratio during lithium treatment, which is in vivo.
**Ns for the patient represent independent observations; other Ns are numbers of individuals assayed.
***Significantly different from patient value, $p<.005$.
†Data from Lyttkens and associates' study of normal women (4); other control data are from our sample of normal men.

hour in a medium containing 40 mM lithium chloride in a sodium chloride medium. Following this, the cells were washed and then suspended in a medium containing 1.5 mEq/liter of lithium in 135 mM sodium chloride, and the change in internal lithium in the presence and absence of 10^{-4} M phloretin was determined (20).

Those patients diagnosed as having bipolar affective illness were placed on lithium therapy according to a standard research protocol that is used on our units. Following attainment of a steady-state lithium dosage, blood was drawn from these individuals on several occasions for determination of the in vivo lithium ratio (5). In addition, a sample of blood was obtained from patients on a steady-state lithium dosage for redetermination of lithium influx rates and phloretin-sensitive Li^+-Na^+ counterflow.

In vivo and in vitro Li^+ ratio values are expressed as (milliequivalents of lithium per liter of RBCs)/(milliequivalents of lithium per liter of plasma or media).

Analytical Procedures and Reagents

Cells and medium were analyzed for K^+, Na^+, and Li^+ by atomic absorption spectrophotometry, for hemoglobin by spectrophotometry, and for water content by weighing before and after drying at 110° C for 14 hours. ^{22}Na was measured in the medium and cells by counting the gamma radiation in a crystal scintillation spectrometer.

Statistical Methods

Linear Li^+ influx rates were determined independently in each half of the patient's blood sample by regression analysis of the RBC Li^+ concentrations determined at 0, 1, and 2 hours of incubation. Values used in the analyses were averages of the values from each half of the sample. Assays that proved to be poorly estimated by a linear regression ($r^2<.85$) were discarded and redetermined. Significance of group differences was determined by two-tailed t tests using the 95% confidence level.

RESULTS

In the course of studying lithium transport parameters of normal control subjects and manic-depressive patients, we discovered a patient whose Li^+ ratio of 1.0 indicated a severe derangement of lithium transport. The characterization of the defect of lithium transport in this patient is followed by the overall results of our studies in normal and patient samples.

High Lithium Ratio in One Patient

Table 2 shows the in vitro and in vivo lithium ratio in a 21-year-old black man admitted after a week of overactivity, restlessness, insomnia, pressure of speech, tangential associations, euphoria, and grandiose ideas related to his forthcoming graduation. This was the patient's first psychiatric admission, and he fulfilled both the *RDC* and *DSM-II* criteria for manic illness.

During the first 2 weeks of lithium treatment his affect normalized, his thinking slowed and became more organized, and grandiose, delusional thinking abated.

TABLE 3
Li+ Transport Rates in Patient's Family*

Subject	Sex	Age	Phloretin-Sensitive Counterflow (mEq/liter cells/4 hours)	Li+ Influx(mEq/liter ce... Ouabain-Sensitive	Inhib...
Father	M	55	.08**	.89**	
Mother	F	41	.16	.33	
Siblings					
1 (patient)	M	21	.05**	.51	
2	F	20	.01***	.35	
3	M	18	.16	.46	
4	F	17	.12	.34	
5	M	16	—	—	
6	F	13	.06	.34	
7	F	12	.00***	.42	
8	F	10	.15	.39	
Controls†					
Males (N=21)			.31±.08	—	
Females (N=9)			.21± .09	—	
Combined (N=18)			—	.36±.09	

*Values are means of duplicate determinations for each individual on one occasion, except for sibling 7, who was assayed on 2 occasions, ... was assayed on 4 occasions while on lithium for counterflow and twice when not on lithium for influx rates.
**Significantly different from controls, p<.01.
***Significantly different from controls, p<.05.
†Counterflow values are given separately for males and females because these values differ significantly (p<.005).

He was discharged after 6 weeks of hospitalization, during which his behavior returned to normal, and was maintained on 1800 mg/day of lithium carbonate for the next 5 months. Lithium was discontinued 1 year ago, and the patient has done well since then.

As can be seen in table 2, the Li^+ ratios for this patient both before and during lithium therapy were approximately 3 times higher than those seen in normal controls for the same periods. The patient's ratio during lithium treatment approaches the passive distribution of a monovalent cation across the RBC membrane (15), suggesting an absence of or defect(s) in the pathway(s) responsible for the maintenance of the normal lithium ratio. One such pathway, the phloretin-sensitive Li^+-Na^+ counterflow pathway, was significantly reduced in this patient before and after treatment in comparison with normal controls and was also reduced by lithium treatment (table 2). Other pathways were normal or slightly elevated in this patient (see table 3).

The close relationship between phloretin-sensitive Na^+-Na^+ exchange and phloretin-sensitive Li^+-Na^+ counterflow (23) suggested an investigation of this pathway using Na^+, Li^+, or K^+ as the external monovalent cation. Both the patient and the patient's father, who also showed a significantly decreased Li^+-Na^+ counterflow, demonstrated a near or total absence of phloretin-sensitive, Li^+-stimulated sodium exchange (figure 2).

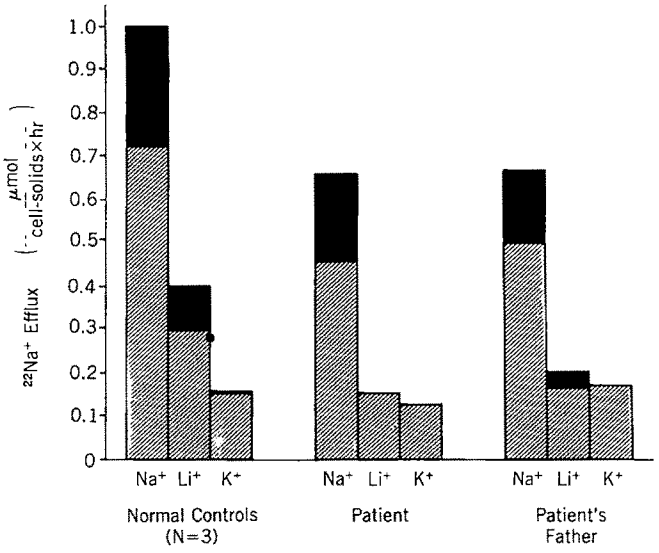
To determine the locus of the defect, RBCs from the patient and a normal control were preloaded to a lithium content of 1.3 mEq/liter and suspended in plasma from both individuals. Plasma contained 1.5 mEq of lithium per liter. The patient's cells were unable to extrude lithium in his own or the control plasma, while control cells extruded similar amounts in both patient and control plasma (.50 and .46 mEq/lite... ly, in 4 hours). Moreover, the results of ... hematologic work-up found no cytoplasm... might account for the patient's defective ce...

Inherited Basis of the Li^+-Na^+ Counterflo...

Table 3 shows the data obtained for the ... transport via the three major pathways i... the patient's first-degree relatives (parent... full siblings). All family members d... phloretin-sensitive Li^+-Na^+ counterflo... counterflow levels for the father and 2 c... (numbers 2 and 7 in table 3) significantly ... absent. In contrast, ouabain-sensitive L... not decreased in any of the patient's relat... increased in his father. Inhibitor-insensit... was normal in all but the patient's father ... who had decreased influx. We have th... cluded that the decreased or absent phlore... Li^+-Na^+ counterflow found in this pati... bers of his family is under genetic contro... pedigree for the phloretin-sensitive Li ... flow defect in this family is shown in figu... mission pattern seen here is compatible ... inheritance. Father-to-son transmission ... sistent with autosomal control of the co... fect.

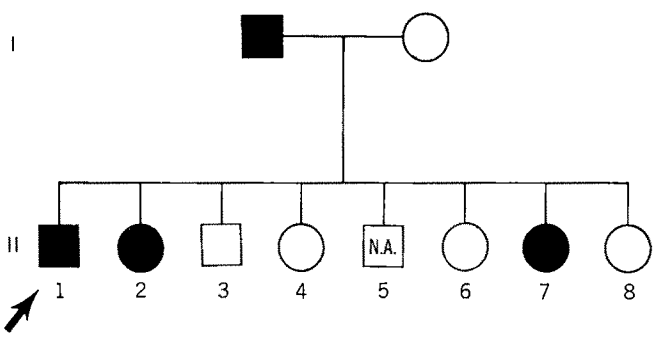
[1]A complete hematologic work-up of the patient w... Dr. Henri Frischer and associates, Department of ... Presbyterian-St. Luke's Hospital-Medical Center ... included all standard hematologic tests as well as ... tery of red cell enzyme determinations. The patient ... trolyte, pH, and water contents were determined ... Gunn of the Department of Pharmacological and P... ences, University of Chicago. The results of all of ... within normal limits. A complete summary of this ... from Dr. Ostrow.

FIGURE 2
$^{22}Na^+$ Efflux Measurements*



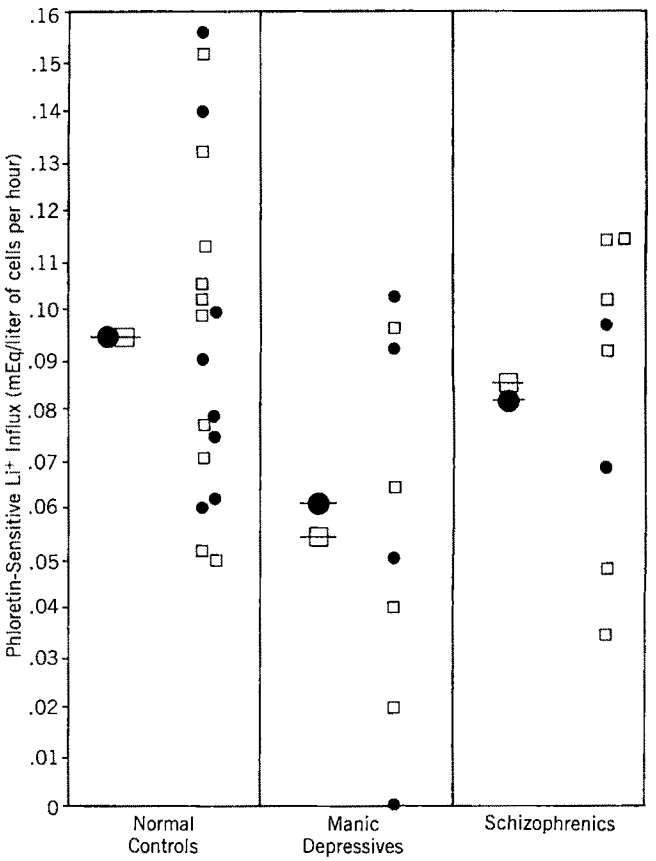
*Each bar represents the rate of $^{22}Na^+$ efflux from cells incubated in media containing the indicated counter-ion, with the solid portion indicating the proportion inhibited by 0.1 mM phloretin, determined using the method of Haas (22).

FIGURE 3
Pedigree for Li^+-Na^+ Counterflow Defect in Patient's Family*



*The patient is indicated by an arrow; data were unavailable for sibling 5. Open symbols=counterflow in normal limits; closed symbols=values outside the 95% confidence interval.

FIGURE 4
Phloretin-Sensitive Li^+ Influx Rates*



*Determined in duplicate using methods described in references 19 and 20. Squares indicate males, circles females. Larger symbols indicate means.

Transport Parameters in Patient Groups

Figures 4–6 show the individual results and group means of the various lithium influx rates as measured in controls, manic-depressives, and schizophrenics. None of these patients was receiving any medication, including lithium, at the time of the study. We observed a decrease in phloretin-sensitive Li^+ influx measured in magnesium chloride medium in the manic-depressive group only (figure 4). Mean ouabain-sensitive Li^+ influx (figure 5) was significantly increased in both the manic-depressive ($p < .02$) and schizophrenic ($p < .03$) groups. We also observed a significant increase in inhibitor-insensitive Li^+ influx in the manic-depressive group, while this parameter, measured in a small group of schizophrenic patients, clustered at the low end of the normal range (figure 6). Unipolar depressives (10 patients) as a group showed no alteration in any lithium transport parameter, while RBCs from unipolar depressives with hypomania (bipolar II, N=7) had as a group increased phloretin-sensitive Li^+ influx ($p<.05$).

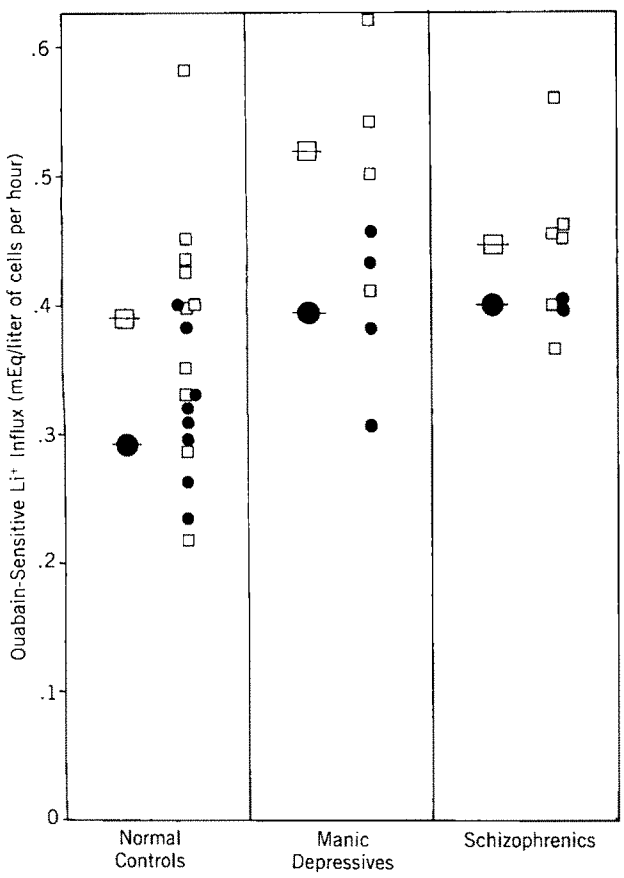
Table 4 gives direct measurements of phloretin-sensitive Li^+-Na^+ counterflow in normal subjects, schizophrenics, and manic-depressives either before or during lithium therapy. Again, manic-depressives, as a group, showed lower average Li^+-Na^+ counterflow than did normal subjects, due largely to the difference between manic-depressive subjects and male control subjects. Female manic-depressives had counterflow rates similar to female control subjects. However, the counterflow level in RBCs from female controls was significantly lower than that in male controls ($p<.005$). Note also that there was a decreased counterflow level in both male and female subjects on lithium at the time of assay. Although the overall group means do not show a statistically significant decrease due to lithium therapy, when the subset of 8 subjects whose counterflow levels were measured both before and during lithium therapy were examined individually, there was a uniform 35%–45% decrease in Li^+-Na^+ counterflow in response to the treatment.

TABLE 4
Phloretin-Sensitive Li^+-Na^+ Counterflow Measurements in Subject Groups*

			Phloretin-Sensitive Li^+-Na^+ Counterflow (mEq Li^+ efflux/liter RBCs·4 hr)								
	Age (years)		Total			Males			Females		
Subject Group	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD
Normal controls	26.4	3.6	30	.279	.092	21	.309**	.076	9	.2[illegible]	.093
Manic-depressives	31.2	12.3	35	.223***	.098	12	.243***	.096	23	.2[illegible]	.099
Manic-depressives on lithium	28.1	9.1	11	.162†	.079	5	.185††	.079	6	.1[illegible]	.082
Schizophrenics	25.0	5.2	10	.258	.083	6	.297	.080	4	.2[illegible]	.052

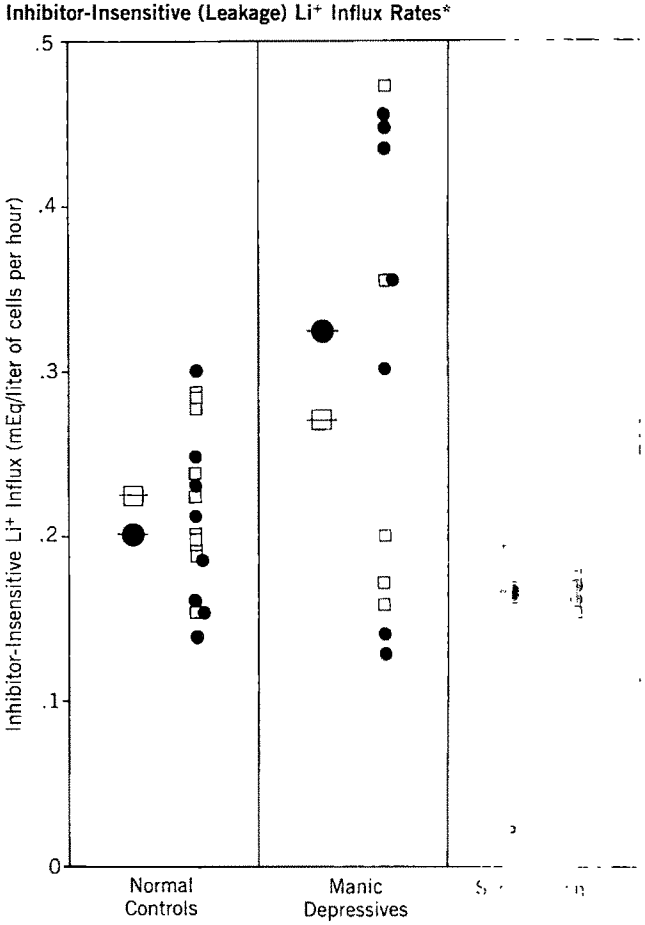
*Values are for duplicate determinations done on 1 or 2 occasions.
**Significantly different from female controls, $p<.005$.
***Significantly different from controls, $p<.05$.
†Significantly different from controls, $p<.001$.
††Significantly different from controls, $p<.01$.

FIGURE 5
Ouabain-Sensitive Li^+ Influx Rates*



*Determined in duplicate using methods described in references 19 and 20. Squares indicate males, circles females. Larger symbols indicate means.

FIGURE 6
Inhibitor-Insensitive (Leakage) Li^+ Influx Rates*

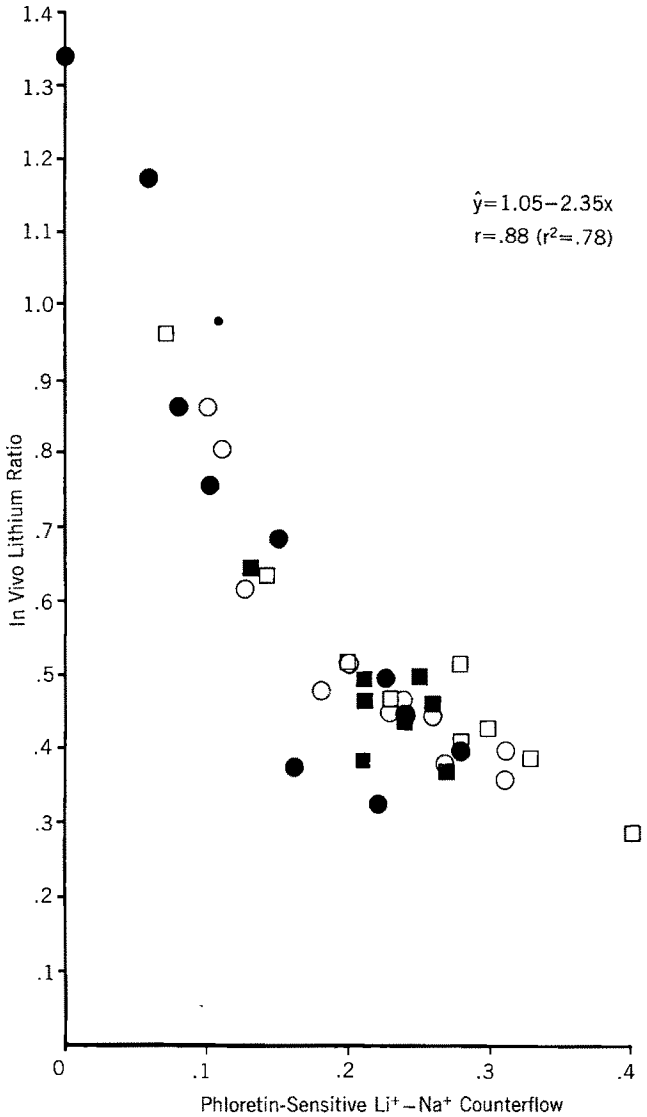


*Determined in duplicate using methods described in refer[illegible]. Squares indicate males, circles females. Larger symbols [illegible]

Correlation Between In Vitro Li^+-Na^+ Counterflow and In Vivo Li^+ Ratio

Figure 7 shows the correlation (r=.88) between the in vitro measurement of RBC Li^+-Na^+ counterflow before lithium therapy and the actual in vivo Li^+ ratio observed during steady-state lithium therapy in our group of manic-depressive patients. Simi[illegible] tions were obtained using both net coun[illegible] phloretin-sensitive counterflow measure[illegible] indicates that although we observed a very [illegible] of values for both phloretin-sensitive Li^+ [illegible] in vivo Li^+ ratio in manic-depressive patie[illegible] predict the intracellular Li^+ level attained [illegible]

FIGURE 7
Correlation of In Vitro Li^+-Na^+ Counterflow Measurements with the In Vivo Li^+ Ratio*



*Phloretin-sensitive Li^+-Na^+ counterflow was measured in duplicate before lithium therapy; in vivo Li^+ ratio was measured 3–10 times during steady-state lithium therapy. Open symbols indicate ISPI patients and closed symbols indicate MRH-MC patients. Circles indicate females, squares males.

py quite accurately based on the in vitro measurement of Li^+ transport before the institution of lithium therapy. This high correlation between in vitro counterflow measurements and in vivo ratio was unique among the transport parameters studied (23), indicating the relative importance of phloretin-sensitive Li^+-Na^+ counterflow as a determinant of the steady-state intracellular Li^+ level at a given plasma Li^+ level. Figure 7 also redemonstrates the existence of a subgroup of manic-depressive patients whose red cells possess an abnormally low degree of Li^+-Na^+ counterflow and hence develop abnormally high lithium ratios on lithium therapy.

SUMMARY AND DISCUSSION

The major findings of this clinical study of lithium transport are as follows:

1. The in vivo Li^+ ratio is determined by the dynamic balance between the phloretin-sensitive Li^+-Na^+ counterflow and lithium leakage pathways.
2. Approximately one-fourth of the manic-depressive patients had a marked deficiency in phloretin-sensitive Li^+-Na^+ counterflow, resulting in markedly increased in vivo and in vitro Li^+ ratios.
3. This defect in phloretin-sensitive Li^+ transport cannot be ascribed to either a plasma inhibitor or cytoplasmic factor but rather is an intrinsic property of the RBC membrane.
4. In the case of the patient and his family we studied in detail, the defect in lithium transport is genetically transmitted in a pattern consistent with dominant inheritance under autosomal control.
5. The only significant sex-related difference in lithium transport was the finding that normal males have significantly higher Li^+-Na^+ counterflow than do normal females. None of the parameters studied showed significant variations according to age or race.

Several questions arise concerning the possibility of a relationship between a heritable defect of lithium transport and manic-depressive illness. Our data and those of Duhm and Becker (24) on normal individuals have demonstrated substantial interindividual differences in lithium transport, and evidence from the twin study method indicates that genetic factors contribute to the steady-state Li^+ ratio in normal individuals (7, 8). Therefore, it is reasonable to assume that there may be substantial genetically controlled variability in the various moieties of lithium transport that determine the Li^+ ratio. Clearly, there is not a one-to-one relationship between bipolar affective illness and a Li^+-Na^+ counterflow deficiency. Our preliminary data have suggested that such a reduction in phloretin-sensitive lithium transport is found in a subgroup of manic-depressive patients and is not characteristic of schizophrenics or unipolar depressed patients. However, we have studied bipolar patients with Li^+ ratios and transport values within normal limits who have had good responses to lithium. The father of the manic patient in this report is an example of an individual with a severe RBC Li^+-Na^+ counterflow deficiency and no previously diagnosed major affective illness. However, this man has periodically and regularly absented himself from his family for periods of 1 week or more over the last 20 years, and three of his five siblings have been hospitalized for episodes of mania or depression. In contrast, there is no evidence of affective illness in any of the mother's siblings or her parents. Of possible significance in this regard is the fact that a 20-year-old woman who is next in this sibship and whose red cells lacked phloretin-sensitive Li^+-Na^+ counterflow (sibling 2) has had a major depressive episode.

Additional work must be done to further define the

genetic mechanism involved in the inheritance of the lithium transport system(s). As a reduction in Li^+-Na^+ counterflow is apparently the mechanism that accounts for the elevated Li^+ ratio seen in some bipolar patients, direct measurements of transport moieties may help us to characterize possible subgroups of bipolar patients. In addition, longitudinal studies of apparently "normal" individuals with lithium transport abnormalities, such as sibling 7 in the family described here, may help us to unravel the relationship between such transport defects and susceptibility to and form of affective illness. The finding by Lyttkens and associates (4) that female patients on lithium have higher in vivo Li^+ ratios than do males can be explained by our finding that, on the average, women have a lower level of Li^+-Na^+ counterflow in their red cells than men do.

We hope that a clearer understanding of the biochemical determinants of the lithium ratio and the opportunity to measure these parameters in a large number of patients before the institution of lithium therapy will help clarify the conflicting data concerning the prediction of clinical response to lithium from the lithium ratio (3, 5, 6, 25–28). Methodologic problems in response studies such as small sample sizes and the lack of fixed-dose methodology render the results of such studies preliminary at best. Obviously only a large multicenter collaborative study of lithium transport parameters in a large number of normal subjects and patients with diverse psychiatric diagnoses can help to clarify these issues.

If there is a relationship between the mechanism and control of lithium transport and the pathophysiology of affective illness, what is the nature of that relationship? Our findings of a defect in lithium transport may indicate the presence of a defect in transport system(s) involved in the transport of physiologically significant ions. It is of considerable importance that the defect of lithium transport that we have described here is a deficiency of the Li^+-Na^+ counterflow system, which is mediated by lithium-sodium exchange. There has been considerable empirical evidence for a sodium transport abnormality in affective illness (29–31), and it has been suggested that a primary defect in affective illness may be an inherited defect resulting in a reduced capacity of the neuronal membrane to remove sodium from the cell (32). One can postulate that such an alteration of the cation-exchange system of the membrane might result in the decreased transport of compounds using pathways which require either the co-transport or countertransport of sodium ions. Catecholamine reuptake systems have an absolute requirement for sodium ions (33), and preliminary data from our laboratories indicate that phloretin is a very effective inhibitor of catecholamine reuptake systems (Javaid, Ostrow, Smith, and Davis, unpublished observations).

The finding of three distinct pathways of lithium transport and the demonstration of genetic transmission of a deficiency of lithium-sodium counterflow have implications for the clinical use of lithium. It has been suggested that the toxicity and efficacy of lithium are more closely related to red cell than to [illegible] ium levels (34). We have shown that subst[illegible] individual variability exists in the count[illegible] way of lithium transport, which may affec[illegible] central nervous system lithium concentra[illegible] even if the abnormality in lithium transpor[illegible] ally related to the pathogenesis of mani[illegible] illness, genetically controlled inte[illegible] ferences may be related to other parame[illegible] the time course of response to lithium the[illegible] tibility to lithium toxicity, and optimu[illegible] quirements for long-term lithium prophy[illegible]

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Am J Psychiatry 135:9, September 1978

BRIEF COMMUNICATIONS

Effect of Alcohol Consumption on State Anxiety Changes in and Female Nonalcoholics

BY PATRICK E. LOGUE, PH.D., W. DOYLE GENTRY, PH.D., MARKKU LINNOILA, M.D., AND C. WILLIAM ERWIN, M.D.

Ten male and 10 female nonalcoholic college students were given drinks having alcohol levels of 0, 0.5, 0.8, and 1.2 g/kg. They were then given four complex psychomotor tests, immediately before and after which they were given the Spielberger State Anxiety Inventory, and asked to rate their performance on a five-point scale. Mean anxiety change scores were −.90, .75, 4.75, and 5.55 for the four alcohol doses, respectively. There was no significant correlation between anxiety change and actual performance on the visual vigilance task, but for males there was a significant correlation between anxiety change and perceived level of impairment.

BOTH FOLKLORE and the bulk of the existing literature on alcoholism suggest that excessive alcohol consumption is a behavior pattern that is acquired and maintained because it reduces social anxiety and physiological tension. However, several recent studies (1–5) have indicated that alcohol may in fact lead to increased anxiety/tension rather than its reduction. Mendelson and associates (2) noted that alcoholic subjects experienced an increase in anxiety and depression during a 14-day period of experimental drinking and that the intensity of negative affect was related to the amount of alcohol consumed, i.e., the more the subjects drank the more anxious they became. McNamee and associates (3) also found a "paradoxical increase" in anxiety in 9 out of 12 (75%) chronic male alcoholics in an experimental drinking situation. Nathan and O'Brien (4) compared the drinking and affective behavior of 4 skid-row alcoholics and 4 matched nonalcoholics and reported that n... experienced reduced levels of anxiety w... On the contrary, the alcoholic subjects sho... ened levels of acute anxiety as soon as dri... whereas the nonalcoholic subjects showed ... no change in anxiety with alcohol.

The evidence that excessive alcohol c... may increase rather than decrease anxiety ... the most part on small sample studies of ... holics; all of the studies have looked at ... anxiety relationship in males and with or... (5) have not explored this relationship in ... subjects. As part of a larger project desig... date the sex-dependent effects of alcoho... behavior, our study examined the effe... consumption at varying dosage levels on ... changes reported by nonalcoholic subject... after performance on a battery of comp... motor tests. The study also examined the ... between changes in state anxiety under t... cohol conditions and both perceived im... performance and actual, objective change... formance.

METHOD

Twenty college student volunteers (10 ... females) served as subjects. They were sc... clude those who were on drugs, had a h... evidence of current mental disturbance, h... of a profound sleep disturbance (narcole... were either heavy drinkers or nondrinke... jects were paid for their participation.

All subjects were introduced to all four ... ditions. That is, they were given drinks ... (placebo), 0.5, 0.8, and 1.2 grams of alc... gram of body weight. The drinks were 95... hol combined with lime juice and tonic wa... served chilled. The amount of liquid inge... same in all conditions (4 dl). The time ...

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drinking was 30 minutes. To allow for even absorption of the alcohol, subjects were asked to fast for the 3 hours before each test session.

The subjects were trained to criterion on a battery of four complex psychomotor tests related to driving behavior: a measure of simple reaction time, a critical tracking task, a continuous performance task, and a visual vigilance task. They were then randomly assigned by means of a Latin square design to the various alcohol conditions. Drinks were administered in a double-blind fashion at the same time each day (3:00 p.m.), with the exception of a fifth experimental condition in which the medium 0.8 g/kg dose of alcohol was administered in the morning (9:00 a.m.) to assess circadian (rhythm) effects. Subjects were given the Spielberger State Anxiety Inventory (6), which takes about 5 minutes to complete, immediately before and after psychomotor test performance. This inventory is designed to measure "a transitory emotional state or condition of the human organism that is characterized by subjective, consciously perceived feelings of tension and apprehension, and heightened autonomic nervous system activity." The inventory requires the subject to respond in terms of how he or she feels at that moment to 20 items such as, "I am jittery," "I feel nervous," and "I feel self-confident." Subjects were also asked to rate their level of performance on the psychomotor test battery on a five-point scale ranging from "very good" (one) to "very impaired" (five) after each testing session.

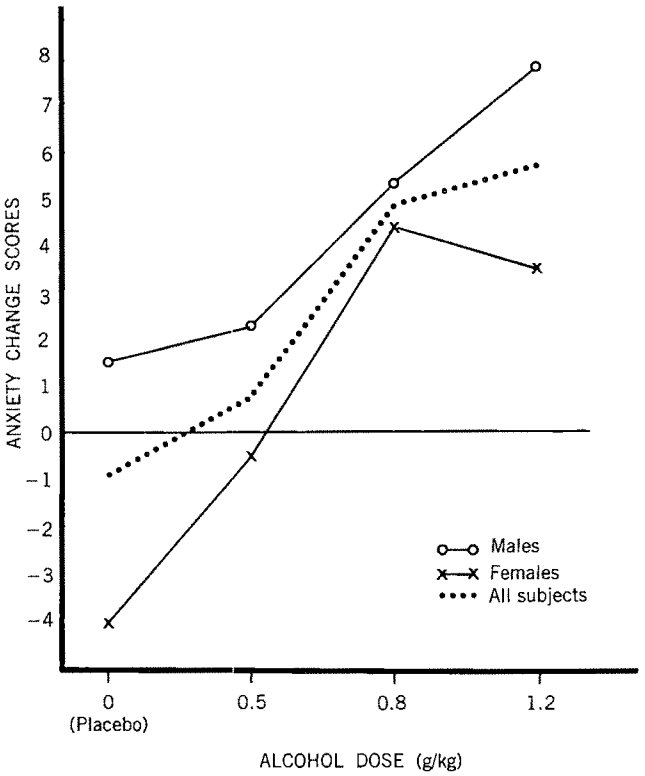
Subjects were tested in the various alcohol conditions at weekly intervals, and all testing took place in a soundproof, air-conditioned room. Subjects were monitored by a TV camera and if necessary were contacted during the experiment by intercom, which presented recorded task instructions.

RESULTS

Anxiety change scores were computed by subtracting the score obtained after performance on the psychomotor tests from the score obtained before testing in each alcohol condition. The baseline anxiety scores obtained for the various conditions for both males and females were well within normal range (6), and there were no significant differences between male (range, 32.5–34.1) and female (range, 34.9–36.9) subjects.

An analysis of variance obtained for anxiety change scores revealed a significant main effect for alcohol dosage level, $F(3,54)=3.59$, $p<.05$. Mean anxiety change scores were −.90, .75, 4.75, and 5.55 for the placebo, 0.5, 0.8, and 1.2 alcohol dose levels, respectively (see figure 1). As determined by a Duncan multiple-range test, no mean was significantly different at the .05 level from that of the next lower or higher alcohol dosage level. There were no significant sex differences with respect to changes in anxiety level across the four alcohol conditions.

FIGURE 1
Relationship Between Alcohol Dose and Change in State Anxiety Score



A comparison of anxiety change scores for the a.m. and p.m. administrations of 0.8 g/kg alcohol dosage by a two-tailed t test revealed no significant effects due to circadian rhythm.

Pearson product-moment correlation coefficients were computed between anxiety change scores and subjects' ratings of their performance following each session and between anxiety change scores and one measure of actual test performance, i.e., on the visual vigilance task, the task most proximal to the second administration of the anxiety inventory. The correlation between anxiety change and perceived level of impairment was $r=.32$ for all subjects ($p<.01$), $r=.06$ for females (n.s.), and $r=.51$ for males ($p<.002$). The correlation between anxiety change and actual performance on the vigilance task ranged from $r=.01$ to .09 and was nonsignificant in all cases.

DISCUSSION

The results of the present study demonstrate clearly a positive relationship between alcohol ingestion and an increase in self-reported state anxiety in a laboratory task-performance situation. This relationship appears to hold for both male and female subjects and to some extent (i.e., with males) seems to be related to the subjects' perceptions of their performance on a battery of complex psychomotor tests. However, whether the fact that one believes he or she is doing

poorly (i.e., rates his or her performance as "impaired") generates anxiety or whether the rise in anxiety serves as a basis for believing that one's performance is impaired cannot be determined by this study. Actual test performance, on the other hand, was unrelated to changes in anxiety level, i.e., how the subject thinks he or she is doing under the influence of alcohol is related more to mood state than to how he or she is actually doing.

Such findings support previous work which has seriously questioned the traditional notion that alcohol is an anxiety-reducing agent (1–5) and with these studies suggest a further examination of the distinction between social drinking, where alcohol may in fact serve to reduce social anxiety and tension, and chronic alcohol abuse (alcoholism), where a vicious cycle of anxiety-alcohol consumption-impaired performance-anxiety may be operating. Our findings further confirm the relationship between the amount of alcohol consumed and the severity of anxiety in evidence (2, 3). They also extend the alcohol-anxiety relationship to female subjects and to nonalcoholic drinkers. In the latter case, these findings do not support Nathan and O'Brien (4), who found no change in anxiety for nonalcoholics in an experimental drinking situation. However, their study examined drinking beh alcoholics from the skid-row area of whereas our study used college student subjects. Thus, the different findings may flect differences in the two study populat

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Phencyclidine-Induced Psychosis

BY R. MICHAEL ALLEN, M.D., AND STEVEN J. YOUNG, M.D.

During a 13-month period, 9 patients with phencyclidine-induced psychosis were admitted to Darnall Army Hospital. They exhibited hostility, agitation, and tangentiality and had delusions of influence and religious grandiosity. Six subjects reported auditory hallucinations, and 4 were disoriented in at least 1 sphere. Despite treatment with antipsychotic medication, the psychotic episodes often persisted for more than 30 days. Our clinical finding of prolonged psychotic reactions, together with previous reports of the effects of phencyclidine, suggests that phencyclidine provides an intriguing drug model for schizophrenia.

IN THE FALL of 1975, psychiatrists at Fort Hood, Texas, saw several cases of acute psychosis precipitated by "snorting" or intravenous injection of a whitish powder sold under the guise of tetrahyd (THC). When analysis of samples of this two different suppliers showed them to centrated phencyclidine (PCP), we bega the psychosis associated with phencyclid ingestion, or inhalation.

PATIENT SAMPLE

We reviewed the charts of all patien from Darnall Army Hospital, Fort Hood.

At the time this work was done, Dr. Allen was Chie was Staff Psychiatrist, Department of Psychiatry U.S. Army Medical Corps, Fort Hood, Tex. Dr. sistant Professor of Psychiatry, Texas Tech Univer ademic Health Center, 4800 Alberta, El Paso, Tex is now Assistant Professor, Department of Psyc State University, East Lansing, Mich., and is in pr Kalamazoo, Mich.

a diagnosis of drug-induced psychosis from July 1, 1975, to May 31, 1976. We also included additional patients admitted in June and July 1976. We selected cases if the patient developed an acute psychosis after "snorting," swallowing, or injecting a white or yellowish-white powder believed by the patient to be THC and descriptively matching two separate samples that were reported to be high grade phencyclidine by the toxicology laboratory of Brooke Army Medical Center.

Patients were selected for study only if they had no previous psychiatric history and had a satisfactory premorbid adjustment.

Our patient population did not include the numerous drug intoxications of very short duration (3–6 hours) handled by the alcohol and drug treatment program. Our study group represented only 25%–30% of the phencyclidine-intoxicated patients referred to the psychiatric service, the majority of whom were discharged within 24 hours. Thus, our sample of prolonged phencyclidine-induced psychoses probably represented less than 5% of case contacts by all of the helping agencies on the post.

Nine patients with acute psychosis attributable to phencyclidine were hospitalized at Darnall Army Hospital during the 13-month study period. We excluded 5 additional patients because they ingested other psychoactive substances (alcohol, amphetamine, LSD, and cocaine); however, their symptomatology suggested that phencyclidine was the agent responsible for their psychosis. The 9 subjects in the study included 8 men and 1 woman, aged 18 to 24. Most had a history of multiple drug abuse, but none gave a history of a prior psychotic episode. Specific signs and symptoms of the case material are presented in table 1.

Characteristically, the patients were agitated and hostile and exhibited tangentiality and circumstantiality. They were preoccupied with religious and sexual topics. Seven of the 9 subjects had delusional ideas, predominantly religious grandiosity ("I am Jesus Christ") and delusions of influence (e.g., being controlled by voodoo). Hallucinations were common, with 6 patients reporting auditory hallucinations. One-third of the group was considered suicidal, and 4 were disoriented in at least one sphere. We did not observe the neurological abnormalities reported by other authors, presumably because hours or days had elapsed from ingestion to hospitalization and neurologic examination.

TABLE 1
Presence of Psychopathological Findings in 9 Cases of Phencyclidine-Induced Psychosis

	Patient Number								
Finding	1	2	3	4	5	6	7	8	9
Duration of psychosis (days)	6	31	60+	90+	9	30+	13	7	16
Behavior									
Agitated		x	x			x			x
Hostile/violent		x	x			x		x	
Psychomotor retardation				x					
Catatonic posturing			x						
Disrobed in public			x					x	
Eloped from ward			x			x			
Affect									
Euphoric		x				x	x		
Depressed	x								
Anxious/frightened	x	x			x				
Labile		x				x			
Inappropriate				x				x	
Thought process									
Mute			x						
Pressured speech		x					x		
Tangentiality		x	x	x	x	x			x
Blocking				x					
Thought content									
Preoccupation									
Religious		x	x		x		x		
Sexual		x		x	x				x
Delusions									
Religious		x		x			x		
Paranoid			x	x					
Of reference	x								x
Of influence	x	x			x				
Hallucinations									
Visual		x	x						
Auditory	x	x	x	x	x				x
Tactile						x			
Suicidal ideation	x			x	x				
Sensorium									
Disorientation to place		x							x
Disorientation to date		x			x	x			x

RESPONSE TO TREATMENT

All the subjects were treated with antipsychotic drugs, chiefly haloperidol. Only patients 1, 5, 7, and 9 made a full recovery and remained free of psychotic symptoms off medication at follow-up, which varied from 6 weeks to 6 months after discharge. One week after discharge, patient 2 was readmitted with a recurrence of his symptoms after having stopped taking haloperidol and begun ingesting PCP again. His symptoms gradually cleared in 18 days on haloperidol, and treatment with depot fluphenazine was begun as a preventive measure.

Patient 3 showed no improvement after 3 weeks despite taking 800 mg of chlorpromazine and 30 mg of haloperidol daily. She was then referred to a private hospital where her psychosis cleared with 7 electroconvulsive treatments. After release from the hospital the patient took "acid" and was readmitted, according to the private hospital summary, "psychotic and bizarre," a week later. Again she responded to ECT but remained withdrawn and blunted in affect a month after the last treatment.

Three months after his initial admission, patient 4 improved on thiothixene. However, he had not recovered sufficiently to return to duty and received a medical discharge from the Army. Patient 6 still had loose associations, visual hallucinations, and formication one month after admission, despite receiving 20 mg haloperidol per day. Although patient 8's toxic psy-

chosis seemed to clear completely, he developed a depressive schizo-affective psychosis 1 month later when placed in confinement for legal problems.

In summary, the patients who recovered did so gradually. The duration of their psychotic episodes ranged from 6 days to over 90 days in one patient who had still not improved at the end of the study period. Treatment with antipsychotic drugs was generally ineffective. (One patient responded to ECT.) Treatment by sensory deprivation or benign neglect, although mentioned in several articles (1, 2), was rarely helpful to our subjects.

DISCUSSION

Phencyclidine (phencyclohexyl piperidine, PCP) was developed as an intravenous anesthetic and is still used for that purpose in veterinary medicine under the trade name Sernyl. It produces excellent analgesia without respiratory or circulatory depression (3), but patients given the drug may exhibit agitation and bizarre behavior in the postoperative period (4).

Luby and associates (5) and Davies and Beech (6) observed a consistent syndrome of neurological and psychological changes in normal subjects given .075 to .1 mg/kg of phencyclidine intravenously. The neurological changes included nystagmus, ataxia, and diminished pain, touch, and position sense. The first psychological symptom to appear after drug administration was a distortion of body image, followed by feelings of estrangement, disorganization of thought including tangentiality and blocking, hostility and negativism, and, finally, apathy. One subject became "catatonic" (awake but unresponsive to questions) for 23 minutes (6). All these acute symptoms subsided within 1½ hours. Four chronic schizophrenic patients given the same dose of PCP showed similar responses, with worsening of their thought disorder, increased affect, sexual acting out, and hostility that persisted for 6 weeks (5).

Phencyclidine has been shown to be a constituent of street drugs in 14%–50% of samples analyzed (7, 8). Hart and associates (7) found that 10 of 11 samples of putative THC submitted to various agencies in Lawrence, Kans., contained phencyclidine as the only psychoactive agent. PCP is also commonly sold as mescaline, psilocybin, cocaine, or angel dust. Contributing to the prevalence of PCP in the drug culture is the ease with which it can be synthesized in illicit laboratories.

Rainey and Crowder (1) reported 3 cases of schizophreniform psychosis precipitated by street drugs sold as PCP, THC, and methadone but all believed to be phencyclidine. All 3 of their patients were agitated, uncooperative, and disoriented to place and date. Two of them had auditory hallucinations. When the psychoses cleared in 2 to 4 weeks, all 3 patients had amnesia for the episode. A single patient described by Stein (2) displayed similar symptoms for 10 days but retained partial recall for the episode.

Psychological and motor test data fr... studies indicate that PCP mimics the p... toms of schizophrenia after the acute ... have subsided (9–11). Our experience con... observations. Our patients' reactions to p... resembled the effects of PCP on chronic ... ics (3) in duration and symptomatology. ... our subjects were hyperreligious, delusio... nating, and often disoriented. Thus, in our ... perience phencyclidine psychosis often ... acute schizophrenia. Although we tried to ... tients with preexisting or underlying psy... orders, we cannot completely exclude th... that PCP uncovered or activated an und... chotic process. In any event, PCP was clea... important variable in their clinical course.

CONCLUSIONS

One could contend that PCP provides a ... model for schizophrenia than amphet... cyclidine produces a more varied clinica... various categories of schizophrenia (cat... noid, hebephrenic) than does amphetamine. ... Like amphetamine, PCP clearly exacer... phrenia, as noted in previous references. ... effects of PCP appear to be more potent ... lasting than those of amphetamine (3). ... phencyclidine on dopamine systems has ... studied, but such studies should prove ...

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When the Psychotherapist Is Black

BY JESSE O. CAVENAR, JR., M.D., AND JEAN G. SPAULDING, M.D.

The authors present a case report of a white neurotic man treated in long-term psychoanalytic therapy by a black woman psychiatrist. The defense mechanism of reversal—the therapist was white in the patient's early dreams—was evident not only in this patient but in several other white patients treated by the black therapist. The authors suggest that, contrary to the opinions of a few other authors, the reality issues of racial differences can be dealt with successfully in this kind of interracial psychotherapy.

THE LITERATURE on psychoanalysis and psychotherapy when the therapist is black and the patient is white is very meager. There are markedly divided opinions as to the effect this variable may have on the treatment process.

Oberndorf (1) suggested that interracial therapy cannot be effective due to the psychological biases that are present and implied that such treatment should not be attempted. However, Schachter and Butts (2) stated that racial differences "may have a catalytic effect upon the analytic process and lead to a more rapid unfolding of core problems." Other reports in the literature, such as those of Bernard (3), Calnek (4), Grier (5), Kennedy (6), and Waite (7), fall between these two extremes.

Fischer (8) stated that "racial differences between analyst and patient involve issues of unconscious meaning at many levels. These issues and meanings must be recognized and utilized, for there are serious hazards in either overestimating or in ignoring them." Fischer further suggested that the black-white difference is a visible structure on which infantile fantasies may be projected. He concluded that "to ignore the manifest black-white issue is to avoid a piece of the patient's and therapist's everyday reality. To become overly invested in this apparent interracial content, however, represents an effort to deny and negate the deeper intrapsychic conflicts."

Curry (9) suggested that there are important differences in the transference, resistance, and working-through process when the therapist is black. He referred to a complex set of issues he termed "pre-transference." This pre-transference is due to the fact that the therapist does in reality have a characteristic that can mobilize unconscious fears and fantasies, i.e., black color. These fears and fantasies may be unrelated to the individual's past life and are therefore not transference phenomena per se. Curry suggested, as have other authors, that "black" may unconsciously represent bad, evil, darkness, and inferiority. These same fears may be represented in mythology by Satan, Lucifer, or Judas.

Curry implied that this pre-transference reaction may be used as an effective resistance to the formation of a transference. When the racial issue becomes a point of attack on the therapist, it is because of these unconscious fantasies and the anxiety generated by the fantasies. Curry concluded that if the patient's own conflicts become interlaced with "the patient's hypercathexis of a reality situation," i.e., the therapist's blackness, an insurmountable resistance may form that precludes treatment and leads to acting out. Effective interracial psychotherapy is thus possible only if proper attention is directed to these complex pre-transference, transference, and resistance issues.

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We became interested in this racial variable in long-term psychoanalytic psychotherapy involving white male patients and a black female psychiatrist. We were also curious as to whether dream productions would be affected by the racial differences.

It has long been a clinical observation in psychotherapy and psychoanalysis that the patient's dreams frequently disguise a white therapist as a black person. This is most commonly noted in the early and middle phases of treatment as a defensive maneuver and usually ceases as the patient's defenses are lowered. Altman (10) reported a dream in which a white analyst was disguised as a black person via condensation, displacement, and symbolization. We wondered whether the same defensive maneuvers would apply when the therapist is black; that is, would the black therapist be represented as white in an attempt to disguise transference feelings?

The purpose of this paper is to report a case of a white male neurotic patient treated by a black female therapist in which these variables of interracial treatment and dream productions could be studied in detail. This case is representative of several long-term patients treated under similar conditions; all cases illustrated the same principles.

CASE REPORT

The patient was a 25-year-old single man who came for psychiatric evaluation because of marked free-floating anxiety, multiple phobias, and an inability to urinate in public places. He had been born and raised in a small southern town; his parents were from the middle class. He was the second of a set of nonfraternal twins and had one sister who was two years older. He described his father as a sadistic, angry man who was emotionally distant from the children and his mother as a seductive woman who frequently walked around the house and yard nude in view of the children. She bathed the patient until he was in early adolescence. When the patient was 13 years old his mother and father separated briefly. The mother took only the patient with her when she left; he slept with her until they returned home. The father accused the patient of having seduced his mother, and his inability to urinate in public places dated from that experience.

We felt that the patient had an obsessive-compulsive personality with hysterical and phobic features. His main defenses were isolation of affect, reaction formations, repression, and displacement. Psychoanalytic psychotherapy was judged the treatment of choice.

The patient presented his first dream in hour 6. His early feelings about the therapist were well disguised by condensation, displacement, and symbolization. He reported, "The dream occurred at a place I had never been before. It seemed to be a church and I went to a classroom with 15 other people. I knew no one: it was a class on vectors. There was a girl to my left, two seats down from me; she looked over and smiled. It made me feel good, and I had a sense of well-being. After the class I walked by a restroom, and I knew I could go in and urinate without any tension." The girl in the dream was white.

The strongly erotic nature of the evolving transference was indicated in a dream reported in the 10t... on the campus of the university for a summe... go to the athletic field for the field trip; one b... are girls, and a woman instructor is there. I f... the field trip; after the class broke up, the ... gested to me that we have a separate meeting ... denly I didn't want to be there; I became c... urination. I saw two men who had been in a ... and they started to shove snow into their pan... and packed my pants full of snow and walke... instructor." In this dream again there were ... contrast, the white snow seems to distract fr... of black.

The patient presented the first dream in v... pist appeared undisguised—except for race ... hour. "I was in the basement of the hospital. ... do electrical work in the morgue. While wai... vator, I saw you and two other people about ... felt anxious, and you appeared to be agitated ... me. I said, 'I'm here to work in the basem... shook hands with me. I saw outside, to the be... tal. Some windows had been broken, and I ... dalism. Somebody is pretty sorry.' I saw a m... gressive and confident; I felt that was the v... be."

Two weeks later the patient brought the fo... "I was going to my parents' home in the co... by a house where blacks lived; children were ... yard. All the black families have ten kids w... The black family greeted me; they always see... Their house had garbage around it; most of the... was surprised when I got to my house, becau... shack, too, like the house in which the blac... dream occurred 5 weeks before any verba... by the patient of the fact that the therapist w...

In the 37th hour, the patient reported a... was lying on a couch with a woman. She wa... advances; she was affectionate, and snuggle... was not in a motherly way. Her husband can... was concerned that he might notice this. She ... had long black hair that I stroked." He had ... ciating to this dream material and stated that ... any black women. The patient persisted in h... knowledge of a black woman. When the thera... fronted him with the fact that she was black ... denial, anger, and attempts to make her Vie... waiian—"anything but black."

Over the subsequent hours the patient's cas... intensified. He had many dreams of unsh... dressed in dirty, torn clothing, and many o... cerned janitors and servants. The patient d... as a servant or slave in relationship to his sup... for several hours. After this, the material ... open discussion of his prejudice toward bla... lowing a lengthy period of examining his or... openly and said, "It's unjust for me to be p... let it influence any feelings I may have tow... the first black person with whom I've ever ... I've looked for things about black people t... about. If one did something that I felt was im... generalize to all black people. I don't like the ... don't like myself, I guess."

As the projections by the patient were gr... through, it became clear to him that he actu... downtrodden, oppressed, and in other ways ... perceptions of blacks. This phase of the tre...

illustrated by the following dream, reported in the 74th treatment hour: "I went to this black woman's house. It was you. A picture of your mother was there. I thought, 'Her mother does look black after all.' Then my mother, father, brother, and I were sitting around a table. It was as though we were more black than white."

Over the following 18 months, an intense erotic transference neurosis developed. This transference neurosis was worked through as any other would be; no alteration of technique was made necessary by the racial differences. The patient experienced good symptomatic relief of his phobias and urinary problem and a marked lessening of his anxiety. He began to date girls for the first time in his life, his relationships with both black and white authority figures and peers improved, and he began to take several courses to further his education.

At the end of 3 years of therapy, the patient began to bring typical termination dreams. One such dream was the following: "I was standing on an island and the water began to rise. I needed shelter, so I went to a nuclear fallout shelter. You were there with a group of people. I needed to urinate, but there was a man occupying the bathroom. I needed to go and find my own bathroom. I went into the woods to urinate: I wasn't uptight at all. I saw a woman in the distance, and I knew I could talk with her." We felt that this dream was like those presented by Oremland (11) in which the therapist, undisguised, is involved with an initial symptom that brought the patient to treatment. The dream appears to indicate resolution of these symptoms. This patient's psychotherapy went on to a successful conclusion several months later; no additional resistance was encountered.

COMMENT

The case reported here is one of several long-term psychoanalytic psychotherapy cases we have experienced in which the therapist was black and the patient was white. We believe that interracial psychotherapy can be as productive as any other psychotherapy. Certain different features must be recognized and dealt with accordingly.

The dreams of a white patient will be different in that the black therapist may be disguised as white. The defense mechanism involved is reversal, and it appears to work in the identical manner as when the therapist is white and disguised as black. Clearly it is an attempt of the dream work to deal with anxiety. In our experience, it is common for reversal to occur in dreams; in fact, we would be concerned about the integrity of ego functioning if it did not occur. In our patients this reversal has continued to appear in dreams until a sufficient number of the conflicts have been worked through to permit the therapist to appear undisguised in dreams. A transference of sufficient intensity must also have formed; in the absence of this transference there would be little intrapsychic reason to cathect the therapist sufficiently to dream of him or her.

In our experience, the dreams in which the black therapist was disguised as white revealed little overt anxiety. When the reversal disappears as a defense mechanism and blacks first appear undisguised in dreams, overt anxiety is most often present. In the dreams this anxiety may be noted in the manifest content by the blacks being large, hostile, threatening, or even murderous. Whether the anxiety is greater than in the usual situation, when the white therapist is dreamed of as black, we cannot say with any degree of certainty. Our impression, however, is that the anxiety in the patients we treated was greater.

Another common finding when the therapist is black is the tendency of the white patient to overidentify with the therapist's blackness. All of the patients whom we have treated have done this to some degree; it is our impression that patients who have low self-esteem, are passive, and feel oppressed and constricted intrapsychically overidentify more readily with the black therapist.

The overidentification with the black therapist may have many meanings. Most often, in our experience, it is a hostile identification, a way of appearing compliant and complacent but expressing hostility. In some patients, what appears to be an overidentification is not an identification at all but a massive projection of the patient's unconscious conflicts. The patient merely identifies with what he or she believes the black therapist's plight or station to be, when in fact it is totally distorted.

Although the same conflicts must be worked through in this kind of interracial therapy as in any other treatment, the sequence of the unfolding of the conflicts may be altered by racial issues. The differences in race may serve as a scaffold for multiple projections by the patient. Projections pertaining to race must be dealt with early in the treatment by helping the patient to appreciate that these are unconscious conflicts that the patient is experiencing. Our experience suggests that once this is accomplished the transference neurosis will develop as it does in any other treatment process and the patient can proceed to a satisfactory working through and termination. Although other authors have reported that the reality issues of racial differences cannot be adequately dealt with in some patients, we suggest from our experience that this is not the case.

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Psychiatric Training for the Primary Care Obstetrician-Gynecologist Resident

BY PRISCILLA DAY BOEKELHEIDE, M.D.

The author describes a model for the psychiatric training of obstetrics-gynecology house staff members who serve as primary care physicians for women. By integrating psychiatric skills with specialty training, the psychiatrist can enhance continuity of patient care for the resident during his transition to the role of primary care obstetrician-gynecologist.

WELL BEFORE THE 1975 position statement by the American College of Obstetricians and Gynecologists and the American Board of Obstetrics and Gynecology on the role of the obstetrician-gynecologist in the primary care of women (1), I was invited to share a woman's point of view with the obstetrics and gynecology house staff of a large inner city nonprofit hospital who viewed themselves as total physicians offering comprehensive continuity of care to women. The house officers already recognized that they were often the first medical contact for their patients, that their responsibility should not be limited to acute illness, and that normative life crises required diverse skills such as marital, pregnancy, and sexual counseling, menopausal care, and supportive therapy for the cancer patient. The keys to their development as primary care physicians were their eagerness to learn and to accept the role and the use of a peer collaborative psychiatric consultant on their team.

In addition to the senior psychiatric consultant, the house staff included a teaching-atten three residents, two interns, and one students. No grant or special exter tributed to the program, and most obst cology teaching situations could re shaped toward the same goals. My acce program was probably enhanced by m a fellow resident but also a Fellow o College of Obstetricians and Gynecol

The mutual academic goal was train tient care by increasing understand n focus was often broadened to includ ents, her husband or boyfriend, her ch grandparents and neighbors if neces

METHOD

The tools were the staff's medical b empathy, and their eagerness. Throu mal discussion and demonstration we lowing areas over a period of 20 mon techniques, 2) selection and use of psy 3) personality diagnosis, 4) psychosoc patients' everyday lives, and 5) health medical illness. This focus offered a un obstetrician-gynecologist as a speci practice preventive mental health car consideration was on the doctor-pat and on the chief drug, tincture of doc Balint (3).

The psychiatric consultant guided t air of mutual inquiry. As early as feas trist helped to sort out the majority o the staff could handle themselves a advised as necessary. In most of th stances there was joint observation a few patients were referred for furthe tion. When staff anxiety was hig

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knowledge such as legal involvement was required, direct consultation with the patient was available. Occasionally there were conflicting opinions, e.g., between traditional medical/surgical and psychiatric opinions about a hysterectomy.

CLINICAL VIGNETTES

Some examples that typify primary care throughout the life cycle follow:

1. A young father brought his newborn for a circumcision check. His anxiety offered the intern an opportunity to discuss his feelings as a new father and as a recently neglected (as he saw it) sexual partner.
2. A mother brought her 10-year-old pubescent daughter to check her vaginal discharge, asking covertly for sex education help.
3. A grandmother accompanied her pregnant, unmarried adolescent granddaughter. Social issues of abortion, placement, and illegitimacy were discussed.
4. A young, single, nulliparous, feminist insisted on a hysterectomy, challenging the house officers' value systems.

In addition, the overburdened abusing mother and the frankly psychotic pregnant woman required application of learned medical skills as well as the peer consultant's specialized viewpoint to meet the patients' psychosocial-medical needs. The aim was to keep the personal physician involved as much as possible, encouraging him/her to participate by using interviewing, diagnostic, and treatment skills as needed for brief supportive psychotherapy, Masters and Johnson's or Seman's techniques, etc.

RESULTS

As the house staff reported progress with their patients, they were encouraged to be more flexible in having patients return according to psychological needs; e.g., the postpartum patient who was depressed was seen briefly but frequently rather than for the usual one visit at four to six weeks. The staff learned quickly to focus on the emotional problem, not necessarily the presenting obstetrics or gynecology problem. As a result, some important changes occurred:

1. Labeling of patients changed from "that hysteric" to "Mrs. A," and family dynamics were examined.
2. In selected instances various social services, the pediatrics department, or the occupational therapy section helped in the functioning of the obstetrics-gynecology service and hence in training of its staff.
3. Interdepartmental practices were examined for therapeutic interaction.
4. "Nuisance" calls from patients declined dramatically as the house officers learned to meet dependency needs more directly in the clinics.
5. More appropriate referrals for psychiatric consultation were made, reflecting a focus on prophylaxis and preventive care.
6. Staff time with patients became more productive and the house officers became conditioned gradually to what Burnham (4) calls "practice shock," i.e., the pleasure of treating all types of patients instead of only the unusual, esoteric, or very difficult.
7. The usual clinic diagnostic categories such as toxemia, unwanted pregnancy, and imperforate hymen were redefined to include such concepts as anxiety, dependency, ambivalence, and nonconsummation of marriage.
8. Problem orientation broadened to include the patient's family, body image, and sexual identity.

DISCUSSION

Many demands are placed on the obstetrician-gynecologist when he/she changes from the omniscient and omnipotent authoritarian figure and becomes a true participant in primary health care delivery and service for a large segment of the population. As the primary care physician for women he/she must be prepared to offer periodic comprehensive evaluation of their health status, which includes health education, preventive care, health maintenance, referral to and integration of other health services, and traditional therapeutic services throughout the life cycle of his/her female patients.

Sound psychiatric interventions, once thought to interfere with the busy clinician's schedule, can be integrated into the usual 10- to 20-minute patient visit. This addition makes clinical orientation a more relevant experience for both patient and physician. Through teaching and example during the residency years the psychiatrist can aid this transition from obstetrician-gynecologist to primary care physician.

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Psychiatric Consultation in an HMO: A Model for Educatio Primary Care

BY LOUIS F. RITTELMEYER, JR., M.D., AND WILLIAM E. FLYNN, M.D.

The authors describe a program based on a biopsychosocial model of illness, which uses a liaison-consultation approach in an outpatient setting. It is directed toward integrating psychiatric education and services with primary care in a health maintenance organization. This program can serve as a useful model for studying the validity and effectiveness of integrating psychiatric concepts with primary health care.

PSYCHIATRY'S INVOLVEMENT with the education of primary physicians has gone through an evolutionary process since its inception about 30 years ago. Stimulated by the need for psychiatric manpower during World War II, it has developed into organized programs for medical students, practicing physicians, and residents in other fields, principally family medicine (1, 2).

For four years the Georgetown University Department of Psychiatry has provided psychiatric educational and clinical services for a university-sponsored health maintenance organization (HMO). In this paper, we will focus on two aspects of this experience: 1) the conceptual model used in designing and carrying out the psychiatrists' roles and responsibilities; and 2) the methods employed in the educational programs for primary care physicians and nurse practitioners and for psychiatry residents assigned to provide services to the Georgetown University Community Health Plan (GUCHP).

THE SETTING

When the program for GUCHP was begun in July 1974, there were two centers providing patient care—one in an inner-city neighborhood in the northeast part of Washington, D.C., the other in a "new town" in Virginia, located more than 20 miles from the Georgetown University Medical Center. The been in operation for about 1½ years. ture of fee-for-service patients and erro paid plan. (In 1975 a third center was op cal middle-class suburb in Maryland fourth opened in a suburban site in V

Primary care is provided by teams physician (internist or pediatrician), a r er, and support personnel. Patients specialized care are referred to outside so, several specialists work part-time provide obstetrical and gynecological chiatric care is provided in each cente ric social worker and psychiatry resi supervision of Department of Psychia ferral may be made to private psychi view by the center's psychiatric consu

As the only full-time mental heal each center, the psychiatric social work psychiatric services including the sc consultants and psychiatry residents. will be discussed later.) He or she also mary caregivers by evaluating patie individual, group, and family psyc serving as liaison with outside pra agencies.

THE MODEL

The educational program was based o tions that 1) the primary care teams a resources for comprehensive care to pa ing those with emotional problems, 2. titioners require continuous in-servic consultation to provide optimum care f significant psychosocial dysfunction. chiatrists involved require an unders close working relationship with prima order to function effectively in this sc sumptions were shared by the presider primary physician, and his support of t been fundamental to its development.

In addition, we assumed that the contract between GUCHP and its en the doctor-patient relationship in signifi defined ways (in contrast to fee-for-se and that differences in the expectations and patient could be anticipated.

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The psychiatric program applies a liaison-consultation approach in an outpatient setting. It uses current concepts of psychosomatic medicine, as described by Lipowski (3), to help primary physicians in "assessing the relative contribution of psychological, biological, and social factors to the development, course, and outcome of physical and psychiatric disorders." The consultant supports the primary practitioners in caring for even their most troublesome patients, i.e., the anxious, depressed, angry, and hypochondriacal. He/she functions as "an integral component of the general health care structure rather than a separate [mental health] service," a role similar to the one defined for psychiatrists in the New Haven HMO (4).

This approach derives from a belief in what is now termed a biopsychosocial model, which has been described in detail by Engel, who summarizes its essence clearly: "To provide a basis for understanding the determinants of disease and arriving at rational treatments and patterns of health care, a medical model must also take into account the patient, the social context in which he lives, and the complementary system devised by society to deal with the disruptive effects of illness, that is, the physician role and the health care system" (5, p. 132).

METHOD

Instruction of primary physicians and nurses is similar in most respects to that used in continuing education and in family practice residency programs here (2, 6) and elsewhere (7–9). Regularly scheduled consultations, with a case-oriented focus, provide the basic framework. In addition to discussions of individual patients and families, patients are interviewed and selected articles are reviewed.The referring physician is present when the consultant interviews a patient; this emphasizes the consultant's role as a teacher.

During consultations, which are held at least once weekly in each center and last 20–30 minutes, primary practitioners meet with the consultant to discuss new patients and those who need follow-up by the primary practitioner. In the course of a typical three-hour consultation, 6 to 10 practitioners (and occasionally administrative personnel) are consulted with, individually or as patient-care teams. These are working sessions in which decisions on patient management and disposition are made and the psychiatric social worker participates.

The tone of the conferences is informal. One physician might use several brief anecdotes to obtain the consultant's help in understanding a problem; another might give a detailed case history. There is an open-door policy allowing staff members to enter and leave without disturbing the flow of discussion and permitting all to learn from the experiences of each one. There is also an atmosphere of goal-directedness. Everyone present is interested in working on patient-care problems. Even when discussions focus on staff issues, the objective remains that of better serving patients.

Another feature of the sessions, most apparent in the center located in the "new town," is the mutual value placed on the time spent together. The physicians coined a term, "psych time," to designate the period that each of them meets with the consultant. This time is jealously protected, and patient appointments are seldom allowed to intrude on it. This attitude developed slowly, but over a two-year period became a part of the center's subculture. Now it so pervades the center that newcomers are expected to fit into the routine, and most have little difficulty doing so.

Cases presented to the psychiatrist lead to recommendations that follow one of three general patterns: 1) suggestions for continuing evaluation or care by the primary care team; 2) further evaluation by the social worker or resident; or 3) referral for treatment by one of the mental health professionals. Acceptance of patients for psychiatric evaluation or treatment does not end the patient's relationship with the primary care team, which continues to render other necessary care.

Training is also provided for psychiatry residents; it is intended to meet the increasing demand for collaboration between psychiatrists and primary physicians. This training is designed to facilitate the integration of psychiatric services in an HMO, which requires a different orientation and additional skills that often are not emphasized in other training settings (10).

Residents are assigned to the centers in six-month rotations. Their training is centered around case consultation and brief outpatient therapy (the limit on visits provided under the prepaid plan is 20 per year). The residents' work is supervised by the consultant.

CASE REPORTS

The kinds of problems addressed run the gamut of those seen in a busy ambulatory practice: anxiety, depression, marital conflict, alcoholism, emotional disturbances of children and adolescents, and a variety of diagnostic and management problems with chronically ill patients. How some are dealt with can be seen in these examples.

Case 1. A 49-year-old divorced woman complained of severe pain in her feet. Although this symptom had existed for only a few weeks, she had a history of multiple complaints, difficult interpersonal relationships, and an inconsistent work record. A thorough workup, including consultation with two orthopedic surgeons, failed to establish a diagnosis of any pathology. Treatment had consisted of numerous medications to relieve pain, none of which was effective.

The consultant considered this to be a case of hypochondriasis and recommended a supportive regimen consisting chiefly of regularly scheduled visits to the center, with the care-giver offering his time (usually 15 minutes), attention, suggestions for reestablishing relationships with friends and nearby family members, and encouragement to regain her

functional ability to work more productively. Initially she was seen weekly, later biweekly. The consultant's involvement continued for about three months, when the practitioner, a physician's assistant, had the confidence to carry on without further help.

Case 2. A 5-year-old girl had recently begun soiling herself following the birth of her only sibling. The pediatrician was prepared to deal with the child's reaction to her new sibling, but the parents insisted on a more detailed organic workup. It further developed that these parents had acted similarly in another health crisis (involving care of the newborn), and the physician expressed disappointment that the family had not learned from that experience. The consultation focused on the dynamics of the parents' need to urge further testing. The physician found it helpful to identify and understand the pattern of the parents' behavior and to be supportive in his dealings with them.

The model for this approach has been described by Kahana and Bibring (11). It is used extensively in helping the primary practitioners develop an approach to their more difficult patients.

Case 3. A physician's assistant presented the case of an alcoholic woman who made repeated demands for minor tranquilizers but refused treatment for her alcoholism. The consultant offered the assistant some basic information about alcoholism, Alcoholics Anonymous, and the use of disulfiram (Antabuse).

One month later the patient had not attempted to stop drinking, but she was not demanding tranquilizers and was considering a visit to A.A. The physician's assistant was encouraged to go to an A.A. meeting himself so that he could discuss A.A. more knowledgably with the patient. Subsequently, the patient stopped drinking and began attending A.A. meetings. The likelihood of relapses was discussed, and the practitioner continued to follow the patient with a more comfortable sense of the role he could play in the woman's care.

Case 4. An internist presented the case of a 32-year-old woman who wanted to see him for counseling on a regular basis. She had undergone intensive psychotherapy previously but felt she had not received much help and wanted to try again with this physician. The patient's wishes and expectations were discussed with the psychiatric consultant, which helped the physician realize the unreality of her request. He was able to be supportive to the patient and at the same time set clear limits as to his availability for counseling.

COMMENT

We believe that this program is integrative in two important respects: 1) it combines psychiatric in-service training and consultation for primary practitioners with the service responsibilities of the health center; and 2) it integrates psychiatric services and the training of psychiatry residents with the general health care offered in an HMO.

For this integrative effort to be effective, the consulting psychiatrist and the social worker play key roles. The psychiatrist must have a conviction that the assumptions on which the program is based are valid, and he must be prepared to demonstrate its usefulness to skeptical and, at times, reluctant primary physicians. The social worker must possess the organizational and interpersonal skills to coordinate psychiatric patient-care and educational activities as an integral part of primary care services.

Our experience has borne out the assumptions with which we began the program. It is clear that primary physicians can function well in giving comprehensive care to patients, including many patients with emotional problems. These physicians require inservice training and consultation on a continuing basis and in most instances respond positively when it is offered to them.

At this time, documentation of these conclusions is anecdotal. With more experience and after review of the continuing modifications that will result, a more quantitative assessment will be possible. For the present, it is enough to know that the assumptions appear to be valid and further assessment of them is justified. We can draw some tentative conclusions about the differences between this kind of program in an HMO and a similar one offered to physicians in fee-for-service practice. The receptivity of the physicians is significantly greater than when postgraduate seminars were offered to practitioners in the Washington, D.C., area several years ago (2). In contrast to the fewer than 5% of physicians in general who avail themselves of such continuing education opportunities, those in the HMO actively participate with few exceptions. (Regular attendance—i.e., at least every other week—varies from virtually 100% in one center to more than 50% in the other two.)

Several factors must be considered in understanding this response. The consultations are convenient, i.e., they are conducted in the center itself, and no travel is involved. In addition, there is no personal financial sacrifice by the physicians to participate; it is considered part of his working day. Another factor that also appears to play a part is the limited options for both patients and physicians to "shop" when one or the other encounters difficulty in the doctor-patient relationship. Patients have only three or four choices of primary physicians; the physicians are obliged by the HMO contract to provide services to enrolled patients. This limits the physicians' ability to avoid unpleasant patients, and consultation becomes a way of dealing with such patients more effectively and with less frustration.

We believe that the consultation approach taken by the psychiatrist is crucial and that the liaison-consultation model described has been significant in making the program work. Two experiences in GHCHP support this belief.

Before the initiation of this program, a part-time psychiatrist provided consultation in a traditional manner for 1½ years. Neither he nor the social worker was ever fully integrated into the functioning of the centers, there were fewer diagnoses of psychiatric prob-

lems, and little attention was paid to management of emotional disorders by most of the primary physicians.

In one of the centers, the liaison-consultation approach was abandoned temporarily (for reasons beyond our control). During that time and for many months after, integration of services and a collaborative approach to patient care were difficult to achieve. This was true despite the center medical director's concurrence in our approach and his own skillful use of it in caring for his patients.

Finally, the psychiatric consultants' administrative role is significant. Decisions on patient care are made jointly, with primary physician, social worker, and consultant each contributing his own expertise. Referral to a mental health professional is seldom made without the consultant's concurrence, and responsibility for ongoing care is always shared, regardless of who provides the psychotherapy. Because of the consultant's continuing involvement, the primary physician more readily accepts the principal psychotherapeutic role.

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Success or Failure: Psychotherapeutic Considerations for Women in Conflict

BY CAROL C. NADELSON, M.D., MALKAH T. NOTMAN, M.D., AND MONA B. BENNETT, M.D.

The authors describe conflicts experienced by women who request therapeutic intervention because of symptoms of depression, anxiety, marital discord, difficulty in asserting themselves, or inability to complete work that would lead to advancement. They discuss the developmentally based difficulties experienced by women in making career choices, advancing their careers, and facing midlife issues. The therapist who deals with such patients must be aware of reality-based factors and of his or her own values and attitudes as they influence choice of therapeutic approach.

AMONG THE MANY CHANGES of the past decade has been an increase in the number of women entering professional careers. Establishing a professional identity is a more complex and difficult problem for women than for men (1) because self-actualization, the pursuit of independent goals, and risk-taking have not been seen by our society as consistent with "femininity." Women are often conflicted about activities regarded as aggressive or competitive because they fear loss of approval and rejection if they violate established norms.

Until recently, women entering careers thereby chose a life pattern that set them apart from most of their peers. The capacity to tolerate being "deviant" has important psychological implications. Psychotherapists see women who are successful in many areas but who are conflicted about the discrepancy be-

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tween their aspirations and activities and their early familial and developmental experiences. Therapists are influenced by the same cultural, familial, and intrapsychic factors as are their patients. Thus, they may find it difficult to objectively examine the nature and intensity of the conflicts presented. For every therapist the ability to differentiate what is actually mentally healthy from the idea of what is healthy according to subjective personal values becomes critical. This differentiation is more complicated than is often acknowledged, and the problem is made more acute by the rapidity of change in values and mores in recent years.

Concepts of what is healthy as well as what is appropriate, rational, or acceptable clearly differ among individuals; objective criteria are not available. In 1970 Broverman and associates (2) demonstrated that views of mental health are affected by sex role stereotypes. Although some changes have occurred since their study, the personal and cultural values that enter into therapeutic decisions continue to be particularly salient in the treatment of women.

Biological and reproductive factors are also inevitably important in any discussion of therapy of women. Although these factors clearly do not constitute a woman's identity, women's lives are more influenced by their biological timetables than are men's lives. All women are concerned in one way or another about this reality from the beginning of adolescence. Although men are concerned about fertility, they do not organize their life plans around reproductive issues in the same way women do. Thus, considerations of goals and values as well as of the developmental life cycle and reproductive issues provide a framework for understanding the problems currently presented by women in therapy.

CAREER CHOICE

Early in life one often makes choices on the basis of idealism, romanticism, faddism, or family pressure without adequate appreciation of the complexities and realities inherent in any choice. The rapid increase in the acceptability of and even insistence on women's achievement has found many women unprepared for making these decisions or for following through on decisions already made. This is particularly true for young women whose early developmental experiences were in families with a traditional orientation. The past identifications of such women may not provide models for future expectations. Anxiety may occur when a young women attempts a life course that she believes would either exceed her mother's achievements or meet with disapproval, particularly from her mother. She may be concerned about expressing aggressive or competitive feelings. In the past a woman might have avoided the entire dilemma, but current expectations, including her mother's, often make that option less feasible today.

CAREER DEVELOPMENT

The transition from student life to actively establishing a career requires a confrontation with pressure that may be quite different from those which were fantasied earlier in life. Women are often poorly prepared to face the demands of a career, perhaps because they have not conceptualized themselves as functioning in this role. They may find themselves unable to make long-range career plans and unconsciously sabotaging the efforts of others to help them. They may regress and become more dependent, helpless, or childlike to gain approval and aid from male colleagues; they may choose jobs that are less interesting and rewarding when confronted with the option of assuming responsibility, or they may deprecate their achievements to avoid jeopardizing relationships with men. Although these behaviors might have been successful for women in some areas, they are generally counterproductive from the perspective of attaining professional goals. Many women seek therapy because of the symptom of depression, anxiety, marital discord, difficulty in asserting themselves, or inability to complete work that would lead to advancement.

Conflict avoidance and regression diminish anxiety by preventing the open expression of ambition or self-assertion. Because acknowledging aggression, even when it is conceptualized in terms of mastery and creativity, is more threatening to women than to men[1] the therapist must be ready to confront resistances to achievement when achievement is perceived as aggression. Because women often view competitive activity as injurious to others, they may feel guilty about competing (3).

Women may become depressed after recognizing anger or discontent with a situation they had previously accepted. This occurs because the woman feels that by experiencing or expressing anger she has violated her internalized ego ideal as a nonaggressive person. She may continue to see herself as helpless and powerless[1] or may retreat out of fear of losing the relationship if she expresses angry feelings. Even "successful" women may maintain this self-image.

The need to avoid conflict leads women to structure their lives so that certain wishes are never tested or fulfilled. Sometimes a life event serves to avoid a confrontation; e.g., an unplanned pregnancy may make it less possible to return to work. At times, conflicts are externalized and projected and/or displaced onto family, bosses, peers, the demands of work or the presence of discrimination. This is particularly complex for a therapist to unravel because there may be considerable reality in the woman's perception of the external difficulties. The therapist must ally himself or herself with the patient in her exploration of her conflict about self-assertion and help her to examine the ways in which she may mask her competent self. It

[1] Miller J, Nadelson C, Notman M, et al: Reconsiderations of aggression and self-esteem in women (unpublished paper, 1977).

important not to simply share her externalizations.

Many women approaching the age of 30 come for treatment when they begin to face the possibility that they may not marry or when they decide not to marry. Even if they have been ambivalent about marriage, the reality of not marrying or of not having children may foster a sense of deviance or defectiveness and lower self-esteem. On the other hand, the peer pressure currently applied toward not having children may make it difficult for some women to face their ambivalence or their sadness about giving up the possibility of having a child. Some women have difficulty taking major professional steps or even making such personal decisions as moving to a better apartment or buying a car at this time. They may fear that these changes would seem to represent a commitment to being unmarried. They may blame their career for their social isolation. Some women find a masochistic solution—they might make choices that avoid serious and potentially rewarding career commitments in order to keep relationship "options" open. They may then become angry and disappointed when rewards do not materialize.

Some women handle these conflicts by adopting a life pattern in which they function as "one of the boys." This may be adaptive because it permits them to be strong and competent peers; it also avoids sexualization of relationships. The cost, however, may be an inability to accept sexual or dependent feelings. These counterdependent mechanisms are often accompanied by rigidity and can interfere with empathy in interpersonal relationships.

Women who attempt to retain their emphasis on relationships without sacrificing the development of competence and assertiveness have a difficult time, particularly in such "masculine" fields as engineering, architecture, and management. They need support and reality testing, and they must also maintain sufficient self-esteem to prevent failure.

Women who combine marriage and a career often experience role strain (4). This may occur early in their careers or at a point when they begin to increase their involvement in activities that do not involve their families. Their husbands may develop a sense of loss and abandonment and may become competitive, demanding, or regressed. Children also often experience such changes as losses. Guilt over these reactions can lead women to give up their outside interests or may stimulate them to overcompensate and become too self-sacrificing. Some women respond by displacing or projecting their anxiety and are thus seen as overcritical, intolerant, or unresponsive. Some women externalize in this situation and see their family as the major source of their problems rather than understanding their own conflicted responses to pressure. Problems with children are especially likely to precipitate guilt and anxiety (5).

At times marital or sexual problems occur. There has been considerable speculation about the possible increase in male impotence as a result of the increase in women's aggressive behavior and sexual demands. Although there is no good evidence to support this idea, the concern evoked reflects men's anxiety about their masculinity when traditional roles are changed, when they receive more demands for sexual performance and for assuming traditionally feminine tasks and roles. Psychological withdrawal, including loss of sexual interest or potency, sometimes rigid conpensatory assertions of masculinity, or other symptoms may occur as attempts to restore self-confidence and diminish anxiety.

The specific problems of the dual-career family are rarely considered before a couple embarks on this path (6). Generally, each partner makes plans and sets individual goals; these might not be consistent with family demands. Problems often appear in dual-career families when children are born. Conflicting demands force confrontations because alteration of schedules and plans are required. In this situation, as with other conflicts, the wife usually is the accommodating person, limiting her goals and expectations (7). She may then seek treatment because of depression, inability to handle anger, marital friction, or problems with children.

A woman returning to a career may find reentry stressful; it is also difficult to increase a time commitment if she has been working part-time. Positions may not be available, the demands of a particular field may have changed, she may not have the credentials to enter at the level she seeks, or she may find herself working with younger and less experienced colleagues. Women are often unable to confront their passivity and their internal restrictions on aggression and its expression when a more active commitment is necessary in the new role they seek. Depression and anxiety may result when they challenge their internalized self-image, or aggression may be mobilized, resulting in guilt feelings. Some women act out and seek their own "space" by leaving their families. Others avoid the challenge by backing down and refusing positions of authority and responsibility, although they often state that they want these positions. At times a woman views her husband or family as critical or unaccepting as a way of dealing with her own ambivalent feelings.

MIDLIFE ISSUES

As people approach midlife they reflect on the choices they have made. Women who have not married or who have not had children may feel they have paid too heavy a price for the choices they made. When underlying anger is mobilized, they may find it difficult to handle it appropriately or productively and may seek treatment. On the other hand, women who have placed family commitments before their careers may face losing their primary source of identity with the onset of menopause. Divorce, the death of a spouse, the fact that children are leaving home may precipitate anxiety, depression, or regressive symptoms. Women with important career investments seem

to be less vulnerable to depression in the midlife years. Those who have not had children have generally come to terms with this before they reach menopause. Women who are heavily invested in family roles respond more strongly to the menopausal changes (8).

THERAPEUTIC IMPLICATIONS

Although many of the issues considered here are clearly applicable to men as well as women, we will emphasize them as they relate to women.

Therapy Referral

Specific requests may be made for male or female therapists by patients or by other therapists. Women therapists are most often chosen for referral of women patients to avoid a sexualized or a potentially difficult authoritarian transference, for the therapist's "maternal" qualities, and/or to provide role models. Women who choose a woman therapist often feel that she would be more responsive to their wishes for self-actualization. The reasons for choosing a therapist may be based on stereotyped or traditional views without regard for the characteristics of the individual therapist (9). Nevertheless, these preferences may be important. The patient's feeling of greater comfort or empathy can facilitate the development of a positive therapeutic alliance.

Reality-Based Factors

The realities of the patient's specific life situation are important factors that influence the choice of therapeutic modality or approach. A method of problem-solving or conflict resolution that is optimal under certain conditions may not be realistic in others. It is obvious that a woman who must decide about continuing or aborting a pregnancy requires a short, focused therapeutic approach rather than long-term therapy. A 40-year-old woman with conflicts about becoming pregnant cannot spend several years in psychoanalysis before deciding whether or not to have a child. Less obvious but important variables are the needs, priorities, and emotional resources relating to a particular situation that may be externally stressful. For example, a woman medical student with problems in her relationships with men might be a good candidate for intensive psychotherapy or psychoanalysis, but while she is in medical school she may benefit more from a therapeutic experience that is less demanding and puts less strain on her academic performance.

Values, Attitudes, and Countertransference Issues

The values and attitudes of the therapist can influence clinical formulation, choice of treatment modality, and even the focus of therapy. For example, because social relatedness is generally considered to be more critical for women than men, a woman with serious problems in interpersonal relationships but who is successful in other areas of her life may find that her therapist is more concerned with her interpersonal relationships than her career-related issues, regardless of the priority the patient assigns to this area. This interpersonal emphasis is more likely to occur with a isolated but occupationally successful woman than with a man. Demands involving work or academic requirements are often treated as resistance in women. Some therapists still regard success for women in traditional terms; such therapists may not take self-actualization related to nonfamily areas of life seriously or may see it as postponable for women. Therapists and patients may limit their exploration of alternatives because they see career and family involvement as mutually exclusive or too complex.

Therapists may also find it difficult to understand women who decide to have children when they are older or who are ambivalent about having children. These therapists may not realistically explore career goals and possibilities or potential problems and pitfalls. The woman house officer who wants to have a baby but who has undertaken a very demanding program may need the opportunity to explore why she chose to become so committed at the particular time she did, just as the 40-year-old woman with an established career who suddenly decides to have a baby and give up her career must understand this decision. Both women may be acting out in a self-destructive fashion or may have important positive reasons for their decisions.

Another area of concern is the therapist's view of what constitutes psychopathology in women. The therapist who finds it difficult to accept aggressive or assertive behavior in women may accept a woman's passive, masochistic stance rather than help her master her anxiety or depression by taking a more active self-directed position. On the other hand, the therapist may pressure the patient into unwanted competitive activity and major career commitment because the therapist feels this is appropriate. Regressive behavior as a defensive style may be seen in a woman as characterologically primitive when it is a style of presentation or a manifestation of low self-esteem not necessarily related to major defects in ego functioning. A supportive rather than explorative therapeutic approach may then be recommended.

Countertransference feelings may lead therapists to overidentify with patients and to project their own experience onto them. They may be in awe of women who are effective professionally and not attend to the concerns they present. The patient herself may support this perception because of her need for control, a quality that is important in her professional success. Thus, both patient and therapist may collude to accept a view that leaves out important areas for therapeutic work. In such instances therapists may foster a more egalitarian or intimate relationship or a more distant one than is helpful to the patient.

The impact of feminist views has led many patients to mistrust formulations that use psychoanalytic language (10) because of the implications of such terms as

penis envy. The therapist or analyst who interprets this as resistance without understanding the patient's concerns will come to a therapeutic impasse. It may be necessary to explain, to avoid jargon, and to specifically reinforce the alliance between individuals rather than ideologies. A stance perceived by the patient as provocative, paternalistic, or authoritarian may make it difficult or impossible for her to develop a trusting relationship.

COMMENT

Feminine identity, reexamined in the light of recent evidence and in the context of social changes, includes the integration of self-esteem, self-assertion, and independence as well as nurturance. A developmentally oriented view of feminine development and conflict supports the view that it differs from male development because of the impact of the reproductive timetable. Both childhood and adult developmental phases must be incorporated into concepts of femininity as a changing process with different expressions at different life stages.

For the professional woman this integration is particularly important. The therapist must be sensitive to problems arising from conflicts between the demands of professional adaptation and internalized contradictory expectations deriving from developmental experience. The therapist must be aware of the impact of real life events as well as intrapsychic conflict in symptom formation. Formulations and therapeutic approaches must be modified in the light of data from a number of disciplines and in the context of social change.

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Psychotropic Drugs, Summer Heat and Humidity, and Hyperpyrexia: A Danger Restated

BY STEPHAN C. MANN, M.D., AND WILLIAM P. BOGER, M.D.

The authors note an apparent unawareness of, as well as a paucity of recent literature pertaining to, hyperpyrexia as a complication of psychotropic drug treatment. They report a case of hyperpyrexia in a patient receiving psychotropic agents during a summer heat wave; massive muscle destruction (rhabdomyolysis) with myoglobinuria and acute renal failure make this case of particular interest. Causative mechanisms, preventive measures, and the benefits of prompt recognition and treatment of this condition are discussed.

IN A June 1970 report (1) of heat stroke fatalities among phenothiazine-treated patients, Zelman and Guillan stated, "The phenomenon of hyperpyrexia in phenothiazine therapy is not new, but has been little discussed. . . . We feel it urgent that . . . [our] experience be reported in order that such catastrophes may be anticipated and guarded against" (pp. 1787, 1789). During a summer heat wave we observed an excessive temperature elevation and subsequent massive muscle destruction (rhabdomyolysis) in a patient treated with a standard regimen of psychotropic agents. The surprise generated among colleagues spanning several institutions, specialties, and levels of tenure suggested a reexamination of life-threatening hyperpyrexia in patients treated with phenothiazines, neuroleptics of other classes, and other drugs commonly used concurrently to see to what extent these summer dangers have remained "little discussed" since Zelman and Guillan's report.

When one scans the major textbooks of psychiatry, internal medicine, and physiology as well as the much-consulted *Physicians' Desk Reference* (2) and *AMA Drug Evaluations* (3) one finds only the casual statement that "hyperpyrexia may occur" with neuroleptic treatment. No attention is directed to the degree of danger involved or to the compounding effects of agents used in conjunction with neuroleptics. Among textbooks of pharmacology Goodman and Gilman's volume (4) does mention the possibility of fatal hyperpyrexia associated with neuroleptic treatment and thus apparently contains the most direct source of this information in a readily available text. Even among manuals devoted to psychotropic drug side effects, Shader and DiMascio's book (5) appears to be alone in clearly mentioning temperature elevation as potentially dangerous.[1]

The *Cumulated Index Medicus* from 1970 to the present was scanned manually under the headings of Tranquilizing Agents, Tranquilizing Agents—Major, Phenothiazines, Psychopharmacology, Body Temperature, Body Temperature Regulation, and Fever. The computerized MEDLINE retrieval system was employed for a literature search back to 1969 using the subject headings of Tranquilizers, Major Tranquilizers, Hyperthermia, Fevers of Unknown Origin, Body Temperature, Body Temperature Regulation, and Body Temperature Changes. Finally, we examined the bibliographies of authors of those articles considered relevant in editions of *The Science Citation Index* dating back to 1970. This search revealed only a few pertinent articles and letters to the editor scattered throughout general medical, anesthesiological, and pediatric publications (6–10). Most strikingly, we found only one article devoted principally to this topic in a psychiatric journal (11). There is clearly a dearth of information on the seriousness of psychotropic drug-induced hyperpyrexia and, accordingly, a restatement of the problem is indicated.

PHYSIOLOGICAL IMPLICATIONS

The hypothalamus, the body's major center for thermoregulation, prevents overheating by both increasing perspiration and bringing heat to the body surface through peripheral vasodilation. Three avenues are available for the dissipation of bodily heat—radiation, conduction to objects and air, and evaporation of sweat. As the ambient temperature approaches that of the body, radiation and conduction will yield a net heat gain, rather than heat loss, leaving evaporation as the only major mechanism of heat loss. However, in dry air the healthy body is capable of maintaining a rela-

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[1]A detailed list of the various types of books discussed here is available from Dr. Mann on request.

tively normal body temperature in environments up to 130°F (54.5°C) (12). Heat is brought to the surface by dilated peripheral vessels and effectively lost through evaporation. High humidity will impair this mechanism, since less sweat can be lost to the already highly saturated air.

It has been known for some time that phenothiazines suppress thermoregulation at the hypothalamic level, leaving phenothiazine-treated animals vulnerable to high or low environmental temperatures (13). Similar hypothalamic suppression has been demonstrated in humans and forms the basis of the familiar therapeutic technique of phenothiazine-induced hypothermia. This hypothalamic suppression is a major mechanism by which life-threatening hyperpyrexia is caused. It occurs in varying degrees with neuroleptic agents of classes other than the phenothiazines and is apparently moderated through the effects of these agents on several centrally acting biogenic amines (14, 15).

Peripheral mechanisms are also important in producing hyperpyrexia. Neuroleptics exert peripheral *alpha*-adrenergic and cholinergic blockade of varying degrees. These effects run roughly parallel and are greatest with the so-called "low potency" neuroleptics such as aliphatic phenothiazines, piperidine phenothiazines, and chlorprothixene. With "high potency" agents such as piperazine phenothiazines, thiothixene, and haloperidol, these peripheral blockade effects are less marked. The adverse effects of such influences on thermoregulation at high temperature and humidity follow logically from the physiology discussed above. *Alpha*-adrenergic blockade widely dilates the peripheral vessels, depriving the body of insulation from the seething environment. Sweating, already impaired by high humidity, is further compromised by the well-known suppressing influence of cholinergic blockade, and heat loss becomes a potential impossibility. Antiparkinsonian agents, possessing great anticholinergic activity of their own, are the most common culprits in compounding the problem. Concurrent administration of tricyclic antidepressants, monoamine oxidase inhibitors, and lithium may further aggravate the situation in that they themselves may cause severe hyperthermia, acting presumably by a central mechanism (16). In addition, tricyclic antidepressants have marked anticholinergic activity.

Neuroleptics are also capable of causing hyperpyrexia in the absence of high environmental temperatures and by mechanisms less well understood than those described above. The neuroleptic "malignant" syndrome characterized by hyperpyrexia, muscular rigidity, and coma, first described by Delay and Deniker (17) and observed by others (18–20), has been discussed by Meltzer (20) as being possibly related to the syndrome of malignant hyperpyrexia.

Malignant hyperpyrexia is an autosomally inherited disease that predisposes individuals to react to potent inhalational anesthetics with a rapid and often fatal rise in temperature, frequently accompanied by muscular hypertonicity (21, 22). Elevated creatine phosphokinase (CPK) levels are found in a considerable number of patients who have recovered from this syndrome and also in asymptomatic relatives of patients so affected (22). CPK is an enzyme found in brain and in both cardiac and skeletal muscle. Numerous clinical conditions involving damage to these tissues, e.g., strokes, convulsions, myocardial infarctions, bodily trauma, multiple intramuscular injections, and muscular dystrophies, can cause elevated serum CPK values. The source of these elevations can be determined by specific isoenzyme assays. It is believed that the "resting state" elevations in CPK found in individuals with malignant hyperpyrexia reflect a subclinical myopathy that makes these patients vulnerable when stressed by the offending agent (22).

There have been repeated confirmations of the finding of elevated skeletal muscle CPK in both patients suffering from a wide variety of psychotic conditions and in their nonpsychotic relatives (23–26). Citing these findings and noting the similarity of neuroleptics to anesthetic agents implicated in malignant hyperpyrexia, Meltzer (20) has hypothesized a similar mechanism of disordered muscular function rendering certain psychotic patients—those with the highest CPK elevations—more likely to develop the neuroleptic "malignant" syndrome.

CASE REPORT

A 48-year-old male psychiatric patient had been hospitalized at Coatesville Veterans Administration Hospital for almost 3 years, and for 4 weeks he had been receiving 700 mg of chlorpromazine per day, 1.5 cc of fluphenazine enanthate I.M. every two weeks, and 6 mg of biperiden per day. Early on the morning of September 1 he eloped from the hospital and hitchhiked to central Philadelphia, which was in the midst of a record-breaking heat wave. Unable to gain entrance to a friend's apartment, he wandered the streets during the late morning and afternoon until he became dizzy and collapsed. It was not until evening that he was found lying unconscious on the sidewalk and brought by the police to a local hospital. There, still unconscious, he was described as exhibiting "purposeless chorioathetotic movements of the arms, but no seizure activity." His blood pressure was recorded as 70/0. He was dry and hyperpneic, and his eyes were fixed midline with pupils midpoint and reactive to light. There is no record of a temperature having been taken at the time. He rapidly regained consciousness and became "combative." Since his papers readily identified him as a psychiatric patient, he was transported to the Philadelphia Veterans Administration Hospital for treatment of what was considered to be an acute functional psychiatric disturbance.

The patient arrived at the Veterans Administration Hospital about 2 hours after being found by the police. He was alert and complained of leg cramps, weakness, and thirst. He was sweating and had a rectal temperature of 103°F (39.5°C). Blood pressure was 80/40 and heart rate was 140 beats per minute. No bruises or swelling were noted. Skin turgor was fair, and there was no apparent focus of infection. On neurological examination no focal abnormalities were noted, and

the "purposeless movements" were no longer present. However, moderate bilateral weakness of the upper and lower extremities was noted.

Sodium concentration was 140 mEq/liter; chloride, 102 mEq/liter; potassium, 6.4 mEq/liter; carbon dioxide, 13 mEq/liter; and blood urea nitrogen, 22 mg/ml. Hemoglobin content was 14.6 mg/100 ml, and white blood count was 15,700/cu mm. Arterial blood pH was 7.29; Po_2 was 70 mm Hg; Pco_2 was 25 mm Hg; and total bicarbonate was 11.6 mEq/liter. Chest X ray revealed no acute infiltrates. The clinical impression at the time was "hyperpyrexia and dehydration secondary to heat stroke." Blood, urine, and cerebrospinal fluid cultures were later reported as showing no abnormalities.

Initially, the patient responded well to fluid replacement, and urinary output was good. However, 5 hours after admission, urinary output fell to 40 cc per hour and responded little to mannitol infusion or furosemide I.M. Generalized muscle weakness progressed to almost complete flaccidity. Urinary myoglobin as determined by immunoassay was positive, and CPK rose rapidly to a peak of 166,700 units (normal is less than 45). This excessive elevation in CPK was indicative of massive muscle necrosis; a diagnosis of acute rhabdomyolysis was made and was subsequently confirmed by muscle biopsy.[2] Thus hyperpyrexia, with dehydration and hypotension, had caused massive muscle destruction leading to myoglobinuria and acute tubular necrosis.

A stormy course ensued and peritoneal dialysis was employed several times in the management of the impaired renal function. Over the course of several months the patient showed remarkable improvement in muscle strength. His renal function normalized surprisingly well. However, the severe renal damage that he had suffered contributed to an anemia that proved to be quite refractory to treatment.

DISCUSSION

Summer heat kills more than 4,000 people per year in the United States, with most deaths occurring in the humid urban areas (27). The typical individuals involved in heat morbidity are predisposed by chronic diseases such as arteriosclerosis, alcoholism, and diabetes mellitus. Such patients in particular and even persons not so predisposed are at greater risk when exposed to excessive thermal challenges if they are under treatment with psychotropic agents. As the body temperature rises above 106°F (41.1°C), damage to vital organs, e.g., brain, heart, liver, kidneys, and muscle, may occur (27, 28). The renal excretion of pigmentary breakdown products resulting from extensive muscle damage may lead to acute tubular necrosis. Hyperpyrexia can produce numerous abnormalities related to bleeding and clotting, including consumptive coagulopathy.

While three separate syndromes are usually delineated in association with high environmental temperatures, i.e., heat cramps, heat exhaustion, and heat pyrexia (heat stroke), considerable overlap exists (21). The fatalities reported by Zelman and Guillan (1) presented the classic picture of heat stroke—hot and dry skin, rectal temperature above 106°F (41.1°C), severe central nervous system disturbance, and an absence of sweating, indicating a potentially fatal breakdown in thermoregulation. Failure to employ aggressive measures promptly may result in death.

The patient reported here suffered severe hyperpyrexic morbidity resulting from an elevated body temperature probably well above the 103°F (39.5°C) recorded several hours after the termination of an extended exposure to excessive heat. That his body temperature had returned to 103°F (39.5°C) and that he was sweating when first seen at the Philadelphia Veterans Administration Hospital indicates a thermoregulatory apparatus sufficiently intact to enable his body to avail itself of the air-conditioned, dehumidified hospital receiving ward. This patient's symptomatology is best interpreted as a severe form of heat exhaustion—a syndrome characterized by hypotension and dehydration, but with unimpaired thermoregulatory capability. Even in the milder forms of heat exhaustion, the associated hypotension might prove particularly deleterious to patients with already compromised circulation, and the associated dehydration could precipitate lithium intoxication in those patients under treatment with lithium.

Psychiatric patients may be disproportionately exposed to summer heat and humidity, since many of them live in small, poorly ventilated city rooms and frequently wander aimlessly about the city streets. When sustained increased psychomotor activity is present in such patients, it results in a marked increase in body heat, making them particularly vulnerable to heat-induced illness. Hospitalized patients are also at risk, since many psychiatric inpatient facilities are not yet air-conditioned. Seclusion rooms are a specific concern because they are often small, hot, and lacking in cross-ventilation, and it is in such rooms that those patients who are most agitated, heavily medicated, and even struggling vigorously against restraints are often confined.

A primary consideration in minimizing the risk of hyperpyrexia should be the administration of neuroleptics in the lowest dosage effective in a given situation. While large doses of antipsychotic medication are frequently needed in treating acute psychosis, it has become quite evident that maintenance schedules are often unduly high. When large doses are required in extremely hot weather, drugs low in anticholinergic and anti-*alpha*-adrenergic activity, i.e., high-potency agents, should be preferentially selected. Rapid tranquilization and megadose techniques might best be employed with care during such periods. Most importantly, antiparkinsonian agents should not be prescribed prophylactically and should be withdrawn as soon as possible. Tricyclic antidepressants and lithium should be added to the therapeutic regimen with appropriate caution.

Awareness on the part of emergency ward physi-

[2]CPK elevations in the neuroleptic "malignant" syndrome have not been reported to approach the level seen in this patient.

cians, and psychiatrists in particular, of the potential of psychotropic drugs to cause life-threatening hyperpyrexia should ensure the more frequent recognition of this syndrome and the prompt initiation of appropriate therapy. The better recognition of drug-induced hyperpyrexia should reduce the indiscriminate prescribing of antibiotics and other medications in the management of psychiatric patients with fevers of unknown origin.

The type of treatment is dictated by the severity of the hyperpyrexia. In the case of heat exhaustion, removal to a cool environment, rehydration, and rest will generally suffice (21, 27). However, heat pyrexia (heat stroke) is a medical emergency. The patient must be immersed in an ice water bath to reduce rectal temperature to 101°F (38.3°C). Vigorous massage to promote dermal circulation is indicated. General measures to sustain the respiratory and cardiovascular systems are mandatory. For further details on the treatment of this life-threatening situation the reader is referred to *Harrison's Principles of Internal Medicine* (21) and Clowes and associates' report on heat stroke (27).

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Psychosocial Aspects of a Tay-Sachs Screening Clinic

BY GERALD SCHNEIDERMAN, M.D., J. ALEXANDER LOWDEN, M.D., PH.D., AND QUENTIN RAE-GRANT, M.D.

The authors administered questionnaires to participants in a Tay-Sachs screening clinic. They received 322 responses. Although the majority of the respondents expressed anxiety about the possibility that they might be carriers, most indicated that this anxiety brought them to the clinic. The authors conclude that a well-publicized and well-organized screening clinic can function successfully and deal with the issue of anticipated loss without producing mass fear. An important factor in their clinic was the fact that they were able to offer an alternative to a Tay-Sachs child through amniocentesis.

CARRIER DETECTION PROGRAMS for genetic disease have come under attention recently because of scientific advances. We are studying the psychological effects of carrier screening programs on an involved population. The purpose of this paper is to examine a population participating in a Tay-Sachs screening clinic.

BIOCHEMISTRY AND GENETICS

Tay-Sachs disease is an inborn error of ganglioside catabolism (1–3) that affects young infants. The patient appears well at birth and develops normally for six to eight months, but then psychomotor degeneration begins. The child becomes blind and spastic, convulses frequently, and dies from aspiration pneumonia by the time he or she is three to five years old. The disease is caused by a defect in the activity of the enzyme hexosaminidase A, which normally hydrolyzes the β-1,3-glycosidic linkage between the terminal *N*-acetylgalactosamine and galactose in the G_{M2}ganglioside molecule. The lipid cannot be catabolized and is stored in large amounts in nervous tissue and in smaller amounts in other organs. The disease is 100 times more common in Jewish infants than in others. Among Ashkenazi Jews, the calculated carrier frequency is 1 in 30 (4) to 1 in 15 (5).

The total hexosaminidase activity varies greatly from one individual to another, but the percentage accounted for by hexosaminidase A is relatively constant. By measuring the percentage of hexosaminidase A in serum or tissues, affected infants can be identified and carriers of the genetic disorder detected with a high degree of accuracy. The enzyme is normally present in the amniotic fluid cells and cultured fibroblasts, so that the diagnosis can be made in utero by amniocentesis (6).

Because heterozygote detection is relatively simple, because the disease is far more common in a defined population group, and because a simple and accurate test is available for antenatal diagnosis, mass carrier screening programs have been undertaken to search for heterozygotes in families with no previous history of Tay-Sachs disease.

PREVIOUS STUDY

In a previous study we interviewed 95 families two years after they had participated in a testing program (7). We observed families who were presented with the possibility of losing a child. Their anxiety was heightened while they were undergoing testing and waiting for the results. However, this was followed by relief for the unaffected families and a cautious optimism for carrier couples. In all of the families interviewed there was no long-term morbidity.

Although the screening program stirs up feelings in the families about life and death, amniocentesis allows families to face the anxiety produced by the testing program with the knowledge and the resulting hope that affected families can still produce normal, healthy children.

PRESENT STUDY

Our latest study is prospective and involves the clients who came to a Tay-Sachs clinic on the day of screening. Questionnaires were given to all of those who came for testing.

Presented at the Annual Research Day, Department of Psychiatry, University of Toronto, Toronto, Ont., Canada, September 10, 1976.

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Family Backgrounds

The number of questionnaires answered was 322. The average age of the married men who answered the questionnaire was 30.97, and the average age of the married women was 28.77. The average number of children per family was 1.91. The average age of the single men who answered the questionnaire was 22.54; that of the single women was 22.05. The occupational levels according to Hollingshead and Redlich varied from class I to class IV, but 82.6% were in classes I and II.

Observations and Findings

The medical staff of the local community hospitals had organized publicity for the screening clinic through television, radio, and newspapers and through local synagogues. They spoke to local lay groups about the clinic and educated volunteers to work in the screening clinic. On the days that the clinic was held, medical personnel were in attendance to answer questions.

The clinic was efficiently organized and staffed by volunteers who helped participants feel less anxious. Their knowledge enabled them to answer queries with comfort and ease. In general, women asked questions more directly; the men phrased their questions in a jocular defensive manner. Enough technicians were available that the wait to have blood taken was short. Coffee and light snacks were available for the participants.

Several single women who were about to be married questioned the medical personnel about Tay-Sachs; they were very concerned about their future should they and their fiancées both be carriers. The physician's response was to separate the issues of marriage and Tay-Sachs disease.

According to the questionnaires, in 75% of the married couples testing was initiated by the wife. Although some of the respondents were misinformed on a number of facts about Tay-Sachs disease, most of them understood the concept of the disease. Seventy percent favored abortion of an affected fetus, but 29% of the group who answered the questionnaire did not respond to this question. Although 75% of the respondents expressed anxiety about finding that they were carriers, the majority indicated that anxiety is what led them to participate in the clinic. Although some of the respondents had misgivings about participating—such as the fear of blood and needles—these were temporary. The majority of the participants felt that the program was worthwhile and helpful and were pleased that it was available. When asked about their feelings about the future of the Jewish family as an institution in the light of the fact that Tay-Sachs disease occurs predominantly in Jewish families, 25% of the respondents expressed concern.

DISCUSSION

Our findings indicate that a well-publicized and well-organized clinic can function successfully and deal with the issue of anticipated loss without producing mass fear and hysteria. The important issue is that the clinic offers an alternative to a Tay-Sachs child through amniocentesis. Our previous findings showed that there were no long-term psychological sequelae to a screening program. Although the participants expressed anxiety, they were reassured by the volunteer and medical staff.

The one major area of concern occurred in single people about to be married. It should be pointed out that marriage and preventable genetic disease are separate issues and ought not to be connected in making a choice of partners. We hope that the Tay-Sachs screening program will serve as a model for the prevention of other genetic diseases (8).

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Recidivism Among Sex Assault Victims

BY JILL MILLER, DEBORAH MOELLER, R.N., ARTHUR KAUFMAN, M.D., PETER DIVASTO, PH.D., DOROTHY PATHAK, PH.D., AND JOAN CHRISTY

The concept of recidivism has traditionally been applied to the assailant in a crime. However, recent work in the field of victimology has evolved a concept of victim recidivism. A recent national survey of victims of violent crimes revealed that 28% were prior victims of violent crime (1). Data on rape victims, however, were not included in the survey.

A growing body of medical literature on sex assault victims concerns itself primarily with the impact of the assault on (2, 3) and the proper treatment of (4, 5) the rape victim. However, data on the prevalence and characteristics of rape victim recidivism also deserve examination for they might shed light on risk factors for potential victims in the population. We obtained information relevant to this subject from the data sheets completed for rape victims seen by the Sexual Assault Response Team of the Department of Family, Community, and Emergency Medicine of the University of New Mexico School of Medicine over a 2½-year period.

Method

The University of New Mexico School of Medicine Sexual Assault Response Team has been providing immediate crisis intervention, medical assistance, and long-term follow-up counseling for sexually assaulted victims and their families since 1974. The team consists of medical and nursing students who rotate on a 24-hour call schedule to the emergency room of the county teaching hospital of the medical school. Each team member completes a victim data form, which includes information concerning demographic data, nature and circumstances of the assault, previous assaults, previous need for counseling, emotional and physical condition of the patient, and resulting medical treatment and legal disposition. We reviewed the data forms on 341 victims seen from January 1975 to June 1977 for information relative to past sexual assaults. Data on rape victim recidivists were matched against those of first-time victims, and both groups were then compared to general population statistics for the Albuquerque metropolitan area.

Results

Of the 341 victims seen, 82 (24%) were recidivist victims of sexual assault. The average ages of first-time and recidivist victims were 24 and 23 years, respectively, an insignificant difference. The distribution of educational attainment between both the first-time and recidivist groups was also similar.

In the first-time group, 11 of the 259 contacts (4%), were incestuous whereas of the 82 recidivist victims 15 (18%) of the prior assaults involved incest. This difference is highly significant ($p<.001$).

Tabulation of the economic status of victims 18 years or older (72% of each group) revealed that 33 of 186 first-time victims (18%) compared with 19 of 59 recidivist victims (33%) were either unemployed or receiving public assistance. This difference was also statistically significant ($p<.05$). A measure of relative transiency among all victims showed that 80 of the 259 first-time victims (31%) were recent arrivals (lived in Albuquerque less than one year), whereas 41 of 82 recidivists (50%) were recent arrivals. This difference in proportions is again highly significant ($p<.005$).

A comparison of the two groups' past emotional problems revealed that 54 of the 259 first-time assault

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victims (21%) had previously sought professional help for emotional problems, as had 39 of the 82 recidivists (48%). This difference, too, is highly significant ($p<.001$).

An ethnic comparison of each group with the general Albuquerque population revealed significant differences (see table 1). A major part of the extremely high χ^2 value of the difference of the first-time victims from the general population (observed $\chi^2_3=174.4$, $p<.0005$) was due to the much larger proportion of American Indians in the first-time assault group than in the general population. The observed proportions of American Indians and blacks were larger than expected, while the Chicano proportion was smaller than expected. For the Anglo, the proportion was about the same as expected, i.e., smaller in the first-time victim group than in the general city population and about the same in the recidivist group.

TABLE 1
Ethnic Comparison of Albuquerque Population Census (1970) with First-Time and Recidivist Sex Assault Victims

	City Population (N=243,751)		First-Time Victims (N=259)		Recidivists (N=82)	
Ethnic Group	N	%	N	%	N	%
Anglo	148,122	61	129	50	53	65
Chicano	85,032	35	89	34	17	21
American Indian	3,351	1	34	13	5	6
Black	5,425	2	7	3	7	8
Other	1,821	1				

Discussion

It is likely that the proportion of sex assault recidivism is even higher than the 24% evident in our survey. Many of these data were collected in a crisis setting not necessarily conducive to sensitive historical revelations. In follow-up counseling sessions, many victims revealed for the first time prior incidents of sexual assault. However, some victims did not avail themselves of follow-up services.

For the majority of recidivist victims, treatment for the current assault provided them their first opportunity to discuss the prior assault(s). It was a recurrent observation of many Sexual Assault Response Team members that the trauma of the current assault could not be resolved until the prior assault(s) was adequately addressed. Team members also observed an almost fatalistic attitude toward the current assault among members of this group. There were numerous incidents of violence and abuse in their lives and the current assault seemed just one more in a long series of traumas.

The higher proportion of unemployment among recidivists may reflect the societal association of poverty and crime. In some cases, such as hitchhiking because one does not own a car, this relationship is straightforward. In others, it is less apparent. The unemployment figure may also be indicative of a group less able, for social, physical, or psychological reasons, to obtain and keep a job. These persons may be less effective in overall living and thereby may be easier prey for perpetrators of sexual assault. As a final hypothesis, the unemployment may reflect a lifestyle choice that does not require formal employment.

Another notable feature of this group of patients is their transiency. A large number of them appeared rootless. Some were runaways and had been for months or years. This population is especially at risk for exploitation since they must rely on strangers for food and shelter, which increases their chances of sexual assault. Still others were recent arrivals in the city and, perhaps, not yet "street wise."

The greater prevalence of prior emotional disturbances among recidivist victims may suggest behavioral characteristics that increase their vulnerability to sexual assault. A number of victims in this group were either known or suspected of being either mentally retarded or severely disturbed. Their ability to judge external danger appeared to be grossly impaired. Some exhibited a naive trust of their environment that probably contributed to their assault.

Based on observations of the Sexual Assault Response Team members, recidivist victims were more likely to come from disruptive homes and live a more chaotic lifestyle than first-time victims. Many appeared to have such strong dependency needs that they were recurrently taken advantage of financially and sexually.

An area inviting exploration is whether recidivist victims are more likely to exhibit repetitive destructive behavior patterns. The overwhelming majority of recidivists in our survey had failed to receive medical or counseling services after their prior assaults. If such services had been received, such intervention might have provided insight, support, and modification of their behavior, rendering them less vulnerable to future assaults.

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Sodium Nitroprusside Treatment of ECT-Induced Blood Pressure Elevations

BY DOMINIC CIRAULO, M.D., LEONARD LIND, M.D., CARL SALZMAN, M.D., ROBERT PILON, M.D., AND ROBERT ELKINS, M.D.

Electroconvulsive therapy (ECT) transiently raises blood pressure (1). This hypertensive response is independent of convulsive activity and is thought to result from a rise in peripheral catecholamines secondary to central sympathetic stimulation (1, 2). Patients who are hypertensive often show greater ECT-related increases in blood pressure than those who are normotensive (3). Atropine, which is routinely used as a pre-ECT medication to suppress vagal stimulation, elevates blood pressure further (3), as does succinylcholine, which is used for muscle relaxation (4). In this paper, we will describe a case in which the blood pressure of an elderly hypertensive patient was controlled by using intravenous sodium nitroprusside during each treatment. This technique proved to be simple, reliable, and successful in controlling the potentially hazardous elevations of blood pressure.

Case Report

A 70-year-old man was referred for ECT because of a severe recurrent depression that was resistant to antidepressant medication. The patient had long-standing hypertension as well as ECG evidence of an old myocardial infarction. Two years earlier he had been given ECT for a similar depression. Despite concomitant antihypertensive therapy at that time, blood pressure elevations as high as 240/160 were recorded during the ECT procedure.

During the first treatment in the present ECT series, his blood pressure rose to 250/140. Sodium nitroprusside infusion was used in the remaining 9 ECT treatments, and blood pressure was transiently elevated only to 190/120. He responded well to an otherwise uneventful course of ECT.

Sodium nitroprusside was prepared by mixing 50 mg with 500 cc of 5% dextrose in water just before use; the mixture was shielded from light. The rate of intravenous infusion was titrated according to the patient's blood pressure using a microdrip infusion set. The infusion was begun 15 minutes before ECT and was continued for 5 to 15 minutes following the treatment. In this patient, there were no adverse effects from the nitroprusside infusion, other than transient hypotension during 1 treatment, which was easily controlled by discontinuation of the nitroprusside.

Discussion

The use of nitroprusside in the treatment of hypertension and the control of blood pressure has been well described in the literature. In the late 1920s, Johnson (5) first discussed the potential utilization of nitroprusside in the therapy of hypertensive crisis. The first clinical report of the use of nitroprusside in hypertensive emergencies was made by Page in 1951 (6). In current medical practice, nitroprusside is commonly used for rapid control of hypertensive crises, production of hypotension during surgery to minimize blood loss and obtain "dry" operative fields, and induction of afterload reduction in the treatment of myocardial infarction, dissecting aortic aneurysm, and marked congestive heart failure (7).

Intravenous infusion of sodium nitroprusside results in rapid lowering of arterial blood pressure, with a concomitant decrease in total peripheral resistance. There is a rapid return of the blood pressure to control values after the infusion is discontinued. Heart rate will increase in most patients as a secondary response to a rapid decrease in arterial blood pressure. Dilation of peripheral capacitance and resistance vessels increases pooling of blood and decreases myocardial preload and afterload (7, 8).

The site of action of sodium nitroprusside is at the vascular smooth muscle membrane; therefore, nitroprusside is considered a direct-acting vasodilating agent. The nitroprusside molecule is believed to interact with sulfhydryl groups of the vascular smooth muscle membrane to produce muscle relaxation (9). In general, toxicity is encountered with prolonged administration and rapid infusion rate and results from release of cyanide from the nitroprusside molecule (7). A maximum dose of 3–5 mg/kg of nitroprusside is recommended, with a suggested rate of infusion between 0.5 and 1.5 μg/kg per minute (10). The dangers of sodium nitroprusside use during anesthesia include those associated with hypotension and cyanide poisoning (7, 10).

In summary, sodium nitroprusside appears to be useful in controlling potentially dangerous elevations in blood pressure in hypertensive patients receiving ECT.

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Hysteria and ABO Blood Types

BY PANTELIS M. RINIERIS, M.D., COSTAS N. STEFANIS, M.D., ELEFTHERIOS P. LYKOURAS, M.D., AND ELEFTHERIA K. VARSOU, M.D.

Associations between ABO blood types as genetic markers and certain mental diseases, such as schizophrenia, unipolar and bipolar depression, and obsessive-compulsive neurosis have been reported (1–3). Since, to our knowledge, no attempt has been made to investigate a possible association between ABO blood types and hysteria, the development of which seems to involve genetic factors (4), we carried out a study to explore this possibility.

Method

We studied 75 subjects (21 men and 54 women) who had been diagnosed by 2 independent psychiatrists (P.R. and E.L.) as suffering from hysteria according to the criteria of Feighner and associates (5), i.e., the patients were suffering from a chronic or recurrent illness that began before age 30, and were manifesting at least 25 medically unexplained symptoms in at least 9 of the following groups:

1. Headaches, sickly during majority of life.
2. Blindness, paralysis, anesthesia, aphonia, fits or convulsions, unconsciousness, amnesia, deafness, hallucinations, urinary retention, difficulty in walking, other unexplained "neurological" symptoms.
3. Fatigue, lump in throat, fainting spells, visual blurring, weakness, dysuria.
4. Difficulty in breathing, palpitation, anxiety attacks, chest pains, dizziness.
5. Anorexia, weight loss, marked fluctuations in weight, nausea, abdominal bloating, food intolerances, diarrhea, constipation.
6. Abdominal pain, vomiting.
7. Dysmenorrhea, menstrual irregularity, amenorrhea, excessive bleeding.
8. Sexual indifference, frigidity, dyspareunia, other sexual difficulties, vomiting all 9 months of at least one pregnancy or hospitalization for hypermesis gravidarum.
9. Back pain, joint pain, extremity pain, burning pains of the sexual organs, mouth, or rectum, other bodily pains.
10. Nervousness, fears, depressed feelings, need to quit working or inability to carry on regular duties because of feeling sick, crying easily, feeling life was hopeless, thinking a good deal about dying, wanting to die, thinking about suicide, suicide attempts.

Fifty of these subjects were hospitalized and 25 were seen and followed at the outpatient clinic of the Eginition Hospital in Athens.

ABO blood types were determined on a blind basis by a single technician who had extensive blood typing laboratory experience. The typing was carried out by the standard method adopted by most transfusion centers (6).

To compare our findings with a control sample we used the data provided by Valaoras (7) on ABO blood type distribution in the Greek population.

Results

Table 1 shows the distribution of ABO blood types in the male, female, outpatients, and inpatient subgroups, in the total sample, and in the control sample.

No significant difference was found between the male and female patients and between outpatients and inpatients in relation to their ABO blood types distribution pattern (chi square analysis). The combined patient subgroups had a significantly higher incidence of blood type A ($\chi^2=17.69$, df=1, $p<.001$) and a significantly lower incidence of blood type O ($\chi^2=8.81$, df=1, $p<.01$) than normal controls.

Discussion

A significantly higher incidence of blood type A was found in our patients with hysteria than in controls. This finding suggests an association between hysteria and phenotype A. Since the significance of the relationship between blood groups and disease is still un-

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TABLE 1
Distribution of ABO Blood Types in 75 Hysterical Patients

Group	AB		A		B		O	
	N	%	N	%	N	%	N	%
Males (N=21)	1	4.8	13	61.9	2	9.5	5	23.8
Females (N=54)	2	3.7	35	64.8	4	7.4	13	24.1
Outpatients (N=25)	1	4.0	17	68.0	2	8.0	5	20.0
Inpatients (N=50)	2	4.0	31	62.0	4	8.0	13	26.0
Total patients (N=75)	3	4.0	48	64.0	6	8.0	18	24.0
Controls (N=304,317)	15,277	5.0	122,275	40.2	42,452	13.9	124,313	40.8

clear (8), premature interpretations of our results should be avoided. However, one is tempted to consider our findings as additional evidence in support of the view that hereditary factors may contribute to the development of hysteria (4).

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Amitriptyline-Induced Obesity in Anorexia Nervosa: A Case Report

BY KENNETH S. KENDLER, M.D.

In a significant proportion of patients anorexia nervosa is preceded or followed by obesity (1). In such patients, it is likely that an abnormal CNS body weight "set-point" underlies their illness. Amitriptyline treatment, which can produce modest weight gain in anorexia nervosa (2, 3), produced obesity in the following case. This patient's body weight set-point may have been altered by amitriptyline, which raises questions about the neurochemical basis of this set-point and the relationship of anorexia nervosa to affective illness.

Case Report

Ms. A was a 20-year-old, 155-cm-tall woman with an ideal body weight of 47 kg. She had never been obese as a child, and reached her maximum weight of 46.5 kg at age 14. At 17, weighing 43.5 kg, she became amenorrheic. Significant weight loss began in February 1977. In July, weighing 28 kg, she was hospitalized on a psychiatric service. On admission, she was emaciated and had a distorted body image, compulsive hyperactivity, and a marked fear of obesity. She denied hunger and experienced early satiety upon eating. Her affect was sullen and depressed, with prominent feelings of helplessness and worthlessness. However, other psychiatric symptoms or signs were absent. A thorough medical and neurologic work-up was negative.

The patient was placed on a behavior modification regimen and gained 10.5 kg over an 8-week period. She was then placed on an ad lib diet and lost 2.5 kg over 2 months. In November, weighing 36.0 kg, she was started on amitriptyline, 100 mg/day. Within 8 days her appetite increased and she gained 1.0 kg. She was then discharged to outpatient therapy. Over the next 7 weeks she gained over 1 kg a week, weighing 49 kg by January. She stated that she felt hungry all the time and despite the consumption of large amounts of high-calorie foods rarely felt satiated. Her mood and outlook brightened and she was less sullen. Her weight gain slowed so that the next month she weighed 52.5 kg. At this time she stopped taking amitriptyline. Her weight gain continued despite the return of a depressed mood, helplessness, hopelessness, and, this time, suicidal ideation. By March her weight was 59.5 kg and had appeared to stabilize.

In summary, while on amitriptyline, Ms. A went from a stable, partially treated state of anorexia nervosa to moderate obesity. Despite the discontinuation of the drug, she continued to gain weight, reaching a weight more than 25% over her ideal weight. While she was on amitriptyline, her eating pattern changed from one of lack of hunger with early satiety to persistent hunger and lack of satiety.

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Discussion

Amitriptyline is thought to act predominantly by blocking the reuptake of serotonin (4). The neurochemical control of appetite and body weight, which is poorly understood, probably involves several neurotransmitters. On the basis of animal work, however, it has been proposed (5) that serotonin is involved in determining body weight set-point such that increased functional serotonin would lead to an increased body weight set-point and would then produce increased appetite and decreased satiety until that new increased body weight was reached.

Anorexia nervosa has been shown to be genetically related to affective illness (1). One form of affective illness, bipolar disease, can be viewed as an abnormality in a CNS set-point function, in this case of mood. The close relationship between the set-points of mood and body weight is demonstrated by observations going as far back as Kraepelin (6) that depression is associated with anorexia and weight loss and that hyperphagia, often accompanied by weight gain, occurs in hypomanic and manic states. In a recent case report (7), both mood and body weight set-points were "reset" by pharmacologic intervention; combined chlorimipramine (a specific serotonin uptake blocker) and tryptophan produced obesity and hypomania in a depressed anorectic woman.

Some individuals with anorexia nervosa may have an abnormality in body weight set-point that is related to the abnormality of mood set-point that occurs in bipolar affective illness. Just as in a depressed person without a bipolar diathesis amitriptyline can produce a gradual return to euthymia, so amitriptyline often produces mild weight gain (8). But, again, just as in a person with a bipolar diathesis amitriptyline can produce a marked swing of mood set-point resulting in hypomania or mania (9), in certain cases of anorexia nervosa it may produce a wide swing in body weight set-point resulting in obesity.

In summary, in this case amitriptyline therapy was associated with the production of obesity in a patient with anorexia nervosa. Because of the phobic fear of obesity in many anorectic patients, this is a potentially significant side effect of amitriptyline treatment for this condition. I postulate that amitriptyline produced this effect by its actions on the serotoninergic body weight set-point system. I further suggest that the underlying pathophysiology of those cases of anorexia nervosa that alternate with spontaneous or drug-induced obesity may be an abnormal body weight set-point. This abnormal body weight set-point may be closely related to the abnormal mood set-point of bipolar affective illness.

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Intrapatient Responses to ECT and Tricyclic Antidepressants

BY WILLIAM CORYELL, M.D.

This paper reports the results of a chart review comparison of the therapeutic efficacy of ECT and tricyclics in patients who received ECT during one depressive episode and a tricyclic during another.

Method

The records of all inpatients with depressive illness who received ECT at the Iowa Psychiatric Hospital between 1920 and 1959 were screened for those patients who had records of subsequent discrete episodes treated on an inpatient or outpatient basis between Jan. 1, 1961, and Dec. 31, 1976. The resulting sample of ECT-treated depressive episodes was therefore not biased toward greater severity by the availability of antidepressant therapy. Because the Iowa Psychiatric Hospital has traditionally been a tertiary care center, a large number of patients received tricyclic therapy at the hospital after 1960 only when similar attempts elsewhere had failed. This obvious

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source of bias was avoided by eliminating all patients who had received more than 4 days of antidepressant therapy before referral. To ensure that the episode in question was distinct from that involving earlier ECT treatment, patients who had not had an intervening period of treatment-free remission were eliminated from the sample. Selection criteria also required that ECT and antidepressant treatment efforts meet the criteria for adequacy defined in an earlier chart review by Avery and Winokur (1). Finally, all patients met *Research Diagnostic Criteria* (*RDC*) criteria (2) for primary major depressive disorder.

The present study and that by Avery and Winokur (1) defined treatment response identically, except that the response rater in this study was blind to the patient's identity and therefore rated responses to each of the two treatment modalities without knowledge of the nature of response to the other. Notes by all physicians and ward personnel made over a 4-day period before discharge and a hospital course summary were used to assign patients to one of three categories: not improved, improved, and completely recovered. In cases of outpatient treatment efforts, outcome was assessed from the clinic note that fell closest to 6 weeks after the treatment was started. The rater used the "completely recovered" category only when no evidence of symptomatology could be found among those notes.

Results

Of the 3,264 individuals admitted with depressive disorders between January 1920 and December 1976, only 42 had admissions during both of the defined periods (before Dec. 31, 1959 and after Jan. 1, 1961). Only 11 inpatient and 4 outpatient tricyclic antidepressant treatment efforts satisfied the remaining selection criteria.

The mean ages at the time of ECT and antidepressant treatment efforts were 44.3 years and 55.1 years respectively (see table 1). The duration of depressive symptomatology before the onset of treatment was nonsignificantly longer for ECT treatment efforts. However, duration of hospitalization was significantly shorter than that for antidepressant treatment efforts.

An exceedingly high rate of recovery was noted among ECT treatment efforts. Only 1 out of 18 efforts (3 patients had 2 separate episodes treated with ECT) showed less than complete recovery. Recovery rates were also high for antidepressant treatment efforts; 8 of 15 patients were categorized as completely recovered. Five patients manifested some improvement while only two patients showed no improvement to antidepressant treatment. The difference in response ratings between treatment groups was significant ($\chi^2=7.70$, df=2, $p<.025$). Since no patient failed to respond to ECT, response to antidepressants in previously ECT-resistant patients could not be addressed. The one ECT treatment effort that was rated improved rather than recovered was followed by complete response to antidepressants in a later episode.

TABLE 1
Comparison of Intrapatient ECT and Tricylic Antidepressant Responses

Item	Treatment Episodes: ECT (N=18)*	Treatment Episodes: Antidepressant (N=15)
Mean age at time of treatment (years)	44.3	55.1
Mean duration at onset of treatment (weeks)	18.1**	13.1
Mean duration of hospitalization (days)	36	49***
Rating (in percent)		
Completely recovered	94	53
Improved	6	33
Not improved	0	13

*Includes 3 patients who received ECT for 2 separate episodes; thus, percents are based on number of treatment efforts rather than number of patients.
**$p<.02$, two-tailed t test.
***Applies only to 11 inpatients.

Discussion

These data provide evidence that the efficacy of ECT is superior to that of the tricyclics when these treatments are compared *within* individuals. Such findings are complementary with another chart review study (1) and with well-controlled evaluations of ECT versus tricyclics (3).

Although there is no literature indicating decreasing efficacy of tricyclic antidepressants with age in a given individual, there is a good deal of evidence that the length of depressive episodes increases with age. This factor may have influenced the results reported here. In this sample, however, duration of episodes at treatment onset did not differ significantly between ECT and tricyclic antidepressant treatment efforts.

Other factors included in this design had the potential to reduce artificially the apparent efficacy of tricyclic antidepressants. The group excluded because of inadequate duration of treatment effort might have included patients who responded quickly. As it happened, in this sample all patients excluded because of an inadequate trial of tricyclics required ECT before discharge.

The response patterns presented here are appropriately compared with those noted by Avery and Winokur using identical rating methods. In a large sample reduced by relatively few exclusion criteria, 49% of patients receiving ECT alone showed complete response, whereas only 26% of patients receiving adequate antidepressant therapy alone were so rated. The relatively high quality of response to both modalities we observed was unexpected and deserves comment. The selection criteria resulted in a sample featuring recurrent and definitely discrete episodes. Onset tended to be abrupt. Since patients who experienced good

treatment response in an earlier episode are more likely to go to the same treatment facility without first seeking help elsewhere, our selection probably biased the sample toward patients who respond well to ECT.

Data from a similar but larger study would be relevant to the issue of specificity of different treatment modalities. The present study illustrates the difficulty in obtaining a sample that is both large and unbiased.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Other Views on Women in Psychiatric Residency

SIR: We are quite concerned that some of the serious discriminative abuses outlined by Elissa P. Benedek, M.D., and associates in "Problems for Women in Psychiatric Residency" (November 1977 issue) are occurring in any psychiatric residency program(s) in this country. Unfortunately, the authors did not report where, over what period, and with what frequency the incidents occurred. We would like to provide a contrast: the experience of women residents at the University of Pittsburgh. None of the 12 women currently in training could recall a seductive supervisor. No woman resident has been barred from a clinical service or from working with a particular type of patient because of her gender. Committee assignments, administrative responsibilities, and chief residency appointments are equally available to all residents who merit them. Both men and women residents plan and participate in study, athletic, and social group activities.

Qualified applicants for psychiatric residencies are in increasingly short supply. This is another reason why no resident should feel that he or she must tolerate a discriminatory training program. Women, like all minorities, are at risk for scapegoating in environments that permit such practices. Where each resident is respected as a colleague and dealt with fairly, all should benefit.

We consider the Benedek and associates article unduly polarizing. Men too can have unfortunate experiences with paternalistic or authoritarian supervisors who are unable to separate their personal attitudes from their supervisory efforts. In informal meetings in our program, as well as in residents' process groups, men express feelings of isolation, loneliness, depression, and/or conflicts between professional training and family needs as often as women do. Residents of both sexes have potential problems of prolonged dependency on (or fear of surpassing) a research, clinical, or administrative mentor. In some cases, although male residents share similar concerns (e.g., safety), they may permit the women to appear more anxious and fragile by speaking of these concerns for their female colleagues only.

There are, of course, some problems not shared by male residents. The resident who is pregnant or nursing a child must have a flexible work arrangement. Admittedly, it is not simple to reconcile the needs of the resident and her family with the needs of other residents and the patients. It certainly is true that there are insufficient female role models for women in academic psychiatry. Two solutions we find helpful are interactions with outstanding women from other programs (most often at national meetings) and the use of more senior women residents within our own program as role models. We also agree that many supervisors lack cognitive knowledge of female psychology, a subject that is often omitted from curriculum planning. We would recommend, instead of a separate course on the psychology of women, the integration of male and female psychology into the psychiatric curriculum for residents and for their supervisors.

In summary, we endorse Dr. Benedek and associates' attention to the problems of women residents. However, we must criticize their ambiguous data and their polarization of issues along exclusively gender lines. We hope that those concerned with psychiatric education will not be distracted by their few glaring examples and miss the more subtle, but extremely important, issues that are also described in their paper. We agree with the substance of their concluding recommendations, with the provision that these should apply to, and will benefit, *all* residents and their patients. Finally, a systematic collection of data on the current situations of women residents in psychiatry is clearly needed.

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SIR: We would like to dissociate ourselves from the remarks of Dr. Benedek and associates in the November 1977 issue lest they be seen as speaking for all or most women in American psychiatry. There is no doubt that women in medicine do face some very special problems, most of which are related to the constant pull between career and family obligations—a struggle that men entering medicine also experience. That these problems are magnified by training in and practice of psychiatric medicine, however, seems to be more a product of the rather narcissistic view of the authors than the specialty itself. Placing the "supervision" experience at the core of psychiatric training, the authors perceive particular difficulty in dealing with attitudes of aggression and sexuality of supervisors, whether male or female. The authors see women residents as victims of homoerotic fancy, chauvinistic competition, or savage mothering by females and patting, handholding, dinner invitations, or "even more flagrantly seductive ways" of stereotyping chauvinists of male gender and deplore their exclusion not only from the "unique and real relationships" women crave with their peers but also from committee assignments, team leadership, and individuation. The latter process would indeed do well to have been completed considerably before undertaking psychiatric residency.

We cannot wait for supervision to achieve our identity as women. A woman's gender identity is formed long before—

from parents and grandparents, teachers, Girl Scouts and camp, schoolyard brawls, parties and proms, from elections and love affairs lost and won. Bewailing the absence of female role models as administrators in psychiatry, the authors do not seem to have entertained the notion that many women have eschewed administrative posts because they are too smart to get stuck with them. Administration is tiresome and unrewarding compared with patient care or the struggle for enlightenment in library and laboratory and is seen by many of us as an unutterable bore, suited for those whose appetite for power is equaled by their tolerance for ennui and frustration.

Finally and most seriously, the authors seem to have missed the point on the "projection of an image of competency." Contrary to their assertions, it has very little to do with handling a microphone with aplomb. It has everything to do with doing a job well, with dignity, ability, courtesy, and compassion. Respect and admiration from male colleagues will be earned by first-class work, not by harangue or even consciousness raising. As for research careers—allegedly closed to women because of absent role models, geographic inconvenience, or insufficiently "nurturant" males—their main ingredients are curiosity, keen observation, conjecture, imagination, discipline, and long hours, and few men or women can achieve such careers without tremendous sacrifice by themselves and their families. This brings us to what seemed the most glaring omission in the paper—families. It is extremely difficult for a man or woman to combine a full family life and a major career commitment without an extraordinary trinity behind him or her . . . the spouse, children, and, in the case of a woman, the housekeeper. At least for the present, the career woman with a family is still the mother and is therefore principally responsible for child care and homemaking. Most men with significant careers in medicine already know that spouses of doctors must be "superpeople"—superconfident, supergiving, and supertrusting. As women working for equal opportunity in a demanding profession, we must be pretty well past the stages of individuation and nurture-demanding. We enter a medical career not to individuate but because we *are* individuals and find joy and fulfillment in giving time, energy, and, one hopes, health to people who need our services. As for nurturing, medicine is not a restaurant; perhaps only the reasonably well-fed should apply.

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Dr. Benedek and Associates Reply

SIR: We appreciate the various responses we have received to our article, some formal, some informal, expressing varying degrees of support of or disagreement with our viewpoint. We feel this indicates that our chief goal in writing the article has been reached, namely, raising an area of concern and facilitating a reaction in our readers.

First of all, we wish to clarify that the views and vignettes presented in the paper were not idiosyncratic to us. Editorial space constraints precluded elaboration of the fact that this paper was an outgrowth of a panel on the unique problems of female residents in psychiatry, originally presented at the American Psychiatric Association annual meeting in 1973. The five panelists, residents, or individuals who had recently completed training, presented their own personal experiences and also incorporated those of many other female colleagues from training programs in different parts of the country.

It is important to point out that our motivation in presenting the panel was not to generate research data about women residents but to raise some relevant issues in order to stimulate comparison and contrast by the audience. In fact, the subsequent discussion led us to present a similar panel the following year that paired the original panelists with male counterparts to stimulate even more audience participation. Although the material presented by the men was very relevant and important from the standpoint of residency training, and we acknowledge a certain commonality of stereotyping experiences by both sexes, we chose to focus on the female experience because we wished to emphasize that some of the problems faced by women residents are unique to them because of their gender.

One criticism leveled at us was that our paper seemed very negative as well as idiosyncratic. We certainly were not saying that the entire residency experience, or any segment of it, was basically negative. Indeed, had that been the case, none of the women involved would have stayed in their training programs or perhaps even in the field of psychiatry. Our goal was to highlight certain issues, not for purposes of condemnation but to facilitate awareness and constructive change where appropriate.

Some women have written with sharp claims that none of the phenomena described by us apply to their program. We are glad that some residency programs apparently have moved beyond the problems we outlined, but we think that too many other programs have not reached this desirable state. For example, shortly before the publication of our paper, one of the authors had the experience of having a young male psychiatric faculty member comment to her: "I don't see why we're taking so many women into our training program when we're not geared up for them." He was referring to the fact that, for the first time in the program's considerable history, women constituted 50% of the total number of residents accepted.

To further pursue the ideas formulated in the published paper and the APA panel, a limited pilot survey of residents was conducted in 1976–1977. It indicated that the problems are still very much a part of the residency scene. Some of the preliminary results of this survey were reported in two panels at the 1977 APA annual meeting in Toronto—"The Pregnant Therapist" and "The Woman Hospital Psychiatrist."

GAIL M. BARTON, M.D.
ELISSA BENEDEK, M.D.
CHRISTINE BIENIEK, M.D.
Lansing, Mich.

Roles and Responsibilities of Psychiatry

SIR: In "The Psychiatrist's Responsibility and the Public Trust" (February 1978 issue) Peter Bourne, M.D., outlined the current dilemma in American psychiatry so well that I was shocked to see him lose his perspective and line of reasoning toward the end of the paper. He said, "The major challenge to psychiatry in the next decade is to make our skills available to all Americans" (p. 176). Is this not as gran-

diose and unreasonable as our accepting the burden of changing society, for which Dr. Bourne castigated us severely?

The training of paraprofessionals is a loaded issue. Why do we not train adequate psychiatrists, if we are the specialists in human behavior and the physicians of mental illness? Do we need psychiatrists' assistants? If so, then let us train and supervise them. Let us define the roles of mental health paraprofessionals and pay them adequately.

The comparison of psychiatrists with Rolls Royce craftsman when Fords are needed is a sign of the degradation to the "marketing personality." If 30% of the population needs treatment, then it needs expert treatment, not assistance. If the remaining 70% need help in ordering their lives, then experts in causes for disordered lives should be called in.

When our special body of knowledge exposes the psychopathology rampant in this country, will we be accepted? I am all for a definition of our role and skills. Would Dr. Bourne start the ball rolling?

HELEN STEIN, M.D.
Fresno, Calif.

Dr. Bourne Replies

SIR: Unfortunately, Dr. Stein's concern arises from a misinterpretation of what I said in my article. The statement "The major challenge to psychiatry in the next decade is to make our skills available to all Americans" was not meant to imply that we should train everyone in psychiatric skills. Perhaps I should have said (for greater clarity) that we should try to make our services available to the largest possible number of Americans. This would mean reducing the financial barriers to care, perhaps through national health insurance; training additional psychiatrists; and increasing the involvement of psychologists, psychiatric social workers, and family physicians in areas that do not require psychiatrists. Presumably this would make psychiatrists more available to a larger number of people. Obviously, paraprofessionals can play an important role if they have appropriate training and supervision.

PETER G. BOURNE, M.D.
Washington, D.C.

Dealing with Patients' Suicides

SIR: I would like to comment on the article "Patient Suicide as Part of Psychiatric Residency" by Ralph F. Henn, M.D. (June 1978 issue).

I agree with the author that patient suicide is not an infrequent occurrence during a psychiatric residency. One survey of a university psychiatric residency program indicated that 16% of all residents had had a patient who committed suicide (1). Although this is lower than the figures quoted by the author, it is nevertheless a significant percentage.

The author states that professional "survivor victims" had "bitterness and torment." I would like to share one method of dealing with this. When I was a resident, I was one of four trainees who had a patient commit suicide within a 2-month period. We met together for almost a year to deal with the trauma. We found that the experience of a patient suicide left each of us with feelings of embarrassment, guilt, and self-doubt. We were embarrassed because we felt that we were at fault and that patients of competent trainees and competent psychiatrists did not commit suicide. Some of us felt that we would be afraid ever to see a suicidal patient again. Initially we had each felt alone in our experience. Fortunately, we became aware that we were not unique and decided to meet as a group. Through our group meetings we were able to support each other and work through many of our feelings about the suicide (2).

I would suggest that since this is a common experience in psychiatric residencies, openness be encouraged so that when residents do have patients who commit suicide, they can meet in groups that involve self-examination in a supportive atmosphere. This may help alleviate the pain and loneliness of the experience. In addition, working through feelings about a patient suicide can allow the resident to continue to grow professionally, and to become willing to treat other suicidal patients.

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RENÉE BINDER, M.D.
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Anthropological Cross-Fertilization

SIR: We enjoyed the excellent paper "Overview: Foundations of Cultural Psychiatry" by Armando R. Favazza, M.D., and Mary Oman, M.A. (March 1978 issue).

There have been several attempts to meld psychiatry and anthropology into an interdisciplinary frame of reference. Although these efforts have met with general approval from both fields, there has been insufficient use and follow-through of the extremely important cross-fertilization resulting from such collaboration.

We believe much can be achieved by intimate teamwork between a psychiatrist and an anthropologist or ethologist. Dr. Favazza and Ms. Oman have attempted to cover the broad range of mutually beneficial exchanges and are to be congratulated on their thorough job. We should, however, like to add to their list two areas in which we ourselves have been working.

We have investigated the persistence in humans of phylogenetically embedded response systems that are no longer adaptive, the circumstances that lead to their activation, and their potential to emerge as psychoneurotic, psychophysiological, or psychosomatic disturbances. This frame of reference provides a biological basis that lends itself to incorporation into a medical model without requiring one to resort to metapsychological concepts. It also provides a biological basis for some psychodynamic formulations (1).

We also believe that an unexpected bonus for anthropology is available from psychiatry. Some of these behavioral "fossils" can provide material to aid our understanding of the social interactions of the precursors of homo sapiens. In many instances such behavioral vestiges provide clues that are as valuable as anatomical ones in deciphering the stages

of human emergence (2). For instance, the biological anomaly of a 4:1 ratio of male to female dyslexics and a 2:1 ratio of male to female infant vulnerability of the brain to febrile convulsions, among other clues, set us on a path to radically new ideas about the origin of language (3). Additionally, ethological insights about the nature of existing mother-infant releaser-response mechanisms shed light on the initial emergence of language primarily as a reinforcer of mother-infant bonding behavior rather than a vehicle for communication. We also think that certain features of schizophrenia may represent an earlier biological norm (4).

A helpful fallout of these theoretical investigations has been the enrichment of psychotherapeutic procedures through biologically based psychodynamic understanding (5, 6).

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A. DAVID JONAS, M.D., M.R.C. PSYCH.
DORIS F. JONAS
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Dr. Favazza and Ms. Oman Reply

SIR: The team of A. David Jonas, a psychiatrist, and Doris F. Jonas, an anthropologist, has produced a series of interesting papers that place human behavior patterns in an evolutionary and ethological context. For example, in their paper on schizophrenia they write,

> Paranoid ideation is readily comprehensible when we remember that almost all animals in their natural surroundings remain constantly on the alert for possible danger at all hours of the day and all the days of their lives, so that it is not surprising that we human beings retain a sense of being on guard against menace from the environment, even if this menace be figurative.

Even though ours was an overview paper, we were unable to include every branch of the field of cultural psychiatry. The work of the Jonases is provocative and may eventually clarify our understanding of certain aspects of human behavior. More attention might be given to their psychiatric work if they could demonstrate clearly its relevance to the resolution of everyday clinical problems. However, any attempt to reduce the "medical model" to biology alone will not be successful. Every person has a body and therefore biology is significant. But every person also has a mind, and every person lives in a society and participates in a culture. The physician who can integrate biological, psychological, social, and cultural data as they pertain to the individual patient is practicing the medical model in its truest sense.

ARMANDO R. FAVAZZA, M.D., M.P.H.
MARY OMAN, M.A.
Columbia, Mo.

Treating Tricyclic Overdose with Physostigmine

SIR: Tricyclic overdose is seen whenever these drugs are prescribed for depressed patients. The *Physicians' Desk Reference* (1) states, "Physostigmine Salicylate is reported to reverse symptoms of tricyclic antidepressant poisoning" (1, p. 1090). Listed in order of increasing plasma levels, these symptoms are "abnormal deep tendon reflexes, unconsciousness, respiratory support, ventricular rate ≥120/min., QRS duration ≥100 msec., cardiac arrhythmias, bundle branch block, cardiac arrest, seizures and death" (2).

A 25-year-old woman took a questionable amount of amitriptyline, and the drug showed on the toxicology report. When she came to the ward her pupils were nonreactive, she showed no response to verbal stimuli, her muscles were flaccid, and the nail beds were cyanotic. Blood pressure was 100/68, pulse 140. On a random ECG the longest recorded QRS interval was 90 milliseconds. An I.V. was started. She became progressively unconscious.

It was decided to give physostigmine. Vital signs at the time of injection were as follows: blood pressure 84/64, pulse 140, respiration 40. An immediate grand mal seizure lasting one minute was noted, followed by possible cardiac arrest. A "blue alert" was called, including use of a monitor, oxygen, and suction. The blood pressure stabilized at 152/84, but the patient remained unresponsive, and a second injection of physostigmine was administered. This was followed 10 minutes later by a second convulsion.

No more physostigmine was given. The patient was treated with Lanoxin and other supportive measures and made a recovery to her pretoxic mental status in about 2 days.

Verbal contact with D. G. Spiker of Western Pennsylvania Psychiatric Institute leads me to believe that the physostigmine injections caused the two grand mal convulsions. As Spiker has written, "Whenever medication is used, care must be taken to understand the exact physiological mechanism that is to be altered by the drugs, and efforts must be made to avoid potentially fatal iatrogenic drug interactions" (3).

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M. VERNON ORDIWAY, M.D.
Ridgway, Pa.

On Assessing the Usefulness of the PANESS

SIR: I found the Clinical and Research Report entitled "Clinical Usefulness of the NIMH Physical and Neurologi-

cal Examination for Soft Signs" by Janet A. Camp, M.Phil., and associates (March 1978 issue) particularly distressing. The readers of the *Journal* will be turned off to a diagnostic tool (PANESS) because of negative conclusions drawn by the authors on the basis of a woefully inadequate study. In fact, the only valid conclusion that can be drawn from the study is that we simply do not know the clinical usefulness of the PANESS as a diagnostic tool.

In comparing normal and hyperactive children with respect to their PANESS performance, the authors indiscriminately and without justification lumped together 56 scores representing most of the items comprising PANESS. Is it reasonable to expect that every one of 56 responses to a neurological test battery is equally discriminative of diagnostic groups, or discriminative in the same direction? This is the assumption that must underlie the authors' lumping of the subjects' test scores.

In fact, it was not necessary to lump these scores in order to make comparisons between groups. Multivariate statistical procedures permit comparisons of groups simultaneously along more than one dimension. Individual scores (items) are weighted by these procedures so that optimum separation of groups is achieved. The proper statistical procedure for the Camp and associates study is discriminant analysis or multivariate analysis of variance. In an article on "Anomalies of Motor Development in Hyperactive Boys" Denckla and Rudel (1) arrived at very different conclusions from those of Camp and associates, as an example of the application of this procedure.

Suppose we did accept the authors' use of the lumping procedure and the t test statistic as the valid method of group comparison (neither procedure is, in fact, valid). Out of the 7 age groups for which hyperactives and normals were compared, the Ns for 5 of the hyperactive groups were 2, 5, 4, 2, and 0. Reliable statistical conclusions simply cannot be drawn on the basis of such small samples using t tests. Finally, the authors did not discuss their finding of a significant difference in the opposite direction from that predicted. It may be an artifact of the multiple t test procedure (some significant differences can be expected by chance when many t test comparisons are made), but at least it should have caused the authors to pause and consider the validity of their approach to the validation of the PANESS.

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MELINDA BROMAN, PH.D.
New York, N.Y.

Ms. Camp and Dr. Bialer Reply

SIR: Dr. Broman's statement that "the only valid conclusion . . . is that we simply do not know the clinical usefulness of the PANESS as a diagnostic tool" is precisely the point of our paper, as was clearly stated in our last paragraph. Our intent *was* in fact to "turn off" psychiatrists from using the PANESS as a diagnostic tool until its validity is demonstrated. Our data, in conjunction with those of Werry and Aman (1), are meant to caution physicians against accepting this instrument prematurely.

The PANESS is a first attempt at a standardized soft sign examination and is intended to be administered in its entirety. Our purpose was not to modify the instrument but to ascertain its diagnostic utility. Obviously, not all items discriminate equally well, as an initial stepwise multiple regression analysis of our data indicated. However, items that do discriminate would of necessity raise the total scores of hyperkinetic children. The PANESS items represent a cross-section of typical soft sign examinations. Consequently, they cannot validly discriminate in the "opposite direction," as Dr. Broman suggests, since by definition an item is not considered a clinical soft sign if it is present in normal children. Thus, if the instrument were valid, the total score measure would in fact reflect the higher incidence of soft signs in hyperkinetic children.

The small sample size in a number of the hyperkinetic age level groups underscores the diagnostic problem with the PANESS. The practicing physician wishing to use the examination diagnostically is dealing with a single child. Any soft sign instrument that shows statistical differences based on large samples but cannot detect the presence of these signs in an individual case is worse than useless for diagnostic purposes. Although our study may be statistically inelegant, our findings remain both statistically and practically correct: The PANESS in its current form is an inadequate diagnostic tool for the practicing psychiatrist or pediatrician.

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JANET A. CAMP, M.PHIL.
IRV BIALER, PH.D.
Stony Brook, N.Y.

Postprandial ECG

SIR: I have a question regarding the article "Factors Influencing Phenothiazine-Induced ECG Changes" (January 1978 issue) by Henry A. Nasrallah, M.D. Should not the "obvious implications" of the provocative effect of food intake be that the ECG should be done postprandially at a time when the blood glucose level is likely to be maximal? Under these conditions, which are analogous to the glucose tolerance test in diabetes, any risk to the patient from a food-drug interaction would probably be revealed.

A.A. SCHIFF, M.R.C.S.
Twickenham, England

Dr. Nasrallah Replies

SIR: A fasting pretreatment ECG remains the best baseline measurement with which to compare subsequent ECGs, whether postprandial, posttreatment, or both. If the intent is to observe the phenothiazine effects alone, then the posttreatment ECG must be measured in the fasting state.

Both food intake and phenothiazine treatment can produce benign ECG changes that are reversible by an overnight fast (1, 2). An increase in intracellular-extracellular K^+ ratio has been suggested to mediate both (3). Thus, Dr. Schiff's suggestion is well taken in that the occurrence of ECG changes postprandially before phenothiazine treatment

might be predictive of similar ECG changes after phenothiazine treatment. However, I believe that the overnight fast is more useful in detecting persistent ECG abnormalities, which, if not reversed by K^+ intake (4), can portend higher risk for phenothiazine-induced arrhythmias.

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HENRY A. NASRALLAH, M.D.
La Jolla, Calif.

Cardiovascular Excitability and Emotional Stress

SIR: In his article "Emotional Causes of Sudden Death" (December 1977 issue) Joel E. Dimsdale, M.D., reviewed several papers which showed that the cardiovascular system may respond to emotional stress in an unfavorable way even in healthy individuals.

Our studies of attitude change in neurotic patients under different degrees of emotional arousal yielded a result that may have some relevance to Dr. Dimsdale's article. We discussed therapeutically relevant topics with patients who were artificially aroused through inhalation of adrenaline. We found that these patients exhibited significantly greater cardiac lability when those topics were introduced in a subsequent drug-free session than did patients who had been treated in the same way but with a placebo inhalant (1).

This finding suggests that a person can be conditioned to a state of heightened cardiovascular excitability by a topic even if this state was originally triggered by a different mechanism and only accidentally became associated with the topic. Therefore, it is possible that those persons who experience frequent stressful situations develop excitatory cardiovascular responses not only to the actual stress situations, but coincidentally to other situations which had only minimally affected the autonomic nervous system. In such cases, the resulting damage to the cardiovascular system could greatly exceed the amount expected from the summation of the individual stress situations.

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RUDOLF HOEHN-SARIC, M.D.
Baltimore, Md.

Dr. Dimsdale Replies

SIR: I appreciate Dr. Hoehn-Saric's calling his work to my attention. The evidence relating emotional factors to heart disease is, as I indicated in my review, particularly compelling because of the convergence of findings from different approaches. The major thrust of Dr. Hoehn-Saric's work was to investigate attitude change in psychiatric patients who were exposed to physiological arousal by adrenaline inhalation (1). A serendipitous but important finding was that combining adrenaline inhalation with discussion of therapeutically relevant topics resulted in the patient's "learning" a response of autonomic arousal when later confronted with the same topics. This arousal was manifested by significant changes in heart rate variability while the patient was discussing the focal topic, even when no adrenaline was inhaled.

This work is a logical progression of one of the studies quoted in my review. Newton and Gantt (2) demonstrated in dogs that a persistent change in heart rate could be learned after a single conditioning situation.

We tend to regard the autonomic nervous system response to emotion as an automatic consequence of arousal as opposed to a learned response. Recent work on conditioning the autonomic nervous system (3) is frequently perceived as a radical innovation in normal physiology. The intriguing implication of Hoehn-Saric's work is that such "learning" in the autonomic nervous system is the norm, not the exception, that biofeedback occurs in daily life.

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JOEL E. DIMSDALE, M.D.
Boston, Mass.

Prescription Drug-Induced Organic Brain Syndrome

SIR: We were very interested in the Clinical and Research Report "Prescription Drug-Induced Organic Brain Syndrome" by Toshihiko Maruta, M.D. (March 1978 issue) because we were recently baffled by a similar case.

A 37-year-old truck driver was transferred to our hospital from another psychiatric facility for a full neurological workup and further psychiatric care. He had been at the other facility for approximately two months and had been ill for two months before that. The main symptoms of his illness were sudden work avoidance (with a record of 15 years with the same company), withdrawal, crying spells, episodes of staying in bed, and, finally, suicide threats, which prompted his involuntary hospitalization. Over this four-month period (two as a private psychiatric outpatient and two as an inpatient), the patient had received a total of nine different medications—tranylcypromine, imipramine, desipramine, amitriptyline, perphenazine, chlorpromazine, haloperidol, flurazepam, and biperiden—at different times, with no change in his condition. In fact, before he was transferred to our unit, the patient had had decreased mobility, slurred speech, marked blepharospasm, loss of tongue movement, left facial weakness, confusion, disorientation, and decreased cognitive functioning. In short, he was believed to have had a cerebro-vascular accident. Prior to his symptoms that began

five months earlier, this patient had never visited a psychiatrist or shown symptoms indicating that he should.

Under our care, the patient was taken off all drug therapy, which at that time consisted of haloperidol, desipramine, and biperiden. The results of the neurological workup, which included electroencephalography, skull series, brain scan, and CAT scan, were all negative except for the EEG, which showed some focal slowing of the postereotemporal and occipital regions on the left. The patient began to improve, showing normal mobility, more spontaneous coherent speech, increased interest in himself and his surroundings, and general improvement in his cognitive functioning. A series of three more EEGs was done over the ensuing 22 days with a gradual return to normalcy by the time the last one was done. The patient maintained his improvement, and was discharged without medications on the 24th hospitalization day. Three weeks later he was back at work.

This patient may well have undergone the full episode of depression with return to mental health despite all treatment; however, we feel that the prescription drugs, especially the use of polypharmacy, clouded and augmented his illness. This points out the necessity for regular review of medications and the justifications for them.

WINSTON W. SHEN, M.D.
ANGELA M. WOOD, M.D.
New Orleans, La.

Psychopathology of the Team Concept

SIR: I would like to comment and expand on a point discussed in the article "Becoming an Administrator: The Vicissitudes of Middle Management in Mental Health Organizations" (March 1978 issue) by David M. Dressler, M.D.

The treatment team, which should be or once was a useful therapeutic modality in psychiatry, has been misused, distorted, and politically exploited. As with other therapeutic modalities, the improper use of the team concept can have detrimental, or even fatal, side effects.

In a treatment facility like Napa State Hospital, the members of the treatment team are the psychiatric technician, social worker, psychologist, rehabilitation therapist, occupational therapist, and other lay personnel. The ward psychiatrist or physician then acts as supervisor of the treatment team. However, the supervisory capacity of the psychiatrist has been undermined by the use of vague terminology such as "member of . . ." or "consultant to the treatment team." The function of the physician has been further confused by PRU 57, which does not adequately define the roles of administrators or team leaders when they are nonmedical professionals. Moreover, the addition of the "team leader" (based on the "herd" concept of the social scientist; that is, the strongest in the herd is the leader) has further obscured the supervisory role of the psychiatrist. Unfortunately, the homogenous "wild herd" does not have specialty members or licensure restrictions.

There are many ways in which the treatment team can become pathologic. The psychiatrist may merge imperceptibly with the treatment team and lose his identity. When this happens, the psychiatrist can no longer function to monitor psychiatric treatment. Conversely, the team members may lose their identities. There is overlapping of the roles of the various disciplines, or there is blurring or loss of ego boundaries. (The team members begin to respond to stimuli within the team rather than to external stimuli—that is, to the patient, who can no longer be viewed objectively.) This resembles the autistic thinking of the psychotic. Reality testing has become impaired.

The team member may fail, by omission or commission, to give the feedback of his own specialty or discipline. The team member may cross boundaries and dominate other disciplines, suppressing important feedback from those disciplines, thus misleading the team. It is here that the monitory capacity of the psychiatrist or ward physician should come in.

With regard to the team leader, the position of coordinator can be misused, and a supervisory and decision-making position be assumed. When a nonphysician team leader starts making decisions for the group, the danger of pathological decisions exists, especially if the process cannot be controlled by the supervisory capacity of the psychiatrist.

The psychiatrist's or ward physician's supervisory role has been blurred by the addition of the team leader and lay administrator. This has weakened and confused the physician's supervisory role. Is this a political maneuver of the paramedical workers to gain control and power in the mental health field? When the physician is a weak supervisor, he can easily be overruled by a strong team leader or the rest of the treatment team. Pathologic coalition and conniving cannot be halted if the psychiatrist is given one vote in the treatment team. On the other hand, the treatment team can be addicting to the physician; more and more he relies on the treatment team and no longer uses his discretion, ignoring even his legal responsibilities.

When the psychiatrist or ward physician becomes too dominant, he may suppress the initiative, enthusiasm, and motivation of the team members. In order to avoid this situation, it would be advantageous to have another psychiatrist or physician available for consultation. However, with PRU 57, the physicians have been so isolated from each other, especially under nonmedical program directors, that there is no mechanism for mutual feedback and peer review. Is this another political exploitation of the system?

The addition of lay administrators, which is due to the lack of doctors, has made the team concept more complex. The original purpose of the lay administrator was to assist the physician in his administrative duties so that the physician could devote more time to clinical work. PRU 57 has allowed the lay administrator to supervise psychiatric technicians, social workers, psychologists, and rehabilitation therapists. This supervisory capacity has been misused and has now extended to the treatment team and to the physician. Occasionally, lay administrators have even overruled the team decisions and the clinical decisions of the physician. The lay administrator may become a dictator in his own program, ignoring the treatment needs of the patient.

Another difficulty is that the educational background of the lay administrator may not be adequate to allow him to perform peer review of some of the professionals he supervises. For example, a program director who is a social worker cannot perform peer review for a psychologist. The system becomes more implausible when the nonmedical program director has no one to review his work: PRU 57 has no built-in mechanism to review the program director's work.

The only solution to the problems of the pathologic treatment team is to start redefining roles of various treatment team members. As for PRU 57, it should be either revised or discarded.

MARIANO A. FAVIS, JR., M.D.
Napa, Calif.

Dr. Dressler Replies

SIR: The letter by Dr. Favis raises important issues concerning the design and operation of clinical management teams and, more importantly, the role of psychiatrists in multidisciplinary organizations. The team concept can be extremely valuable when patients have multiple problems and when organizations are structurally complex, with many staff members and diverse decentralized programs. Although the leader of the team need not be a psychiatrist, his role relationship to team members and the sponsoring organization needs to be clearly and appropriately defined with respect to level of responsibility and authority (1). The psychiatrist may serve as a primary care clinician, medical back-up physician, team leader, clinical supervisor, or consultant. Although he may function in more than one capacity, vague or contradictory role definitions lead to ineffective team functioning, which ultimately has an adverse effect on the quality of patient care.

The role of psychiatrists in mental health organizations is undergoing significant change at the present time. Other professional disciplines are demanding a more significant role in decision-making processes affecting patient care, thereby challenging the traditional autonomous prerogatives of the psychiatrist. Although these changes may be to some extent both reasonable and appropriate, the psychiatrist will gradually lose his central position as a health care provider unless he is willing to assume positions of significant responsibility in mental health organizations. Unfortunately, consumer pressures and governmental regulations seem to be opposing this objective. Improved public relations on the part of concerned groups such as APA and increased emphasis on mental health administration in residency training programs may help to reverse this trend.

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DAVID M. DRESSLER, M.D.
New Britain, Conn.

Usefulness of Lithium for Aggressiveness

SIR: Four years ago I published a report on the usefulness of lithium in aggressive prisoners (1). However, subsequent clinical reports have attested to the effectiveness of lithium in preventing aggressive outbursts (2, 3). The composition of groups studied has varied, including subjects with chronic psychosis, personality disorder, brain injury, and epilepsy. Furthermore, aggressive children with various diagnoses have been noted to improve with lithium administration. In the face of an array of clinical diagnoses, common underlying features have been suggested and include 1) undiagnosed, atypical affective disorder, 2) family history of affective illness, and 3) affective elements apparent in the illness. Traditional diagnosis alone will not be enough, nor is a description centered on affective illness likely to be complete. Rather, I would suggest that there is a common underlying neuropsychological disturbance which is behaviorally manifested in the following ways: 1) extreme stimulus sensitivity—a "hairtrigger," 2) the inability to reflect on the meaning or intent of the stimulus, i.e., the lack of reflective, introspective review to assess accidental or purposeful attack, and 3) maximal response—little capacity to modulate the expression of anger.

In cases I have seen, the absence of the capacity for reflective consideration of the meaning of the stimulus seems to be most important. After lithium was given the person still retained some stimulus sensitivity, although it was reduced, and could on occasion be angry and even violent. However, the intervening step of reflective consideration of the appropriateness of a violent response had been strengthened, allowing the person to constructively review the nature and setting of the stimulus.

Since these three characteristics do not imply any psychiatric diagnosis per se, they form a behavioral triad that may be worthy of further investigation and may be useful in identifying these individuals with explosive violence who might benefit from lithium. Furthermore, these descriptions may suggest a basic strategy for study of these explosively violent individuals.

I offer these suggestions not as a final definition of a lithium-responsive, violent population but rather as a point for future investigation in this important area.

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JOE P. TUPIN, M.D.
Sacramento, Calif.

Correction

In "Obesity and Psychoanalysis" by Colleen Rand, Ph.D., and Albert J. Stunkard, M.D., in the May 1978 issue, the last sentence of the precis is incorrect. The decrease in the percentage of obese patients suffering from body image disparagement was from 40% to 14%. Also, in the section Body Image Disparagement, the second sentence of the second paragraph should read, "Twenty-nine obese patients reported severe and 35 mild body image disparagement. . . ."

There was an error in the reference list for "Sudden Infant Death Syndrome: Impact on Families and a Direction for Change" by Stanley E. Weinstein, M.S.W., in the July 1978 issue. Reference 4 should be "Bergman AB: A Study in the Management of Sudden Infant Death Syndrome in the United States. Seattle, Children's Orthopedic Hospital and Medical Center and the University of Washington School of Public Health and Community Medicine, 1973, p 11."

The Official Actions section of the August 1978 issue (p. 1028) carried the heading "The American Board of Psychiatry and Neurology Commission on Certification in Administrative Psychiatry." The Commission on Certification in Administrative Psychiatry is not part of the American Board of Psychiatry and Neurology but a component of the American Psychiatric Association.

The staff regrets these errors.

BOOK REVIEWS

Psychopharmacology: A Generation of Progress, *edited by Morris A. Lipton, Alberto DiMascio, and Keith F. Killam. New York, N.Y., Raven Press, 1978, 1,731 pp., $49.50.*

The purpose of this all-encompassing volume is well stated in its preface, which notes that it was developed by the American College of Neuropsychopharmacology for the purpose of providing a comprehensive survey of progress in that field. The reader who even scans the book will see that it performs this function particularly well.

Even a glance at the contents reminds us of the inroads the relatively new science of neuropsychopharmacology has made. To most of us it is a reminder that we have much—very much—to learn if we are to be competent in the understanding and use of these agents. The psychotropic drugs burst on the scene less than a quarter of a century ago, and for many psychiatrists their use for a while was empirical. There is no longer an excuse for this; the user now is required to be well acquainted with the uses and the possible abuses of these pharmacological agents. The drugs in question were the first really effective additions to the psychiatrist's therapeutic armamentarium. They are powerful agents, and expertise is required in their use lest the last condition of the patient be worse than the first.

The entire field is covered in this volume, beginning with the ethics of the use of the drugs, their biochemical bases, and their clinical utilization. Next, the mechanism of their action is reviewed on a basic molecular level, and the known biochemical and pharmacological actions involved in initiating or regulating neuronal transmission are explained.

Data are presented on the use and the role of psychotropic drugs in the overall treatment program for various categories of emotionally and mentally ill patients. No excessive claims are made for these medications, individual variations in responses to them are recognized, and some reasons are suggested for their actions.

Even to mention the subheadings in this extensive volume would require more space than we have here at our disposal. Starting with a historical overview by the editors and followed by a chapter titled "In the Service of Psychopharmacology Research: The Psychopharmacology Service Center-Psychopharmacology Research Branch (NIMH) Program 1956–1976" by Jerome Levine, the chapters follow as they should, i.e., "Strategies of Basic Research" by Seymour Kety and "Strategies in Clinical Psychopharmacology" by Heinz Lehmann. The mention of the names of these authors and those of other chapters in the section titled Neuroanatomical, Histochemical, and Neurophysiological Mechanisms of Drug Action would convince the reader that he or she is being instructed by experts.

The final segment of the book considers a few of the hazards associated with these powerful new agents along with a consideration of drugs that have been around for a long time and are often subject to misuse. A chapter on "Opiate Receptors and Opioid Peptides: A Ten-Year Overview" by Avram Goldstein is worthy of special note, as is a chapter on "Cannabis: A Review of Progress," by Louis Harris. The neurophysiology and neurochemistry of barbiturates and alcohol are reviewed by Michiko Okamoto.

Nancy Mello gives an authoritative statement on "Alcoholism and the Behavioral Pharmacology of Alcohol: 1967–1977," and Roger Meyer discusses the "Behavioral Pharmacology of Marihuana." Both of these are especially well done, but the same can be said of most of the other chapters. Apparently, nothing that has to do with psychopharmacology was forgotten. A reviewer is at his wit's end trying to even mention some of the excellent material contained in the volume. Pediatric psychopharmacology, geriatric psychopharmacology—you name it, it is all here, with notable stops to discuss clinical research, drug assessments, toxicology, and side effects. Taken together, a review of the subjects covered in this book would necessitate another volume of similar heft (1,731 pages!).

One thing I am sure of—the completeness of the work. It definitely encompasses a "generation of progress." It is a reference book that will be consulted for years to come. I can also state without fear of contradiction that no library concerned with any aspect of pharmacology would be complete without it. It was a prodigious task to get materials and authors together and properly orchestrate them. All concerned should be complimented. The volume is enthusiastically recommended.

F.J.B.

Adaptation to Life, *by George E. Vaillant. Boston, Mass., Little, Brown and Co., 1977, 396 pp., $9.95.*

This book describes the results of a 30-year follow-up study of adult development. The study began in 1938, when the men studied were college students at Harvard University. Dr. Vaillant has been able to follow 95 of the original 268 subjects. These men were interviewed approximately 30 years after they started college in the late 1930s and early 1940s. Dr. Vaillant had access to all of the information that was collected during the subjects' college years and during the intervening years, but most of the book is dedicated to describing his own follow-up interviews.

Dr. Vaillant followed his subjects literally to the four corners of the United States. These are fascinating men who comprise the upper crust of our society. Each has his own individual pattern of development, making it impossible to group them by types. They have characteristics that make them, at times, interesting, creative, intelligent, open-minded, and invigorating. At other times they come across as depressed, pathetic, prejudiced, and full of psychopathology. Obviously, they represent a continuous mixture of all of the above.

The book is aimed at the psychologically aware and intelligent reader. It presents, in the best of Robert White's *Lives in Progress* (1) tradition, the complexity of man and his

internal and external environments. It is extremely well written, flows easily, and presents the people whom Dr. Vaillant studied well and with little jargon. I believe that at times Dr. Vaillant's fear of alienating the intelligent public runs away with him. For example, he describes the two defense mechanisms of dissociation and projection used by one of his subjects and adds, "These *clumsy* psychiatric terms are defined in Appendix A; in this chapter, argot will be translated into English" (p. 16; my italics).

The book's major weakness is also its strength. It presents the lives of these Harvard men well, in readable fashion, and makes one believe that what happens in childhood counts later in life. Its weakness lies in the fact that there is no serious attempt to discuss the study's findings in the general context of the behavioral and social sciences. The scholarly review is weak, and the discussion of concepts of mental health is one-sided, value-laden, and superficial.

Dr. Vaillant seems to shy away from any serious attempt to validate his clinical observations by the usual methods of rating scales, psychological testing, pencil and paper tests, ratings of the interviews, or behavioral observations of others (e.g., spouses). He is obviously familiar with these psychological techniques, and he had enough subjects to make the use of other observational methods possible. I do not know why he elected not to do so. I believe that scientifically (from my biased perspective), it would have made *Adaptation to Life* a better book. One simple rating that would have been interesting is the self-rating of the interviewer of the subjects (Dr. Vaillant). It seems to me that what is called counter-transference in the therapeutic area ran rampant through the interviews. Ratings such as "How likable was the subject?" and "How warm, distant, anxious, depressed, confused, fatherly, motherly, the interviewer felt toward his subjects right after the interview and a week later" would have helped to objectify the observations. The lack of an index adds to the feeling that this is not a scientific treatise.

Returning full circle to my earlier statement, the above weakness might have strengthened the book for certain readers. It is easy to recommend Dr. Vaillant's book to anyone interested in the developmental psychology of adults. It is a worthwhile addition to the literature, and it is exceptionally well written.

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Clinical Psychopharmacology, *edited by Jerrold G. Bernstein, M.D. Littleton, Mass., PSG Publishing Co., 1978, 154 pp., $20.00.*

During the past decade a plethora of books on psychopharmacology have appeared. They vary from excellent textbooks to volumes summarizing and elucidating relevant psychopharmacological research to practical workbooks for the busy clinician (1–3). This small volume is a compilation of papers presented at a symposium held in Boston. Dr. Bernstein has done a fine job in editing this volume; all of the chapters are very readable and hold the reader's interest throughout.

The book begins with an overview of the biological basis of mental illness by Seymour Kety. Dr. Kety takes a historical tour through the findings that form the biological basis of psychiatry. He surveys both genetic and chemical data to demonstrate that mental illness has organic determinants, but he does not lose the reader in technical biochemical formulations.

Dr. Jonathan Cole's chapter on antianxiety drugs also reviews the biochemical basis of these medications. He explains how animal models are used to test the properties of antianxiety drugs. Clinicians who use minor tranquilizers should be aware of the laboratory basis for studying the behavioral properties of these medications. Although there are many references to comparison of these medications with placebo effects, one omission in this volume involves the specific issues of placebo phenomena. Dr. Cole ends his chapter with a clinical reminder of the often forgotten noxious effects of minor tranquilizers. An unusual side effect of benzodiazepines, for example, is hostile, irritable behavior.

David Greenblatt's chapter on insomnia is complemented by useful, easy-to-read tables. He provides a nice overview of the medical and psychiatric causes of sleep disturbance. He also clearly discusses the various advantages and difficulties of commonly used hypnotics. One area not covered is how to take a careful sleep history to rule out such other sleep disorders as sleep apnea and narcolepsy (4).

Dr. Bernstein's chapter on the chemotherapy of psychosis provides an overview of neuroleptic medication. He offers facts about the side effects of these medications and outlines the various phases of antipsychotic drug treatment of severely ill psychiatric patients. Dr. Alberto DiMascio's chapter on long-acting psychotropic drugs complements the previous chapter on the acute psychoses.

Dr. Gerald Klerman's contribution on the psychopharmacological treatment of depression is very interesting. He discusses the incidence of depression and especially the increased frequency of this disorder in women. His brief discussion of available treatments for mood disorders provides a balanced overview of psychological, social, and organic interventions. Dr. Bernard Levy's discussion of atypical manic-depressive illness takes into account Adolf Meyer's observation that manic-depressive disease has a multifaceted presentation. Excellent clinical examples of syndromes of acute psychosis in adolescence, recurrent paranoid psychosis, and recurrent depressions in very successful individuals remind the reader that bipolar illness can occur in various forms. Dr. Keith Conner and Dr. Rachel Gittelman-Klein provide two chapters on psychopharmacology of children. Their chapters overlap in content but provide information about the approach to the pharmacological treatment of childhood disorders, with emphasis on childhood depressions as well as hyperkinetic phenomena.

A chapter on the medical aspects of psychotropic drugs includes commonly seen side effects of these medications. Part of the difficulty with publishing a book on psychopharmacology is the rapid advancement of knowledge in this field. This chapter is already outdated because of a recent study by Burckhardt and associates (5) showing a persistent at-rest tachycardia in patients receiving tricyclic medication. However, the data provided in this chapter are generally useful to clinicians. It would have been helpful if information about the hepatotoxic effects of these medications had been included. The book concludes with chapters on drug abuse and its management and a brief statement on the patient's right to know about psychotropic medications.

This book provides an opportunity to refresh one's general knowledge about psychopharmacology. However, it could

have given over more space to the approach to diagnosis of psychiatric conditions. Without the ability to diagnose, rational treatment is often difficult. Another useful addition would be a further discussion of Donald Klein's approaches to anxiety and depression (6). Despite these omissions, this book would be useful for nonpsychiatric physicians, for psychiatrists who rarely use medication, and for other health professionals who are interested in psychopharmacology. Its readability and size also make it useful for medical students.

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THOMAS N. WISE, M.D.
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Research on the Effects of Television Advertising on Children: A Review of the Literature and Recommendations for Future Research. Report Prepared for the National Science Foundation and Research Applied to National Needs (RANN). *Washington, D.C., U.S. Government Printing Office, 1977, 229 pp., $3.75 (paper).*

This is an extensive and quite technical research report summarizing the present state of research and knowledge regarding television advertising as it affects children. It recommends a plan of future research on the subject. A definitive verdict about the effects of television advertising on children is disclaimed, but any intelligent reader will draw some conclusions from the material presented.

Questions about the effects of television on children have concerned professionals and parents for the last two decades. Considerably more attention has been given to the effects of television violence on children, at least in journals directed toward health professionals. Articles on the subject of violence are numerous and often impassioned—rightfully so, since statistics document the fact that by the time they have graduated from high school most children will have spent more time in front of the television set than in school. Television has become one of the dominant forces affecting development, acculturation, and learning of society's moral and ethical values.

This report suggests four fundamental concerns that have to do with whether the products advertised on television are safe, whether the vulnerability of children is exploited, whether the specific techniques used are deceptive or misleading to children, and whether the culmulative exposure may adversely affect development. As is usual, three groups are involved in the debate: industry, government, and consumer organizations. The report points out that relations among these groups have often been contentious. Both industry and consumer groups attack the government, but industry tends to complain of too much government interference, while consumer groups tend to complain of too much timidity on the part of governmental agencies. The consumer groups also believe that industry is less concerned with the possible harm to children than with selling advertising, while industry accuses the consumer groups of being self-appointed guardians who do not represent the views of the majority of parents.

By means of a questionnaire survey of interested parties, a review of the literature, and consideration of which issues were amenable to empirical testing that might also subsequently affect national policy, the research group arrived at the following list of 10 issues: 1) children's ability to distinguish television commercials from program materials, 2) the influence of format and audiovisual techniques on children's perceptions of commercial messages, 3) source effects and self-concept appeals in children's television advertising, 4) the effects of premium offers in children's television advertising, 5) violence and unsafe acts in television commercials directed toward children, 6) the impact on children of proprietary medicine advertising, 7) the effects on children of television food advertising, 8) the effects of volume and repetition of television commercials, 9) the impact of television advertising on consumer socialization, and 10) television advertising and parent-child relations.

A chapter of the report is devoted to each of these issues, and these chapters become the substance of the report. Although the chapters vary in length from 6 to 18 pages, each consists of an introduction, a discussion of the current regulations, a discussion of incidence, a discussion of research evidence, a summary, and, finally, recommendations for additional research. Thus, although the report is probably overly detailed for most readers who have some interest in the subject, its structure makes it easy to absorb the general ideas without dwelling on all of the particulars.

In formulating their recommendations for future research the authors believe that three levels of action are necessary: one to test specific hypotheses, another to document how children perceive actual individual commercials, and a third to better understand the role of television in children's lives. "Ultimately," they conclude, "the research should lead to creation of standardized measures for testing commercials to insure that they are truthful, accurate, and fair to children's perceptions."

The remainder of the report consists of a technical evaluation of 21 primary empirical studies of television advertising. A statistical summary of the responses to the survey questionnaire mentioned above is included. Official guidelines are reproduced as they pertain to children. Finally, an extensive bibliography is offered for those who might want to read in more detail on certain aspects of the report.

This report will be of particular interest to mental health professionals who deal with children, especially those interested in child development, child rearing, parent education, and primary prevention of mental disorders.

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Respectful Treatment: The Human Side of Medical Care, *by Martin R. Lipp, M.D. Hagerstown, Md., Harper & Row Medical Department, 1977, 222 pp., $11.50 (paper).*

Respect is the most important value in human relationships. Everyone knows what is meant by self-respect and

respect for others, but both concepts are hard to define. The author wryly quotes H.L. Mencken's cynical definition of self-respect as "the secure feeling that no one, as yet, is suspicious." Dictionaries have trouble defining the term. The *Concise Oxford* misses the target ("regard with deference, esteem, or honor") and becomes more meaningful only when the definition is made operational ("avoid degrading or insulting or injuring or interfering with or interrupting, treat with consideration, spare, refrain from offending or corrupting or tempting"). Perhaps the Declaration of Independence comes nearest to capturing the meaning. "All men are born equal" may be untrue except as a political ideal or as an article of faith, but respect implies a sense of basic human equality.

Lipp's short book is a highly successful attempt to assist the physician in making operational his or her respect for the patient, and particularly for patients of psychiatrists in a general teaching hospital. The author is intimately familiar not only with acute psychiatric wards but with all those parts of a good teaching hospital in which psychiatric advice is sought—major surgical wards, special care units, dialysis units, oncology wards, and emergency departments. He writes with a firm grasp of the philosophical, theoretical, and practical issues that confront the physician assisting in the treatment of grossly disabled or disfigured persons, chronically or terminally ill patients, and "problem patients" of every variety—unreasonable, angry, petulant, overly affectionate, addicted, psychotic, suicidal, and violent.

There is a valuable chapter on the family of the patient, and a final chapter (very practically written) concerning the necessity of the physician's having self-respect and the "occupational hazards of physicianhood." The section on psychotherapy and counseling initially ruffled my feathers, but after rereading I found no quarrel with it. The relationship between any physician and his or her patient is the powerful vehicle of all treatment, and the variously labeled psychotherapies enhance that power in critically important ways—yet only weakly.

The book's title is apt to be misleading, suggesting another motherhood sermon. Lipp even sells himself short in his preface—"My primary aim is to provide a handbook in practical psychiatry for non-psychiatrists." I would strongly recommend the book as valuable or even essential reading for all medical students and physicians, including psychiatrists of differing orientations and levels of experience. It is certainly a must for any psychiatrist working as a consultant in an acute general hospital.

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Attention, Voluntary Contraction and Event-Related Cerebral Potentials. Progress in Clinical Neurophysiology, Vol. 1, *edited by John E. Desmedt. Basel, Switzerland, S. Karger, 1977, 253 pp., $37.75.*

Auditory Evoked Potentials in Man: Psychopharmacology Correlates of Evoked Potentials. Progress in Clinical Neurophysiology, Vol. 2, *edited by John E. Desmedt. Basel, Switzerland, S. Karger, 1977, 207 pp., $37.75.*

In the last two decades a great deal of new and valuable information about brain functioning has been obtained by analyzing the evoked electrical activity recorded from the intact human scalp. This event-related brain activity has been useful in gaining a better understanding of sensory, perceptual, and cognitive processes in the human brain. More recently, the recording and analysis of evoked neuroelectric activity has provided new diagnostic capabilities to assess functional neuropathology.

The first chapter in volume 1, written by Donchin, Callaway, Cooper, Desmedt, Goff, Hillyard, and Sutton, represents basic methodological criteria for studies of evoked potentials in man. This is an important contribution that should be heeded by every worker in the field. Chapter two, by Desmedt, is also about methodological issues and is equally important. The next two chapters, by Skinner and Yingling, deal specifically with cerebral mechanisms underlying selective attention and should definitely be read by all those concerned with attentional mechanisms. The authors present a novel formulation involving a specific gating process at the thalamocortical level. This thalamocortical process interacts with a lower brain stem reflexive mechanism and results in selective filtering of sensory experience or reflexive orienting responses.

A well-written chapter on the motor and sensory determinants of slow potentials by Syndulko and Lindsley completes the section on selective attention and slow potential shifts. This chapter provides an elegant demonstration of the influence of motor and sensory factors on cortical slow potential shifts in man. Although the three papers in this section are all innovative and excellent, the section as a whole suffers from the glaring omission of chapters dealing with recent human data on the relationship between event-related potentials and attention. The rest of this volume is composed of several well-written chapters on voluntary contraction and event-related potentials.

Volume 2 presents the current state of the neurophysiological mechanisms involved in far-field and late auditory evoked potentials and their clinical uses in man. The various chapters in this section will undoubtedly become indispensable reading to all investigators in this field. The chapter by Goff and associates is an excellent paper on the neural sources of short latency components of the auditory evoked potential in man. The clinical applications of auditory evoked potentials are discussed in a number of well-written chapters (Galambos and Hecox, Starr, Rapin and Schimmel, and Peronnet and Michel). These and other chapters will be of great interest to those concerned with the use of electrophysiological techniques to assess brain dysfunction.

The last section of the book includes three chapters dealing with the effects of various drugs on evoked potentials in man. The chapter by Lader offers a rather brief but valuable discussion of the methodological difficulties encountered in using evoked potentials to assess the action of psychotropic drugs. Lewis, Dustman, and Beck's chapter provides a unique description of the acute and chronic effects of marijuana intake on cortical function. They present strong evidence for the persistent deleterious effects of chronic use of relatively small doses of marijuana. The chapter by Saletu is an excellent demonstration of the use of evoked potential techniques to conduct retrospective and prospective studies of the action of psychotropic drugs on the CNS.

These first two volumes in the series can be considered an important contribution to this area of research. However, the comprehensiveness of the material and the sequence of topics is somewhat enigmatic and leaves something to be desired. Careful attention should be devoted to the choice and logical sequence of subject matter.

At present these two volumes comprise the most current review of some of the most active areas of research dealing

with evoked brain potentials in man. As such, these books would be most valuable to researchers in these areas and to those interested in using neurophysiological techniques to study brain function or to assess brain dysfunction. These books have the additional value that most of the contributors seem to have spent time considering and elucidating the conceptual aspects of the work being performed in their areas of expertise. These two volumes make a considerable contribution to the field of human neurophysiology and provide new insights that will be of lasting value.

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The Borderline Patient, *by Roy R. Grinker, Sr., M.D., and Beatrice Werble, Ph.D. New York, N.Y., Jason Aronson, 1977, 222 pp., $12.50.*

Ten years after the publication of *The Borderline Syndrome* (1), two of the authors of that major piece of research have come forward with its clinical companion. *The Borderline Patient,* which is based on the research of the previous book, has been supplemented by additional work undertaken in response to methodological criticism of the earlier study. It is intended "to clarify for the clinical psychiatrist what the borderline patient is and what he is not" (p. vi). The authors add, "We wished to describe individual patients so that the clinical psychiatrist could more easily diagnose his own patients and thereby know their prognosis and appropriate treatment if any" (p. 210).

In approaching the subject of the borderline patient, Grinker and Werble begin with the firm conviction that the medical model, with its emphasis on observation, clinical description, diagnosis, and classification, must constitute the foundation of the science of psychiatry. In addition, they operate with two basic assumptions about the borderline syndrome. First, they believe that schizophrenia is not equivalent to psychosis and that the concept of latent or borderline schizophrenia is not viable. Second, they believe that their earlier research established that borderline patients exhibit a group of core phenomena consisting of "developmental distortions of ego functions. These are anger as the main or only affect, a defect in attaining and maintaining affectional relationships, absence of adequate self-identity, and depressive loneliness" (p. 7).

Within this conceptual framework, the authors describe 19 patients given the diagnosis of borderline and assigned to four subgroups. After reviewing the literature and presenting thorough case histories of the 19 patients, the authors consider, in a compact, well-organized chapter, the problem of differential diagnosis, a topic all too rarely explored. Although they consider a case of "narcissistic neurosis," the distinction they draw between borderline patients and patients with narcissistic character disorders is insufficient. Similarly, since the diagnosis of schizo-affective schizophrenia is not discussed in any detail, it is hard to tell how the authors differentiate borderline patients from those with schizo-affective schizophrenia.

The book contains a chapter on the family of the borderline patient in which the authors acknowledge that the small sample size limits the conclusions that can be reached. A chapter on follow-up studies leads to the final chapter, which explores the "stable instability" that the authors believe to be characteristic of these patients.

The Borderline Patient is well written with a refreshing minimum of poorly defined psychiatric and psychoanalytic terms that muddy so much of the literature on this subject. Although one could differ with the particular subgroup which the authors assign some of the patients described, the overall effect of their work is to clarify the clinical manifestations of this confused and confusing syndrome.

Striking in this research is the low incidence of the borderline syndrome: only 14 of 300 consecutively hospitalized patients received this diagnosis. However, both this book and *The Borderline Syndrome* consider only hospitalized patients, thus preselecting those with more severe ego deficiencies; the incidence of the borderline syndrome in nonhospitalized patients may be much greater. The psychodynamic formulations offered with each case description are brief, rather superficial, and sometimes not well substantiated by the material presented.

My major serious reservation about this book grows out of the author's therapeutic nihilism regarding psychotherapy with borderline patients, a position apparently reached as a result of their follow-up studies. Without elaborating the kind of psychotherapy their patients received or presenting data to justify their conclusion, they take the position that borderline patients "require direction, control, and education rather than insight, since they do not have the capacity to develop a transference neurosis suitable for psychoanalytic interpretations" (p. 215). Even if one believes that borderline patients do not develop a transference neurosis characteristic of classical psychoanalysis, they do develop powerful transferences, and, as many writers have convincingly demonstrated, they are certainly able to utilize psychoanalytic interpretations. The authors' dismissal of the usefulness of insight-oriented psychotherapy with borderline patients is unwarranted.

Grinker and Werble have provided us with a most helpful guide to diagnosing the borderline patient, but they are on shaky ground when they move from this area into consideration of appropriate treatment for these patients.

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DAVID I. JOSEPH, M.D.
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Child Psychiatry: Treatment and Research, *edited by Mae F. McMillan, M.D., and Sergio Henao, M.D. New York, N.Y., Brunner/Mazel, 1977, 305 pp., $15.00.*

A collection of the writings of several authors is often said to be the easiest book one can produce. However, this book is too good to have been collected and edited without hard work and struggle. Drs. McMillan and Henao have brought into usable and sensible shape a number of symposium presentations that must have had more roughness and more diversity in earlier forms than they have in print.

Editorial rigor has not homogenized the final product, however. The authors' personal styles persist in the 16 chapters of the publication—styles ranging from the restrained tone of Sally Provence to the witty and erudite review by Norman Garmezy to the fragmentary, sophomoric, and unorganized commentary of Michael Baizerman. The good is mixed with the not so good. Some of the styles and sub-

stance of different chapters stand out as essays of extraordinary merit. Worth the cost of the entire book is the brilliant, tightly reasoned, and judicious article by Saul Harrison titled "Reassessment of Eclecticism in Child Psychiatric Treatment." That chapter is praiseworthy through and through. For other chapters it is only difference from its neighbors that occasionally gives a nearly drab chapter some spiciness, imparting a welcome change of pace, as seen, for example, in the chapters by Lee Robins and Eric Wish, Judith Wallerstein, Sally Provence, and Ronald Liebman and associates.

Not even the lingo of public health bureaucracies subdues the sprightliness and human glow of Margaret Morgan Lawrence's chapter on inner city families. An example of the lingo is her question, "What are the effects of deficits existing in mother, infant, family, and community, including deficits in nurturing over several generations, on infant-caretaker interaction?" (p. 79). "Infant-caretaker" must mean mother-child. Not even the pose of bantam rooster daring and dogmatism can salvage much of the chapter on asthmatic children by Ronald Liebman and associates from the Philadelphia Child Guidance Clinic, however. More than assertive oversimplification is required to sell their patriarchal product: "It changes the role of the father in the family by increasing his involvement in a constructive manner." Again, intricate statistical ploys, caveats, and jugglings cannot breathe life into the chapter by Robins and Wish, who studied a group of 223 black men in their developing "deviances." From page 254 we learn, "Qualitative relationships were tested by an age-adjusted actuarial method followed by tests for spuriousness which required holding constant acts that were presumptive causes of both members of a pair of acts as well as the total number of prior acts."

Peter Wolff, on the other hand, interestingly supports the view that child psychiatrists must look at maturation apart from chronological age. Wolff presents material on CNS maturation and behavioral development that, although not startlingly novel, is provocative of thought and consistent with reason:

> The left cerebral hemisphere has been characterized as processing by focal, analytic, or serial strategies, and the right hemisphere as extracting information by the diffuse, holistic, gestalt mode or by strategies of "parallel" processing. . . . Such characterizations are only a rough approximation of actual conditions, but they have the heuristic advantage of grouping a large number of heterogeneous psychological tasks under a few formal rules, and of directing research efforts to a functional analysis of brain mechanisms.

The chapter by Milton Shore is lightly informal but does provide some sustenance. One is assured that Shore has indeed been taught a lot by delinquent adolescents; would that the same were true of other writers on adolescents in this volume.

Several typographical errors mar this otherwise attractive piece of work. Page 263 stirs our curiosity with an obvious misprint—"female-to-female transsexuals." Page 144 would lead us to believe that Dr. Stickney was succeeded as Commissioner of Mental Health and plaintiff in a class action suit in Alabama by an unknown, unheralded figure: "*Wyatt v. Anderholt.*"

Overall, this is a worthwhile, far-ranging, and representative compendium of the field of child psychiatry. It will not be timeless, but we can derive reassurance that as rapidly as it has been superseded the scientific footing of child psychiatry will have been strengthened. Its sampling is a good one for now.

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Endangered Hope: Experiences in Psychiatric Aftercare Facilities, *by David K. Reynolds and Norman L. Farberow. Berkeley, Calif., University of California Press, 1977, 180 pp., $10.95.*

In the introduction to this short informative study the authors state their intention to explore, using an experiential research design, the impact of placement of suicidal patients recently treated in an inpatient psychiatric facility into residential aftercare facilities.

One of the authors (David Reynolds), with a case history based on his own life history, entered a number of aftercare facilities as a "discharged patient" to make firsthand observations of the facilities and gather data about his own reactions and the reactions of the owners and residents to him. His experiences and the brief experience of one other researcher are the major focus of the book. From these experiences, the authors have formulated conclusions and recommendations regarding this phase of treatment for the suicidal patient.

Despite the authors' intention, the study offers relatively little insight into the specific consequences of placement in an aftercare facility on depressed and suicidal patients. Rather, the book's primary value is in highlighting clearly the impact of these settings on all mentally ill persons and in offering constructive recommendations to those involved in these settings as to how they can have a more positive effect on the clients. This volume is particularly timely because of the current concerns about the process of deinstitutionalization and the need for developing appropriate and effective residential settings in the community that can serve as transitions from the hospital to independent living.

The authors delineate four stages that characterize the process encountered by the discharged patient who enters a residential aftercare setting. Although only briefly described, the stages of newcomer status, interim phase, membership phase, and termination phase are valuable concepts that can be used by other researchers in further describing this process.

There are other important subjects that the authors touch on. In some instances they make important contributions to our knowledge. These include a description of the role of owner-managers that highlights the need for the development of adequate training programs for them, 2) the second-class status often felt by residents, in comparison with staff, and the kinds of staff actions that can decrease the stigma of placement in a residential facility, and 3) the conclusion that an effective network of residential facilities must include a variety of settings in which programs are specifically targeted for different types of patient problems and for different levels of independence.

Despite this book's many substantive contributions to our knowledge about placement and care in residential aftercare facilities, there are some shortcomings that call for further research and exposition. In only one of the settings was the research conducted over a fairly lengthy period of time (four weeks). In all other settings, the researcher remained for a much briefer period of time. Furthermore, the two researchers spent time together only in one setting, thereby limiting

the opportunity for consensual validation of their observations, an important methodological consideration. Further research of this nature should allow for a longer period of placement of the observer-researcher in such a setting to more accurately delineate the four stages conceptualized by these authors.

The authors pay insufficient attention to the perceptions of the residents about their own reactions to the pseudo-patient. In emphasizing the reactions of the owners, I think the authors have overlooked an equally important area for study.

In summary, *Endangered Hope* is a valuable addition to our limited existing literature on the experiences of mentally ill individuals who require placement in a residential aftercare setting as part of their treatment plan. In the light of our increasing awareness of the need to focus greater attention on the problems faced by those with chronic mental disabilities, it is my hope that further research in this area will take place.

ALLAN BEIGEL, M.D.
Tucson, Ariz.

Psychotherapy for Better or Worse: The Problem of Negative Effects, *by Hans H. Strupp, Suzanne W. Hadley, and Beverly Gomes-Schwartz. New York, N.Y., Jason Aronson, 1977, 343 pp., no price listed.*

This volume is devoted to the question of whether psychotherapy can have harmful effects or increase a patient's suffering. Those familiar with Dr. Strupp's previous work will not be in the least surprised to find a highly readable, well-researched, and well-referenced discussion of this issue. They will also find that Dr. Strupp and his colleagues have thought of and discussed almost every question that this issue might bring to mind.

The book consists essentially of two parts, the first of which could be called "Dr. Strupp and His Colleagues" and the second, "The Respondents." There are three main sections to the first part: a presentation of the problem of negative effects with a discussion of the relevant literature, a detailed analysis of previous psychotherapy studies relating to claims of harmful or negative effects, and the proposal of a model for evaluating the mental health and psychotherapy experience of a patient.

This model, called the "tripartite model" by Strupp, is based on the fact that three major parties are concerned with a patient: society (including significant people in the patient's life), the individual patient, and the mental health professional. From these three he obtains three readings of the state of a patient, and these three readings make up what he calls the BWS configuration: B is society's perspective, concerned principally with the adaptive qualities of the behavior of the patient; W is the individual's own sense of well-being; and S is the evaluation of the mental health professional, who bases judgment on an assessment of psychological structure. Strupp says that these dimensions should be considered simultaneously in evaluating mental health and changes related to psychotherapy.

The authors make clear in their analysis of previous studies that almost every study has had some serious flaws, and the only study that they feel fulfills certain criteria in terms of selection, methodology, design, etc., found a very low rate of negative effects.

The second part of the book is in some ways even more enjoyable and stimulating than the first. It consists of the replies of a number of well-known people in the fields of psychiatry and psychology to a letter sent to them by Dr. Strupp. In his brief letter he asked them to respond to three questions that can be summarized as follows: 1) Is there a problem of negative effects in psychotherapy? 2) If so, what would constitute a negative effect? 3) Which factors do you consider mainly responsible for a negative effect?

The replies of 70 respondents are printed here, and there is a chapter discussing the "expert's opinions." The replies vary in length from five lines to Paul Meehl's 16 pages, and they are all well worth reading.

Certain points emerge repeatedly, to the extent that I think it is fair to speak of a consensus among psychotherapists. Abram Kardiner wrote in *My Analysis with Freud* (1), "Freud was always infuriated whenever I would say to him that you could not do harm with psychoanalysis. He said, 'When you say that, you also say that it cannot do any good. Because if you cannot do any harm, how can you do good?' " This opinion seems to be shared by most of the respondents, and other areas of agreement also seem to be present.

Exacerbation of symptoms, hospitalization, overt psychosis, suicide, long-term lowered self-esteem, loss of trust in the therapist or in therapy, and excessive dependency on the therapist are considered negative effects by psychotherapists.

There also seems to be fairly wide agreement about a number of factors that can lead to negative effects. Prominent among these are inadequate assessment of the patient, unsuitability of the patient for psychotherapy, poor motivation on the part of the patient, pathology of the therapist (especially hostility and seductiveness), the therapist promising more than he or she can deliver to the patient, and conveying lack of respect and lack of hope to the patient. For the most part, the onus is very much on the therapist, which emphasizes the great difference in outlook between psychotherapists and organically oriented psychiatrists.

Of course, some of the respondents took the opportunity to express their hostilities toward people or approaches they disapproved of, and a considerable number (16 out of 70) advertised their own book or paper, but as a whole the responses make very interesting reading and give us a good representative picture of therapists' thoughts and experiences in this area.

Two replies are especially noteworthy: Goldberg's (page 263), characterized by its clear conceptualization and excellent synthesis of what constitutes negative effects and why they occur, and the lengthy response of Meehl on page 306.

Some experienced therapists may read this book and feel that the authors have not told them anything new. There is a well-known Talmudic saying: "Know how to answer one who comes to ridicule or attack you," and the authors make it clear that one of the main reasons that this book was written was because "contemporary society [has a] critical view of psychotherapy," and "legislators, insurance companies . . . consumers . . . have begun to demand from the mental health professions clear-cut answers to the extraordinarily difficult and complex questions concerning what psychotherapy does and how it does it."

The thoughtful and concerned psychotherapist will benefit greatly by reading this well-written and nicely produced book. It will give the psychotherapist a much fuller understanding of these issues and a feeling of support from a con-

siderable number of outstanding contributors in the field of psychotherapy. It is more permanent, more enjoyable, and much cheaper than a three-day symposium on the topic.

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JOSEPH BERGER, M.B.B.S., B.Sc.
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Suicide: Inside and Out, *by David K. Reynolds and Norman L. Farberow. Berkeley, Calif., University of California Press, 1978, 237 pp., $3.85 (paper).*

This volume details the experience of a pseudo-patient (David Reynolds) who was admitted to a Veterans Hospital for a two-week stay. The presenting complaints are carefully prepared, the case history is well documented, and the pseudo-patient's subjective responses are nicely recorded. The suicidal story is central to the authors' thesis. The authors recognize the highly subjective nature of the experience and make serious attempts to understand patienthood within a special framework.

The book is useful reading from a number of standpoints. It offers, first, a good review of the pseudo-patient in a psychiatric hospital. It presents data on suicide in the psychiatric setting and tries to correlate the frequency of suicide with the absence of humanized behavior and the loss of self-esteem. It does not sufficiently separate suicidal thoughts from suicidal acts.

It is not easy for staff to deal with successful suicide in a psychiatric hospital. A suicide will cause a great deal of soul searching, and a psychological autopsy is a common sequel. The affect and emotional investment of staff are not dissimilar in spirit to the agitation of the surgeon when a patient dies on the operating table. The calculated risks taken in serious circumstances are forgotten in the face of the dramatic turn of events.

The authors recognize how, under the best of conditions and with close observation, many opportunities for suicide exist in psychiatric hospitals. It is impressive how few successful attempts occur in large psychiatric hospitals, where less than ideal conditions may exist. In the hospital where I work, more than 5,000 adults are admitted to the inpatient service per year. Over the years these patients have been increasingly more agitated, with a substantial proportion verbalizing suicidal ideation. Still, a check of the records shows only one successful suicide in the past 5 years. (An additional suicide in that period occurred on the prison service, a circumstance that deserves a review all by itself.)

I point this out to indicate that the book would have been just as valuable to me if the thrust had been away from suicide to a reexamination of the social structure within the hospital and its impact on patients. Humanizing the hospital setting is important not only for patients but for staff. Patients will respond with a greater sense of responsibility, and the hospital community will be more happily structured.

The diary of the patient is well worth reading. It is important to bear in mind that the sense of patienthood in the pseudo-patient is highly variable and that not all observers may slip so easily into the patient role. All in all, I found the book readable and its thesis "good." Others who read this may wish to speculate about other factors that may lead to suicide in the psychiatric hospital.

MARVIN STERN, M.D.
New York, N.Y.

Psychiatrists and the Legal Process: Diagnosis and Debate, *edited by Richard J. Bonnie, LL.B. New York, N.Y., Psychiatric Annals (Insight Communications), 1977, 345 pp., $5.00 (paper).*

The editors of *Psychiatric Annals* apparently discovered one day that in the brief course of that publication's existence a number of very interesting and some distinguished articles on legal psychiatry had been published. They decided that collecting these articles in a volume would be an addition to the legal psychiatric literature, which was almost nonexistent 20 years ago but is now expanding at a rapid rate.

As Richard Bonnie says in his introduction to this collection of 43 articles, "The mushrooming interest in cross-disciplinary work is reflected . . . in the number of treatises and texts that have been published in recent years." This volume is an addition to this burgeoning literature. The authors included represent a *Who's Who* of current legal psychiatry—among them William J. Curran (more identified with legal medicine but contributing an interesting historical paper on legal psychiatry), Seymour Pollack, Carl Malmquist, Abraham Halpern, Seymour Halleck, Donald Hayes Russell, Darold Treffert, Browning Hoffman, Stonewall Stickney, Alan Stone, and Loren Roth.

Like any collection of articles that have appeared in a periodical, the quality varies widely, but some of the articles are extremely important. Among my favorites is "The Chronically Mentally Ill Shuffle to Oblivion" by Robert Reich and Lloyd Siegel, which in 1973 called attention to the neglect of many of those caught up in the process of deinstitutionalization. Darold Treffert's consideration of "The Practical Limits of Patients' Rights" and Browning Hoffman's response to that position represent a dialogue that is worthy of a great deal of consideration, and Stonewall Stickney's retelling of his position in *Wyatt v. Stickney* is an important explication on the background of one of the major forensic psychiatric cases. Many other articles in this collection are equally worth reading and worth having in a convenient reference source.

Richard Bonnie, who is Professor of Law at the University of Virginia School of Law, has divided the articles into four categories: Roles of Psychiatrists in the Legal Process, Psychiatrists and the Criminal Process, Psychiatrists and the Juvenile Process, and Psychiatrists and the Mental Health Process. He has written very useful introductions, summaries, and commentaries for the articles in each section.

It would be difficult to read this collection from start to finish. It contains too many ideas, some of the points of view of the various authors are contradictory rather than complementary, and, in spite of the great efforts of the editor to give continuity and unity to the work, it lacks the bite and the interest of a work by one author. However, if it cannot be read comfortably from start to finish, many sections are extremely readable, and it certainly belongs in the expanding reference shelf of important legal psychiatric works.

One extended section of the book is a consideration of the insanity defense of Loftus E. Becker, Jr., Associate Professor of Law at the University of Minnesota; this represents

five articles that appeared in two issues of the magazine and is really a monograph on the subject. Three articles by Donald Hayes Russell (all from one issue of the magazine) similarly comprise a monograph on juvenile delinquency.

Much in this volume, like much in forensic psychiatry, is controversial. Such topics as the right to refuse treatment or the proper use of the concept of rehabilitation in criminal justice will continue to be debatable.

The title of this book is somewhat unwieldy. On the cover and on the title page the first part of the title, "Psychiatrists and the Legal Process," is put in smaller type than the rest of the title, "Diagnosis and Debate." Whichever of these titles is used, this compilation represents an impressive collection of authors and contributions and a remarkable illustration of the great attention that is being paid to a variety of legal psychiatric topics.

JONAS ROBITSCHER, J.D., M.D.
Atlanta, Ga.

Psychological Medicine: An Introduction to Psychiatry, 8th ed., *by Desmond Curran, M.D., D.P.M., Maurice Partridge, D.M., D.P.M., and Peter Storey, M.D., D.P.M. New York, N.Y., Churchill Livingstone, 1976, 434 pp., $14.50 (paper).*

This is a straightforward and, in my view, somewhat simplistic textbook written expressly for medical students. One has to take into account that this is an English textbook written for English medical students, who are warned against foreign influences. For instance, when writing about schizophrenia, the authors write, "It is important to realize that many psychiatrists abroad, particularly in the U.S.A. and U.S.S.R., would not share the views put forward here, and this should be remembered when reading foreign articles or books."

Psychotherapy is given short shrift and is deemed to be contraindicated in the treatment of schizophrenia, the author's opinion being that it "unfortunately tends to make schizophrenic patients worse." There is a curious chapter titled "Peripheral Psychiatry" that covers psychosomatic medicine, anorexia nervosa, nocturnal enuresis, stammering, sleepwalking, tics, spasmodic torticollis, acute intermittent porphyria, and electroencephalography, all in strange juxtaposition and contained in 12½ pages.

Although it becomes obvious that it is unlikely that this textbook will be used by American medical students, it has been reprinted 11 times and is in its 8th edition. Its description of group therapy is almost worth the price of the book: "Free discussion and straight forward self-disclosure is encouraged among not more than eight patients at a time—the ideal number, as Lord Houghton declared, for 'a polite dinner party,' though, especially as inter-relationship develops, the groups are not always 'polite.' "

RAYMOND VEEDER, M.D.
Hartford, Conn.

Thought, Consciousness, and Reality: Psychiatry and the Humanities, Vol. 2, *edited by Joseph H. Smith, M.D. New Haven, Conn., Yale University Press, 1977, 309 pp., $15.00.*

This collection of essays by five philosophers and four psychoanalysts achieves greater thematic unity than did volume 1 (1). It presents relevant reflections on thought, consciousness, and reality in two groups of essays: five papers under the general heading of Thought and Consciousness and four papers under the title The Construction of the Real.

The nature of thought and its relationship to reality appeared as problems very early in the history of Western philosophical inquiry. They received classic consideration in Plato's "Thaetetus," in which he advanced the idea that thinking is merely silently talking to oneself. The question continued to figure prominently in philosophical discourse throughout the centuries as part of epistemological considerations.

Freud became involved with the problem early in his work. Although his classic paper "Formulations on Two Principles of Mental Functioning" (2) was not published until 1911, he had been occupied much earlier with the nature of man's mental attitude toward reality. A high point in psychoanalytic concern with the character of thought was reached by David Rapaport in his *Organization and Pathology of Thought* (3).

This collection of essays largely takes its theme from the first and keynote essay by Stuart Hampshire, an English philosopher, titled "The Explanation of Thought." The paper constitutes a rethinking of Spinoza's double aspect theory in its relation to the nature of thought. Spinoza was concerned with the ability of reason to influence our actions and with the essence of human freedom. Parenthetically, the latter problem is discussed at length later in this volume in a critical essay by Isaac Frank titled "Spinoza, Freud, and Hampshire on Psychic Freedom."

Hampshire's argument begins with calling attention to the reflective nature of states of mind as a good basis for denying the unrestricted and strict application of deterministic conceptions to mental events. He advances the hypothesis that the explanation of thought will always require links under the laws of thought, which are unlike physical laws in that the former are normative in part. The correlation between a physical input—a chemical in the brain, for instance—and the observable outcome "is of a different kind from the causal connections between behavior and the beliefs, desires, and other propositional attitudes that are involved in man's thinking."

In the second essay, "Words in Thoughts," Zeno Vendler, a professor of philosophy, argues that the Platonic view that thinking is talking to oneself silently is implausible and false. He demonstrates his point of view with subtle analysis and use of syllogisms. For example: "To sum up: talking, saying things, are voluntary actions. Having certain common types of thoughts cannot be voluntary, consequently, thinking in general cannot be talking to oneself, silently or aloud. . . . Language is the means of expressing thoughts, not of having them."

The next essay, "Dreams, Memory, and the Origin of Thoughts," is a very provocative contribution by Stanley R. Palombo, an American psychoanalyst. It is based on information theory and offers an intriguing phylogenetic hypothesis that the "mechanism of dream construction may have been the first step in the evolution of reflective consciousness." Palombo considers conscious, reflective, secondary process thought to be "the apex of a vast pyramid of adaptive mental activity."

Albert Hofstadter, a prominent American philosopher, contributes an elaborate and interesting application of the concept of alienation in his paper, "Consciousness, Thought, and Enownment." Enownment is, in Hofstadter's terminology, the appropriation of "otherness." However,

he says, "Thinking is not the only mode of appropriating otherness. Practice is more fundamental."

The first section of the book concludes with "Analytic Philosophy, Phenomenology, and the Concept of Consciousness," by Harold A. Durfee, an American philosopher. This highly philosophical paper deals with the schism between analytic (classical) philosophy and phenomenology and its reflected analogous conflict in psychological theory. Durfee concludes that "diverse interpretations of the nature of consciousness lie at the heart of this controversy." This discussion is difficult to follow for those unacquainted with these philosophical currents and the specialized vocabulary.

The second part of the book consists of four essays covering a diversity of subjects related to the central theme. John S. Kafka's paper, "Object Constancy, Ambiguity, Paradox, and Time," focuses on ambiguity and temporality. Kafka uses Russell's theory of types in an attempt to understand ambiguity and levels of psychoanalytic explanation. W.W. Meissner's essay delves into the theory of the process of paranoia and analyzes the importance to psychiatry of the distinction between fantasy and reality. In Wolfgang Loch's essay on the concept of truth in analytic theory, scientific and existential truths are distinguished and the consideration of the problem of meaning by Gottlob Frege is introduced.

The profundity of much of the material covered in these essays is truly challenging and intriguing. The collection is hardly for late-night reading, but it is a truly rewarding intellectual experience. I cannot refrain from quoting part of the last paragraph of the introduction:

> While the mind is the ever-present focus of attention of both fields [philosophy and psychiatry], each also tends to approach the real. These essays constitute an interdisciplinary consideration of these mutual concerns and of the theoretical issues implicit therein. They also exemplify that when psychiatry lives on the borderline of the humanistic disciplines . . . it lives uniquely and authentically on its own most significant frontiers.

I recommend this book wholeheartedly to the serious student of these matters.

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SIMON H. NAGLER, M.D.
New York, N.Y.

Working with Patients: Introductory Guidelines for Psychotherapists, *by Helen A. De Rosis, M.D. New York, N.Y., Agathon Press, 1977, 190 pp., $10.50.*

Doctor De Rosis has presented the novice with a straightforward, direct, no-nonsense approach to psychotherapy. She assumes that the beginner will be dealing with hospitalized patients at a stage of illness where interpretive, reconstructive psychotherapy is not likely to be the treatment of choice. Her emphasis is on the healthy aspects of the total person rather than on pathology, and on the need to learn from the patient rather than to impose a theory of personality development on him or her. The book is a worthwhile primer, although it is likely that those who might benefit the most from reading it will dismiss it as too superficial.

ROBERT J. CAMPBELL, M.D.
New York, N.Y.

The Heart Patient Recovers: Social and Psychological Factors, *by Sydney H. Crogg, Ph.D., and Sol Levine, Ph.D. New York, N.Y., Human Sciences Press, 1977, 417 pp., $14.95.*

This volume should be of general interest to *Journal* readers and certainly of specific interest to all primary care physicians, members of health care teams, health planners, and lay persons who are required to cope with the stresses of heart disease. Indeed, the potential readership encompasses a remarkedly large number of persons. Croog and Levine systematically and empirically studied several hundred previously healthy men during the one-year period following their hospitalization for a first myocardial infarction. Their concern was to observe and record this major life event as a stressor whose sequelae are influenced by many social, psychological, and physical variables. To my knowledge, this is the first such endeavor; therefore, the volume brings a fresh and extensive new data base.

Special chapters are given over to discussions of morbidity, mortality, and rehospitalization; employment and work experience; financial influences; institutions; physician advice and compliance; the patient's family; personality and psychological factors; and a review of variables relevant to the status of the patients at the conclusion of one year of study. The dramatic advances made in the hospital treatment of the acute myocardial infarction, including continuous monitoring in coronary care units by highly skilled health care teams, represent a well-known landmark in medical history. Croog and Levine provide a timely and scholarly work that uncovers and reveals the essentials of a quality recovery period through a comprehensive rehabilitative process. Such an idea would seem humanistically obvious; however, the authors are adept at pointing out both the complexity of the comprehensive recovery process and the deficiencies in future health care planning for addressing this issue.

This is a good book. It is unique because, as a very scholarly work, it should directly influence the behaviors and serve as a reference for a broad spectrum of persons in our society, including professionals, politicians, and, most importantly, the heart patient and his or her family. Behavioral scientists who are able to produce high-quality work of social relevance and pragmatism within the appropriate restraints of scientific methodology are to be congratulated.

WILLIAM E. THORNTON, M.D.
Charleston, S.C.

And a Time to Live: Toward Emotional Well-Being During the Crisis of Cancer, *by Robert Chernin Cantor. New York, N.Y., Harper & Row, 1978, 276 pp., $9.95.*

Dr. Cantor has written what amounts to a significant book on personality development. Admittedly, its core is the crisis

of cancer. Cancer is a stress; the disease is anxiety provoking and demands coping techniques. Similarly, even if one does not have cancer, one is not spared other sources of anxiety as one goes through life, and thus defense mechanisms and behavior patterns develop experientially. How one deals with living will determine how one will deal with dying.

It is within this framework that Dr. Cantor has provided us with a useful textbook. To his credit, it is without the usual psychological jargon. The format consists of four parts. Part one, The Cancer Experience, explores all the emotional parameters of the patient's experience: the impotent anger, the frightening fantasies, the guilt, the depression, the very crucial fear of loss of control over self and/or therapy. This section overlaps into part two, The Sphere of Personal Influence. Part Three, The Sphere of Family Influence, presents interesting psychological paradigms of marital and familial patterns of relationships. Part Four, The Search for Meaning, offers religious experiences and personal experiences extrapolated philosophically to substantiate faith and constructive justification for the adverse encounter.

It was in part two that I found myself notably involved with the author, and on two counts. On the first point, with concurrence: the right to die. In the depressed (noncancer) patient, suicide is a symptom. In the case of the patient who has suffered long from a life-threatening illness, the final judgment to discontinue further therapy is the patient's and not the professional's or society's.

The other segment in part two evoked uncomfortable feelings. In the section titled "Actively Influencing the Disease Cancer" the author in essence proposes that mentally and emotionally, "fighting back" can inhibit actual tumor growth or physical incapacitation. He cites the usual anecdotal support plus the capacity of biofeedback to affect bodily physiological functions and the visualization techniques developed by the Simontons. The latter approach encourages the patient to relax and to visualize his cancer, the anti-cancer treatment, and his body's immune responses. Generally, the visualization is in graphic imagery.

There is some question as to the validity of anecdotal confirmation (although someone has hinted that anecdotes are scientific facts that have not as yet been published). Furthermore, transient artificial and actual remissions do occur. There is no dispute with the author here; my issue with him is concern for the lay reader. The nonprofessional (and the professional, too, at times) can interpret this material as evidence of personal culpabitity for deterioration of the illness, that he has failed to develop a positive attitude, or that he is a bad patient. The author is not oblivious to this possibility. However, his caveat can be easily overlooked because it consists of one sentence enveloped in the last paragraph of the section: "Continued tumor growth implies no moral or emotional failure" (p. 135).

Despite the mild objection just stated, this book is a useful one. It has a readable style, almost fictional in its many clinical and poignantly dramatic examples. Its lack of sophistication is an asset, and I recommend it for cancer patients and their families. Workers in the area of family and couple therapy might find part three, The Sphere of Family Influence, interesting and educationally worthwhile.

Dr. Cantor's book is in effect a psychiatric text that uses cancer as a vehicle for stress from which to form psychological concepts. Therefore, it is commended to medical, nursing, ministerial, and other associated health care students. They can avail themselves of the opportunity to understand "human nature" and, as a possible bonus, to learn something about the emotional ramifications of the disease of cancer.

NATHAN SCHNAPER, M.D.
Baltimore, Md.

Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth, *by Richard Jessor and Shirley L. Jessor. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1977, 267 pp., $16.00.*

This research monograph concerns the complex psychosocial variables that influence the adolescent in making the transitions from child to adolescent and from adolescent to young adult. Adolescence is a long period of time that is neither psychologically nor socially homogeneous. Therefore, longitudinal, multifactorial studies like the one reported here are important. This type of research is hard to conduct and difficult to evaluate, as is this study. However, the important trends reported here make this an important research contribution.

The research design involved 400 high school and 200 college youth, male and female, evaluated yearly for four consecutive years by means of a 50-page personal questionnaire. Using parallel samples from the seventh grade through college, the authors provide a picture of the transitions from grade school to high school, high school to college, and college to the adult world.

The authors' social-psychological theory focuses on the role of three component structures of personality: a motivational-instigation structure, a personal belief structure, and a personal control structure. The authors devised a series of measures for each. They then correlated the assessed construct scores with three transition behaviors of adolescents: beginning to drink, starting to use marijuana, and becoming a nonvirgin.

The correlations between scores on the three personality constructs and the three "problem behaviors" are modest but statistically significant. The overall trends of the summary findings have more face validity than the isolated correlations. In brief, problem behaviors tended not to be isolated events but were associated with each other as a syndrome of multiple problematic behaviors. In general, traditional and conventional backgrounds, associates, and attitudes best predicted absence of problematic behavior in adolescence. Adolescents who expressed great concern for personal autonomy, a lack of interest in the conventional goals of church and school, and a jaundiced view of the larger society were most apt to engage in problematic behavior.

These findings may serve as an antidote for those who propose that a laissez-faire approach to self-actualization is the vortex of adolescent development. On the other hand, the authors' personality constructs are very broad and imprecise but their problem behaviors are very specific, so only the most modest of generalizations can be made. More precise personality constructs might have been achieved through the use of more apt measures for each construct. For example, the religious measure uses a very simple liberal-conservative continuum to assess the importance of church membership. The liberal-conservative concept has a low correlation with behavior, as does chruch membership per se. Here the authors could have sharpened their approach through several sophisticated psychometric scales of religiousness that have high correlations with behavior. They ignored such sophisticated measures and instead used their own idiosyncratic

scale. Similar limitations are found in other scaling measures.

Despite my caveats about design, concepts, and assumptions in this study, it stands as an exceptionally well-executed and carefully analyzed piece of research. As a longitudinal study of adolescence it is highly valuable. The working clinician may well find the intricate detail tedious, but the serious scholar will have to take this work into account.

E. MANSELL PATTISON, M.D.
Irvine, Calif.

Observing Behavior, Vol. 1: Theory and Applications in Mental Retardation, *edited by Gene P. Sackett, Ph.D. Baltimore, Md., University Park Press, 1978, 409 pp., $24.50.*

Observing Behavior, Vol. 2: Data Collection and Analysis Methods, *edited by Gene P. Sackett, Ph.D. Baltimore, Md., University Park Press, 1978, 108 pp., $12.50.*

These two volumes comprise the proceedings of a conference on observational/ethological methods in the study of mental retardation held at Lake Wilderness, Wash., in June 1976. The week-long conference involved the intensive interaction of the 41 contributors to these volumes, most of whom are psychologists and specialists in mental retardation. The work has been edited more successfully than most conference reports into clearly organized volumes. However, it remains a publication of congress proceedings with varied material and different degrees of sophistication about other work done in psychology, anthropology, sociology, child psychiatry, and sociobiology relating to observations of the mentally retarded.

The authors accept the general premise that observational methods can be quantified, and that observations can be examined ethologically and ecologically for the purpose of improving the care of the mentally retarded. They begin with the premise that ecology can be acceptably defined as the "complete study of behavior as it occurs in the natural habitat." Some presentations are adduced to prove that this perspective is novel and helpful in approaching retardation. The authors report on parent-infant interaction, cognitive development, language development, group process, and community process.

A particularly intriguing section by Edgerton and Langness describes retarded individuals in a community setting, and there are intriguing clusters of information about studies in language development. However, the overall problem with the first volume is that much of the work represents the beginning development of the view the authors term "ethologic." They provide no convincing synthesis that ethology goes beyond good conventional approaches to language, cognitive development, or mother-child interaction. Trendy phrases, such as "social ethology, ethnology, and ethological psychology," are evident, but the investigations of naturalistic observation versus experimental approaches appear to restate the long-standing, long-unreconciled problems faced by the behavioral sciences.

Volume 2 is more sharply focused on problems in the scientific classification of behavioral observation, its analysis, and its measurement and relates quite specifically to people working on these problems. There are some intriguing data for psychiatrists in the sections on cognitive and language development, but these two volumes are primarily resource documents for investigators in this perspective on human behavior.

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Chicago, Ill.

Social Development in Childhood: Day-Care Programs and Research, *edited by Roger A. Webb. Baltimore, Md., Johns Hopkins University Press, 1977, 192 pp., $12.50; $3.45 (paper).*

As the title suggests, this book is primarily concerned with the social development of children in day-care centers. In his introduction the editor indicates that the six chapters in the book report different research projects on infants and latency-age children. The papers were presented at a Hyman Blumberg symposium on research in early childhood education. Earlier volumes in the Blumberg symposia series dealt generally with the intellectual development of young children. The present research deals with problems in social development, particularly as it is observed in various group care situations. The nine contributors are psychologists who have been involved in research on early childhood development in various sections of the United States.

In chapter one, "Mother-Child Interaction in Day-Care and Home-Reared Children," Mary Blehar reports on her own and other research dealing with the effects of daily substitute care. She reports on what she calls the "strange situation: a method for assessing quality of attachment." This "strange situation" was originally developed in the course of a longitudinal study on the development of infant-mother attachment in the first years of life. In the study, children were observed in their reactions to their mother and then in their responses when a stranger was introduced into the room and the mother left the room. Similar patterns of attachment behavior and changes in these patterns were observed with children up to the age of four. This research work is very important because of the increasing number of women who are working outside the home and who are either placing their children in day-care centers or introducing strangers into the home to take care of their children. Comparisons have also been made between children who remain at home with their own mothers and those who have nonparent figures in the home.

In chapter two Mary Main makes an analysis of a peculiar form of reunion behavior seen in some day-care children. It has been observed that some one-year-olds avoid their mothers after a brief separation in a strange environment. In the "strange situation," the child avoids his or her mother after a brief separation. This avoidance has been shown to be related to angry behavior, to a set of odd behaviors, and to an unwillingness to interact with persons other than the mother. Main quotes Blehar in this volume to indicate that avoidance of the mother in the "strange situation" appears more strongly in day-care children than in home-reared children.

In chapter three, Ramey and Mills discuss the social and intellectual consequences of day care in high-risk infants. The infants in this particular research program ranged in age from three and a half months to nine and a half months. This research indicated that children who were enrolled in a special project scored significantly higher on the Bayley Scales of Infant Development than matched control individuals reared at home. The authors also report that high-risk infants in day-care centers had a social relationship with their

mother similar to that of general population infants. The authors emphasize that day-care programs vary and that additional studies are needed to determine whether or not other day-care programs encourage the infant in his or her social relationship with the mother.

In chapter four Webb, Oliveri, and Harnick write about the socialization of intelligence. They reviewed three studies and indicate that two different approaches to the problems of social influence on intellectual style have been attempted. The research was done in a series of nursery schools in Baltimore City. Children were assigned on a random basis to experimental and control groups. Teachers in the control schools received no special instructions. The experimental teachers were provided with a series of nine lesson plans that were distributed and discussed biweekly at teachers' meetings. The lesson plan had two levels of content. One level was specific to individual lessons, and the second was a study of the teacher-child interaction. The authors report that six months of intensive intervening produced highly significant differences according to the Bayley scales.

Greif, in chapter five, reports on peer interactions in preschool children and emphasizes that preschool children can learn from their own peers as well as from older children and adults. Children exchange information about the world and themselves.

In chapter six Elardo reports on "Project Aware." This program deals with latency-age children. In group meetings the leader tells the children a story and then the children participate in discussions about it. In addition, special guidance is given to teachers.

Some portions of this book are repetitive, particularly those dealing with the "strange situation." The emphasis in all of these chapters is on social rather than intellectual progress in children who were seen either in nurseries or day-care centers. The work is of particular interest to psychologists and child psychiatrists who are concerned about the early development of the child and who are now especially concerned because of the increasing number of mothers who go to work. I recommend this small volume to all those involved in the care and education of infants and small children.

FRANK J. CURRAN, M.D.
New York, N.Y.

The Dynamics of University Protest, *by Donald Light, Jr., and John Spiegel, with C. J. Lammers, R. S. Laufer, M. Levin, and J. L. Norr. Chicago, Ill., Nelson-Hall, 1977, 198 pp., $12.00.*

This book examines such topics as the origin and future of university protest and the literature on student unrest. It also examines the context in which protest occurs: the tactics of students, the responses of faculties, the psychology of the disorders themselves, and the tactics and strategies adopted by administrators to counter student opposition.

The Dynamics of University Protest makes a significant contribution to the literature by bringing together analyses of what were commonalities of response by different groups on campuses across the United States. Most previous books on the subject have described in great detail what occurred at such campuses as Berkeley, Harvard, Columbia, Kent State, and Jackson State.

Several chapters merit special consideration. Chapter two, "Point and Counterpoint in the Literature on Student Unrest" by Molly Levin and John Spiegel, provides a thoughtful synthesis of three stages the research literature went through in attempting to increase understanding of student unrest phenomena. The first stage was the analysis of the personalities and backgrounds of dissenters (pathology-in-students versus pathology-in-society); the second was a search for causes and issues (what students were really protesting about); the third was developing methods of coping with legitimate dissent (issues of process and response).

Chapter seven, "Tactics and Strategies Adopted by University Authorities to Counter Student Opposition," is written by a sociologist, C.J. Lammers. He identifies four basic tactics: "fight off," in which there is a maintenance of the difference in power and student leaders are perceived as subversive ring-leaders; "bug off," in which authorities give up a portion of their power and student leaders are spokesmen; "join in," in which some power is shared and the student leadership are innovators; and "stand off," in which power differences remain and student leaders are perceived as ring-leaders and idealists.

What this book does it does well. The analyses of tactics and strategies could well reflect the curriculum of a college of war. However, the topic, dynamics of protest, might have treated two topics in greater detail. First, what ought to be the interrelationship between the different segments on issues of conflict resolution? In other words, given the central university purposes of research, teaching, and public service, what should the structure of governance be to minimize disruptive distraction from pressing issues while at the same time providing for the constructive resolution of strongly felt differences of opinion? Second, the book could have considered the effects of changes of governance structure within universities since the 1960s that have proved to be viable, particularly when juxtaposed to changes that have not been successful. Both society and the university have changed in important ways in the past decade. These changes fundamentally affect the dynamics of protest, as the debate over investments in South Africa indicates, but they do not receive sufficient coverage.

JOHN M. WHITTLEY, ED. D.
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Ethology: The Biological Study of Animal Behavior, *by Rémy Chauvin; translated by Joyce Diamanti. New York, N.Y., International Universities Press, 1977, 241 pp., $12.50.*

This book condenses a considerable amount of material into 241 pages. Separate books could be written and, in some cases, have been written on many of the individual topics covered. A sampling of areas included in this book follows: 1) building behavior of caddis fly larvae, bees, ants, wasps, and birds; 2) animal learning studies; 3) factors affecting migration of animals; 4) sexual attraction and mating behavior in animals and man; 5) the development of affectional systems; 6) imprinting; 7) social organization of chimpanzees, macaques, gibbons, and howler monkeys; 8) animal communication; and 9) definition of major ethological concepts.

As is necessarily the case with books covering so many topics, *Ethology* could be critiqued as being too superficial. However, the author does an excellent job of telescoping key concepts and issues into a relatively short and readable book. Psychiatrists will find a great deal of helpful information concerning ethology. One of the particularly good fea-

tures of the book is its discussion of both field study data and laboratory data in relation to each other. This is rarely done because ethologists and comparative psychologists often unnecessarily denigrate each other's work.

In contrast to many ethology books, this one does not dwell on the detailed analysis of elementary movements and displacements. This type of analysis of behavior into its submicroscopic component parts has been necessary—perhaps even helpful. However, there is an inherent reductionistic error when such dissection techniques are applied to human behavior. The human organism is obviously more than the sum of its component parts. The author's analogy is to a computer in which the innate program is the expression of the nervous system and anatomy but is alterable by instructions to change programs. There is an interaction between the program and the environment, and this is what should be measured and studied. This model is applied to the solitary animal, the family, and social relations.

In view of the increasing importance of ethology in psychiatry, psychiatrists will find chapter nine a particularly helpful reference because it contains a glossary of key ethological concepts. Although some may find it overly terse and overly simplified, I found it helpful.

This is a book to be scanned, not an in-depth explanation of one ethological topic area. Given this framework, it is a useful book that contains literature references for more extensive reading.

WILLIAM T. MCKINNEY, M.D.
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Modern Psychoanalytic Concepts in a General Psychology. Part One: General Concepts and Principles; Part Two: Motivation, Psychological Issues Monograph 42/43. *by Allan D. Rosenblatt and James T. Thickstun. New York, N.Y., International Universities Press, 1978, 338 pp., $15.00 (paper).*

The authors of this text of current psycholanalytic theory as it relates to a general psychology of human behavior are to be congratulated. They have produced a very satisfying survey of existing psychoanalytic theory. The volume is succinct, well written, effectively organized, and thoroughly documented. The bibliography is excellent, and the index is very easy to use.

Drs. Rosenblatt and Thickstun do not believe that psychoanalysis is or has the potential for becoming a general psychology in itself. Psychoanalysis as such cannot encompass all human behavior or provide a complete explanation of such fields as learning, memory, or perception. However, psychoanalysis makes its unique contribution to a general theory of human behavior, and this monograph outlines what the authors consider to be the most relevant contributions of psychoanalysis.

Part One is devoted to general concepts and principles. General problems of methodology, systematization, scientific explanation, theories, models, validation, and prediction are discussed clearly and yet not in such detail as to distract from the major emphasis of this work. Brief discussions of modern psychoanalytic core propositions, information processing systems in psychoanalytic theory, and such key concepts as psychic determinism, unconscious mental processes, conflict, anxiety, and the metapsychological points of view complete this first section.

In the second part the focus is on motivation. Such issues as instinct and drive, incentive, and cognitive theories are discussed with an eye toward the presentation of the authors' model of the motivational system. In their last chapter they use clinical data to illustrate their formulations.

I found this work very useful, stimulating, and need-fulfilling. We now have an up-to-date work that can be used by students, researchers, teachers, and clinicians. The two parts of this volume are the first of a projected eight-part work. I look forward to the publication of the remaining six sections. If they are as well done as the first two, the authors have done us a great service.

GEORGE H. POLLOCK, M.D., PH.D.
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Behavioral Approaches to Weight Control, *by Edward E. Abramson. New York, N.Y., Springer Publishing Co., 1978, 175 pp., $12.95.*

This short book consists of nine chapters and nine appendixes; three of the chapters are written by the editor. In the preface Abramson states, "This book will provide the necessary information and materials so that the professional without extensive training in behavior modification will be able to choose and implement a behavioral treatment program" (p. x).

Although I would agree that the editor has generally succeeded in attaining his stated objective, the quality of the different contributions varies. Perhaps the most serious problem is that, with the exception of Abramson's three chapters, the material has previously been published in other sources in 1971–1975. For example, Cautela's description of treatment by means of covert conditioning (initially published in 1972), according to Abramson's own analysis of the research data in chapter two, certainly does not appear to be the behavioral treatment of choice. Similarly, some of the other strategies described in the book (e.g., Fowler's "mouthful diet") have not enjoyed substantial empirical support.

In chapter one Abramson presents a very readable and sufficiently comprehensive overview of the etiology of weight gain and some of the traditional remediation methods that have been employed. However, the statement on page 6 about the impossibility of using humans for experimental studies of genetics is too obvious to be included, even for the lay reader. In chapter two, Abramson gives the reader a brief but inclusive view of the behavioral approach to treatment. Christensen, Jeffrey, and Pappas discuss an operant approach to treatment in chapter three, and Wollersheim outlines a number of the treatment strategies she has used with overweight female college students in chapter four. Although the techniques depicted make good theoretical sense, I question the ultimate wisdom of substituting smoking for eating food during the difficult periods in treatment when cravings are likely to occur. The substitution of one potentially addictive behavior (probably more dangerous to overall health than being overweight) for another has no justification. In chapter five Fowler describes his "mouthful diet" (i.e., decreasing the average number of bites of food per day during treatment), and in chapter six Cautela discusses covert conditioning therapy. In chapter seven Lutzker and Lutzker clearly articulate an interesting contingency contracting approach to losing weight. The contract is to be negotiated between two people (e.g., husband and wife).

The importance of exercise in the initial weight loss effort and in maintaining weight loss is indicated in an extremely

short offering in chapter eight by Ginsburg and Baker. Finally, in chapter nine, Abramson discusses several critical clinical considerations (e.g., the interpersonal) in the behavioral treatment of overweight individuals that often would be totally neglected by the neophyte therapist. The appendixes, including questionnaires, a behavioral contract, a physician's permission form, and illustrative case studies, are quite good. More of these would have enhanced the overall value of the book.

In summary, for the therapist interested in obtaining a rather quick idea as to what behavior therapists tend to do with their overweight patients, the book achieves its goal quite admirably. Moreover, the price is reasonable considering how inflation has affected the publishing business. However, in addition to the fact that some of the material is dated, I think the question of how successful patients are in maintaining their initial weight losses could have received more careful scrutiny. Then again, the book does reflect the current status of the art as it is being practiced. Despite deficiencies in our behavioral techniques, they represent the most successful of the psychotherapeutic approaches to the problem of overweight at this time.

MICHEL HERSEN, PH.D.
Pittsburgh, Pa.

Fragile Families, Troubled Children: The Aftermath of Infant Trauma, *by Elizabeth Elmer; Sue Evans and John B. Reinhart, co-investigators. Pittsburgh, Pa., University of Pittsburgh Press, 1977, 156 pp., $7.95.*

This book seeks to identify and compare 17 "abused" and 17 "accident" children at three different times: 1) when the infant was first brought to the attention of the study group, 2) one year later, and 3) eight years later. Various psychological and psychiatric examinations and thorough social work material were accumulated. Not the easiest task was the identification of abused children versus accident children. The latter were those seen with injuries or fractures that were presumably accidental, while the former were those whose parent or parents sporadically abused the child physically.

The initial chapter describes the study itself. Next is a chapter on the follow-up study and then one comprised of three case studies, each given in some detail. The final chapter presents the author's conclusions. In this chapter Elmer focuses on poverty: "By placing the results of our follow-up study in the context of the massive literature concerning poverty, we come to the conclusion that the results of child abuse are less potent for the child's development than class membership."

Actually, the author substantiates many well-known ideas about child abuse but is unable to confirm others. The two groups appear to be similar in many ways. Further studies and follow-up are planned, and this seems essential because the study reports on only a small sample of youngsters and their families. It shows some of the problems in choosing abused children and tries to deal with children so labeled who remain with their parents and those placed elsewhere.

This book was interesting to read, but I am left with the impression that much more needs to be done before we understand this complex problem. The study was begun before laws were enacted concerning child abuse and carried on after various laws were passed. As the author points out, for centuries children were dealt with as parents saw fit; it is only in fairly recent times that adults have begun to consider the rights of the child.

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Psychosocial Treatment of Chronic Mental Patients: Milieu Versus Social-learning Programs, *by Gordon L. Paul and Robert J. Lentz. Cambridge, Mass., Harvard University Press, 1977, 514 pp., $22.50.*

This book is a report and an analysis in depth of a clinical research study designed to assess the relative effectiveness of traditional hospital management, milieu therapy, and a social learning program. The patient groups were carefully matched for age, sex, chronicity, length of hospitalization, and primary psychiatric diagnosis (all were diagnosed as schizophrenic). The severity of their functional impairment was such that all had been rejected for community placement in a previous screening.

Twenty-eight patients were assigned to each of three groups. The traditional treatment group was assessed periodically by the investigators, who tried to avoid having any influence on their management. This group essentially served as the controls in the study. The other two patient groups were placed in physically adjacent units at another location. A specially trained team of "change agents" worked with both groups in an alternating shift arrangement designed to make the treatment program itself the only significant variable.

The study (and the treatment programs) extended over a period of almost four and a half years. New patients (residents) were added as community placement, death, or other factors led to reduction of sample size. In all, 40 individuals were treated in the social learning program and 31 each in the milieu and hospital programs.

The authors do not consider the subjects of this study to be ill but, rather, to have learned inappropriate behaviors or to have failed to learn or to have forgotten behavior needed to function in a normal world. The two psychosocial rehabilitation programs are meticulously defined and described. Major objectives common to both are resocialization, instrumental role performance, reduction or elimination of extreme bizarre behaviors, and support in the community after release. The social learning program used a token economy and attempted to reduce undesirable behavior by ignoring it. The milieu program used both positive and negative feedback, with prompt and unambiguous response to both desirable and undesirable behaviors. Manuals for both programs are included in their entirety in this volume.

Criteria for release to the community until the last six months of the study required the capacity for independent functioning and self-support. In the final period community placement allowing board and care living arrangements was permitted. Under these modified conditions "significant release" required a continuous stay in the community of at least 90 days.

So many factors were assessed in this study that a summary of results in this review must be selective and incomplete. Release to independent functioning and self-support was achieved by 3 patients (residents) in the original social learning group, by 2 in the milieu group, and by one in the hospital group. Release to private extended care board and room facilities was achieved by all but 1 of the original social learning group, by two-thirds of the milieu group, and

by just under half of the hospital group. A cost effectiveness study showed a near-equal cost per patient per day in all three programs (a little more than $10) and a total cost over the four and a half years of about $15,000 for the social learning and milieu programs versus about $17,000 for the hospital program. Cost of psychotropic drugs over the four and a half years was about $1,072 for the hospital group versus $261 for the milieu and $147 for the social learning group. The authors conclude that psychotropic drugs were of little value in the psychosocial programs and may have had a negative effect in some instances.

This volume is much more than an account of treatment outcome comparing medical versus nonmedical approaches to schizophrenia. Other themes include an account of the problems growing out of funding delays and a description of difficulties caused by externally imposed (and seemingly arbitrary) changes in policy and practice. The imposition of a rule limiting seclusion for assaultive behavior to two hours (midway in the project) apparently led to an almost total loss of gains in the psychosocial treatment groups. Only by developing alternative "negative reinforcers" could these losses be recouped. The alternatives to prolonged seclusion or social isolation included heating and humidfying the seclusion room and introduction of a randomly activated klaxon horn. I found this maneuver disquieting, to say the least, particularly since the authors found some patients were committing minor "assaultive" acts for the "privilege" of having two hours of solitude.

I did not think that the results of the study prove that schizophrenia is a learned maladaptative lifestyle—nor do the authors specifically make such a claim. This is an important book, rich with carefully documented observations. It deserves a careful reading by all mental health professionals. The experimental design is a model of excellence. The book itself is exceptionally well crafted, with no evident technical flaws and a splendid index.

WILLIAM A. CANTRELL, M.D.
Houston, Tex.

Medical Information Systems: A Resource for Hospitals, *by Melville H. Hodge. Germantown, Md., Aspen Systems Corp., 1977, 192 pp., $16.75.*

This book is written at a most opportune time. The health professions are under increasing pressure from consumers and government alike to be accountable for their actions. There is growing impatience with the ever-increasing costs of medical care and demands that something be done to contain these costs.

In a very readable, well-outlined format, Mr. Hodge shows how a computerized medical information system can save hospitals money and increase efficiency. Some examples include economic benefits, conservatively estimated as saving from $3 to $5 per patient day by reducing errors, improving timeliness, and enhancing the availability of medical records. A medical information system can also provide more accurate data on personnel workloads, patient activity, and use of facilities. Mr. Hodge says that patient care improves because of the prevention of diagnostic and treatment errors. Finally, a medical information system, by collecting pertinent information, can be used in programs of research and education.

The book is divided into sections, beginning with a list of the theoretical and actual benefits in the areas of economy, patient care, research, and education. The next seciton deals with critical decisions that have to be made in setting up a medical information system—decisions that include what type of system is most suitable for a particular hospital and the measures that are necessary for the successful implementation of a system. Mistakes and errors in this area can be disastrous for the system and result in great financial losses for the hospital. The next section deals with the impact of a functioning system on the various hospital staff members and departments. Mr. Hodge concludes his book with a chapter on how a hospital can take steps to realize the benefits of a medical information system.

This book concerns medical information systems for general hospitals. Very little pertains directly to the functioning of psychiatrists in the system. However, in the hospitals with operational systems studied, psychiatrists were one of the groups of specialists that were in favor of the system. This was true in spite of the fact that they were the group who used the system least.

An issue not addressed by the author is that of confidentiality—what safeguards can be taken to protect patients from unauthorized persons obtaining information. If several hospitals became involved in a common medical information system the quesiton would become even more critical. Recent revelations in the press regarding the ease with which information can be obtained from systems specifically designed to be "burglar proof" are not reassuring. One hopes that the computer industry will give serious thought to how this problem can be resolved.

In conclusion, this book would be valuable to any organization contemplating installation of a medical information system. By his listing of the do's and don't's in such a process, Mr. Hodge shows how to avoid many of the potentially dangerous pitfalls.

GEORGE W. BARTHOLOW, M.D.
Omaha, Neb.

Therapeutic Partnership: Ethical Concerns in Psychotherapy, *by Carl Goldberg. New York, N.Y., Springer Publishing Co., 1977, 264 pp., $17.95.*

Dr. Goldberg's book represents a necessary inquiry that should be made by all persons involved in psychotherapeutic endeavors—a review of the basic assumptions on which psychotherapy is predicated and a pursuit of the implications of these assumptions in one's psychotherapy practice, education, training, and treatment. For the author, this assessment pivots on two presumably interconnecting themes: 1) the more circumscribed and concrete "client-therapist collaborative endeavors in establishing a therapeutic alliance" and 2) the larger and loftier "quest for meaning in human existence." The author believes that in the best of all psychotherapeutic worlds these two would be intimately related. The model of the book is existentially oriented and interpersonal and upholds the necessity of the presence of another person in realizing the autonomy of the self (i.e., the therapeutic centrality of relationship). It is a particular kind of relationship that Dr. Goldberg propounds, i.e., not the formation of the traditional therapeutic alliance but a "therapeutic partnership," not the more familiar working alliance but a "working contract." In essence, he posits an egalitarian and mutual partnership based on maximal informed consent of both parties (which may or may not be affirmed by a written contract), in which it is the client, not the therapist,

who in the final analysis defines health and illness. The book's subheading, *Ethical Concerns in Psychotherapy* refers not to the violation of existing moral or legal codes, which may be relatively easy to identify and deal with, but the more covert, subtle, and insidious departures from a hypothetical existential contract between client and therapist.

The philosophical stance of the book is highly relevant—an apt expression of the growing consciousness of human rights and responsibilities, evident in the current interest in patients' rights and power relations. Despite this theoretical vantage point, the author actively addresses the reputed failure of existentially oriented psychotherapists to depict the therapeutic encounter in other other than poetic or metaphysically vague terms. This is well countered by the author's careful attention to specific and practical aspects of the therapist-client relationship in its immediate applications. The vicissitudes of encounter issues in practice, to which a major portion of the book is devoted, are well developed in separate chapters of considerable breadth, each reflecting a different modality or therapeutic situation, e.g., marital therapy, cotherapy, group therapy, encounter groups, the therapeutic community, community mental health programs, and consultation. In addition, a rather unprecedented appendix provides (for therapist as well as client) "A Client Guide to Selecting a Therapist and Formulating a Therapeutic Contract."

Dr. Goldberg aspires to close the reputed gap between therapist and patient through mutual enlightenment, but it is clear throughout the book which side he takes. By obliterating the distance between the two therapeutic parties Dr. Goldberg means to restore power to the client from the presumably dominating, abusive hands of the traditional psychotherapist, whose subversive purposes he hopes to "demystify" and "demedicalize." Stirred by this strong client advocacy position, Dr. Goldberg relentlessly opts for "power adequacy"—rational (i.e., equitable) use of power in the therapeutic enterprise.

Although this is an undisputedly commendable goal as such, it is sometimes confused in its articulation with a rejection of all psychiatry, medicine, or psychotherapy as previously practiced. The fact that Dr. Goldberg chose Thomas Szasz to write his preface implies that he is the person to whom Dr. Goldberg's major allegiance lies. This may be an unduly limiting alliance because we are thus given to believe that the antipsychiatry postulates that mental illness and mental treatment are myths are prerequsites to our thinking, in that they provide the philosophical backbone of Dr. Goldberg's treatise. This is unfortunate because the author already feels that he has lost a potential audience of "experienced practitioners" who may not be amenable to questioning the premises of their work. Actually, the book will probably be of more extensive value than Dr. Goldberg assumes if read by those with or without strict subscription to the Szaszian framework.

There are two other ways in which this stance may be misleading. First, it has not been definitively determined as yet what therapeutic roles are played in the therapeutic process by the curer's charisma, ambiguity or mystique, or the use of magic and faith. These elements have always been vital parts of the therapeutic endeavor. Second, Dr. Goldberg's statement that "the therapist must teach the client how to rationally use his power or how to regain his low power" (p. 97) may be a contradiction in terms that serves only to compound the problem, i.e., power is being put back into the hands of the very therapists who presumably possessed it in the first place.

Despite statements of this kind, which may be reflective only of the ardor with which Dr. Goldberg pursues his point, *Therapeutic Partnership* is a highly readable piece of work. It adroitly avoids the inherent danger in writing about ethical matters—that of appearing moralistic or above reproach. In general, the author keeps a good balance between well-referenced research studies and an ample supply of case examples and anecdotal material; these are useful and of interest to therapist and client alike. Sometimes, however, Dr. Goldberg seems to have trouble straddling the needs of both parties, vacillating between the theoretical and pragmatic, the scholarly and the mundane. Moreover, in his ideal attempt to address the client and therapist as equals, he often overcompensates the client by portraying the therapist in too negative a light (by recounting instances of the very order he says he intends to avoid, e.g., moral improprieties involving sex relations, overcharging of fees, and nudity). However, all of this may be an inevitable concomitant of a truly ambitious and arduous task, to accomplish in writing what he hopes to accomplish in the therapeutic relationship, a partnership of client and therapist in which unspoken and alienating differences are ultimately united by their common concerns.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Advances in Neurochemistry, Vol. 2, *edited by B.W. Agranoff and M.H. Aprison. New York, N.Y., Plenum Press, 1978, 341 pp., $29.50.*

Evaluations of Drug Interactions, 2nd ed., Supplement, *prepared by the American Pharmaceutical Association with the cooperation and assistance of the American Dental Association, the American Medical Association, the American Podiatry Association, the American Society of Hospital Pharmacists, the Food and Drug Administration, and the National Library of Medicine. Washington, D.C., American Pharmaceutical Association, 1978, 604 pp., $7.00 (paper).*

How Not to Split Up, *by Jane Appleton and William Appleton, M.D. Garden City, N.Y., Doubleday & Co., 1978, 198 pp., $7.95.*

Alcoholism and Treatment, *by David J. Armor, J. Michael Polich, and Harriet B. Stambul. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 343 pp., $16.95.*

Discovering Suicide: Studies in the Social Organization of Sudden Death, *by J. Maxwell Atkinson. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 220 pp., $14.95.*

How to Get Your Child to Listen, *by Thomas J. Banville, Ph.D. New York, N.Y., Condor Publishing Co., 1978, 213 pp., $2.50.*

Scientists Must Write: A Guide to Better Writing for Scientists, Engineers, and Students, *by Robert Barrass. London, England, Chapman and Hall (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1978, 167 pp., $8.95.*

Law and the Mental Health Professions: Friction at the Interface, *edited by Walter E. Barton, M.D., and Charlotte J. Sanborn, A.R.T. New York, N.Y., International Universities Press, 1978, 319 pp., $15.00.*

Videotape Techniques in Psychiatric Training and Treatment, revised ed., *edited by Milton M. Berger, M.D. New York, N.Y., Brunner/Mazel, 1978, 397 pp., $20.00.*

Hormones and Aggression, Vol. 1, 1977, *by Paul F. Brain. Montreal, Que., Canada, Eden Press, 1977, 123 pp., $14.00.*

Perspectives in Endocrine Psychobiology, *edited by F. Brambilla, P.K. Bridges, E. Endröczi, and G. Heuser. New York, N.Y., John Wiley & Sons, 1978, 590 pp., $39.95.*

Toward a Theory of Psychosomatic Disorders: Alexithymia, Pensée Operatoire, Psychosomatisches Phänomen, *edited by W. Bräutigam and M. von Rad. Basel, Switzerland, S. Karger, 1977, 388 pp., $53.50.*

Health Is a Question of Balance, *by Paul Brenner, M.D. New York, N.Y., Vantage Press, 1978, 143 pp., $5.95.*

Home Health Care for the Aged: How to Help Older People Stay in Their Own Homes and Out of Institutions, *by Philip W. Brickner, M.D. New York, N.Y., Appleton-Century-Crofts, 1978, 287 pp., $15.75.*

Mothers, Fathers, and Children: Explorations in the Formation of Character in the First Seven Years, *by Sylvia Brody and Sidney Axelrad, with Ethel Horn, Marsha Moroh, and Marvin Taylor. New York, N.Y., International Universities Press, 1978, 659 pp., $27.50.*

The Other Generation Gap: The Middle-Aged and Their Aging Parents, *by Stephen Z. Cohen and Bruce Michael Gans. Chicago, Ill., Follett Publishing Co., 1978, 283 pp., $10.95.*

A Festering Sweetness: Poems of American People, *edited by Robert Coles. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 51 pp., $6.95; $3.95 (paper).*

Child Abuser: A Study of Child Abusers in Self-Help Group Therapy, *by Marilyn C. Collins, Ph.D. Littleton, Mass., PSG Publishing Co., 1978, 124 pp., $12.50.*

The Endorphins. Advances in Biochemical Psychopharmacology, Vol. 18, *edited by Erminio Costa, M.D., and Marco Trabucchi, M.D. New York, N.Y., Raven Press, 1978, 366 pp., $28.00.*

Clinical Psychiatry in Primary Care, *by Steven L. Dubovsky, M.D., and Michael P. Weissberg, M.D. Baltimore, Md., Williams & Wilkins Co., 1978, 206 pp., $10.95 (paper).*

International Review of Research in Mental Retardation, Vol. 9, *edited by Norman R. Ellis. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 298 pp., $23.00.*

Crisis: A Handbook for Systemic Intervention, *by Jane Ferber, M.D., and John Schoonbeck, Ph.D. Gardiner, N.Y., Institute for Human Studies, 1978, 64 pp., $3.50 (paper).*

Stress Disorders Among Vietnam Veterans: Theory, Research and Treatment, *edited by Charles R. Figley, Ph.D. New York, N.Y., Brunner/Mazel, 1978, 311 pp., $15.00.*

Termination in Psychoanalysis, *by Stephen K. Firestein, M.D. New York, N.Y., International Universities Press, 1978, 262 pp., $15.00.*

Deviants and the Abandoned in French Society: Selections from the Annales Economies, Sociétés, Civilisations, Vol. IV, *edited by Robert Forster and Orest Ranum; translated by Elborg Forster and Patricia M. Ranum. Baltimore, Md., Johns Hopkins University Press, 1978, 245 pp., $14.00; $4.95 (paper).*

A Grammar of Dreams, *by David Foulkes. New York, N.Y., Basic Books, 1978, 441 pp., $20.00.*

The Year Book of Psychiatry and Applied Mental Health 1978, *edited by Daniel X. Freedman, M.D., Arnold J. Friedhoff, M.D., Lawrence C. Kolb, M.D., Reginald S. Lourie, M.D., Sc.D., John C. Nemiah, M.D., and John Romano, M.D. Chicago, Ill., Year Book Medical Publishers, 1978, 412 pp., $29.95.*

Medical Students, Medical Schools and Society During Five Eras: Factors Affecting the Career Choices of Physicians 1958–1976, *by Daniel H. Funkenstein, M.D. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1978, 221 pp., no price listed.*

The Information in Contingency Tables, *by D.V. Gokhale and Solomon Kullback. New York, N.Y., Marcel Dekker, 1978, 356 pp., $29.75.*

Explorations in Psychoanalysis, *by Ralph R. Greenson. New York, N.Y., International Universities Press, 1978, 560 pp., $22.50.*

The Chronic Mental Patient in the Community. Publication 102, *by the Group for the Advancement of Psychiatry Committee on Psychiatry and the Community. New York, N.Y., GAP, 1978, 106 pp., $3.50 (paper).*

Power and Authority in Adolescence: The Origins and Resolutions of Intergenerational Conflict. Publication 101, *by the Group for the Advancement of Psychiatry Committee on Adolescence. New York, N.Y., GAP, 1978, 213 pp., $6.50 (paper).*

Psychotherapy and Its Financial Feasibility Within the National Health Care System. Publication 100, *by the Group for the Advancement of Psychiatry Committee on Therapy. New York, N.Y., GAP, 1978, 45 pp., $2.50 (paper).*

Applied Atomic Spectroscopy, Vol. 1, *edited by E.L. Grove. New York, N.Y., Plenum Press, 1978, 300 pp., $39.50.*

Applied Atomic Spectroscopy, Vol. 2, *edited by E.L. Grove. New York, N.Y., Plenum Press, 1978, 319 pp., $39.50.*

Mind and Language. Wolfson College Lectures 1974, *edited by Samuel Guttenplan. New York, N.Y., Oxford University Press, 1978, 158 pp., $4.00 (paper).*

Psychopathic Behaviour: Approaches to Research, *edited by R.D. Hare and D. Schalling. New York, N.Y., John Wiley & Sons, 1978, 378 pp., $32.00.*

Broadcast Standards Editing, *by Melvin S. Heller, M.D. New York, N.Y., American Broadcasting Companies, 1978, 99 pp., $7.95 (spiral-bound).*

Ecologic-Biochemical Approaches to Treatment of Delinquents and Criminals, *edited by Leonard J. Hippchen, Ph.D. New York, N.Y., Van Nostrand Reinhold Co., 1978, 392 pp., $16.95.*

The Whipping Boy, *by Beth Holmes. New York, N.Y., Richard Marek, 1978, 458 pp., $10.00.*

Kindergarten Is Too Late! *by Masaru Ibuka. New York, N.Y., Simon & Schuster, 1978, 183 pp., $7.95.*

Studies in Geriatric Psychiatry, *edited by A.D. Isaacs and F. Post. New York, N.Y., John Wiley & Sons, 1978, 261 pp., $26.00.*

First Person Singular: Living the Good Life Alone, *by Dr. Stephen M. Johnson. New York, N.Y., Signet Book (New American Library), 1978, 170 pp., $2.25 (paper).*

Clinical Neuropharmacology, Vol. 3, *edited by Harold L. Klawans, M.D. New York, N.Y., Raven Press, 1978, 201 pp., $19.50.*

The New Image of the Person: The Theory and Practice of Clinical Philosophy. Contributions in Philosophy 9, *by Peter Koestenbaum. Westport, Conn., Greenwood Press, 1978, 558 pp., $19.50.*

The Psychology of the Self: A Casebook, *written with the collaboration of Heinz Kohut, M.D.; edited by Arnold Goldberg, M.D. New York, N.Y., International Universities Press, 1978, 456 pp., $20.00.*

Oneness and Separateness: From Infant to Individual, *by Louise J. Kaplan, Ph.D. New York, N.Y., Simon & Schuster, 1978, 280 pp., $9.95.*

Cognitive Development: Research Based on a Neo-Piagetian Approach, *edited by J.A. Keats, K.F. Collis, and G.S. Halford. New York, N.Y., John Wiley & Sons, 1978, 446 pp., no price listed.*

Mary Jane Harper Cried Last Night, *by Joanna Lee and T.S. Cook. New York, N.Y., Signet Book (New American Library), 1978, 152 pp., $1.50 (paper).*

Society, Stress, and Disease, Vol. 3: The Productive and Reproductive Age—Male/Female Roles and Relationships, *edited by Lennart Levi, M.D. New York, N.Y., Oxford University Press, 1978, 279 pp., $41.00.*

The Scientist: A Novel Autobiography, *by John C. Lilly, M.D. Philadelphia, Pa., J.B. Lippincott Co., 1978, 210 pp., $10.00.*

Dimensions of Personality, *edited by Harvey London and John E. Exner, Jr. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 599 pp., no price listed.*

Essays in Humanity and Technology. Papers from the Sauk Valley Conference, 1977, *edited by David Lovekin and Donald Phillip Verene. Dixon, Ill., Sauk Valley College, 1978, 189 pp., no price listed (paper).*

Development and Plasticity of the Brain: An Introduction, *by R.D. Lund. New York, N.Y., Oxford University Press, 1978, 361 pp., $14.95; $7.95 (paper).*

Contributions to the Psychopathology of Schizophrenia, *edited by Brendan A. Maher. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 379 pp., $9.50 (paper).*

Experiencing: A Humanistic Theory of Psychology and Psychiatry, *by Alvin R. Mahrer, Ph.D. New York, N.Y., Brunner/Mazel, 1978, 866 pp., $22.50.*

Handbook of Adolescence: Psychopathology, Antisocial Development, Psychotherapy, *by Carl P. Malmquist, M.D. New York, N.Y., Jason Aronson, 1978, 879 pp., $30.00.*

Community Psychology: Concepts and Applications, *by Philip A. Mann. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 331 pp., $15.95.*

Violence and the Family, *edited by J.P. Martin. New York, N.Y., John Wiley & Sons, 1978, 352 pp., $29.95.*

New Perspectives on Psychotherapy of the Borderline Adult, *edited by James F. Masterson, M.D. New York, N.Y., Brunner/Mazel, 1978, 162 pp., $10.00.*

Victims No More, *by Thomas R. McCabe, Ph.D. Center City, Minn., Hazelden, 1978, 103 pp., $3.95 (paper).*

The Confessions of William James Chidley, *edited by S. McInerney. St. Lucia, Queensland, Australia, University of Queensland Press, 1978, 305 pp., $14.50.*

Students Under Stress: A Study in the Social Psychology of Adaptation, *by David Mechanic. Madison, Wis., University of Wisconsin Press, 1978, 226 pp., $15.00; $5.95 (paper).*

Just This Side of Madness: Creativity and the Drive to Create, *by Carol Ann Morizot. Houston, Tex., Harold House, 1978, 166 pp., $12.95; $6.95 (paper).*

Phobia Free and Flying High, *by Neal Olshan and Julie Wang. New York, N.Y., Condor Publishing Co., 1978, 207 pp., $2.25 (paper).*

Medicine and the Reign of Technology, *by Stanley Joel Reiser. New York, N.Y., Cambridge University Press, 1978, 307 pp., $14.95.*

Sex, Society and the Disabled: A Developmental Inquiry Into Roles, Reactions, and Responsibilities, *by Isabel P. Robinault. Hagerstown, Md., Harper & Row Medical Department, 1978, 262 pp., $14.95.*

Psychoanalysis, Creativity, and Literature: A French-American Inquiry, *edited by Alan Roland. New York, N.Y., Columbia University Press, 1978, 355 pp., $20.00.*

The Experience of Depression, *by Dorothy Rowe. New York, N.Y., John Wiley & Sons, 1978, 273 pp., $19.50.*

Impedance Measurements in Biological Cells, *by Otto F. Schanne and Elena Ruiz P.-Ceretti. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 421 pp., $29.95.*

A Child's Journey: Forces That Shape the Lives of Our Young, *by Julius Segal, Ph.D., and Herbert Yahraes. New York, N.Y., McGraw-Hill Book Co., 1978, 336 pp., $10.95.*

Currents in Alcoholism, Vol. III: Biological, Biochemical, and Clinical Studies, *edited by Frank A. Seixas, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 589 pp., $31.50.*

Currents in Alcoholism, Vol. IV: Psychiatric, Psychological, Social, and Epidemiological Studies, *edited by Frank A. Seixas, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 487 pp., $26.50.*

Psychotherapy: A Personal Approach, *by D. J. Smail. London, England, J.M. Dent & Sons, 1978, 144 pp., $12.50.*

Self-Assessment of Current Knowledge in Forensic and Organic Psychiatry: 1,200 Multiple-Choice Questions and Referenced Answers, *by Selwyn M. Smith, M.D., and Erwin K. Koranyi, M.D. Garden City, N.Y., Medical Examination Publishing Co., 1978, 250 pp., $12.00 (spiral-bound).*

The Adoption Triangle: The Effects of the Sealed Record on Adoptees, Birth Parents, and Adoptive Parents, *by Arthur D. Sorosky, M.D., Annette Baran, M.S.W., and Reuben Pannor, M.S.W. Garden City, N.Y., Anchor Press/Doubleday, 1978, 256 pp., $8.95.*

Critical Issues in Psychiatric Diagnosis, *edited by Robert L. Spitzer, M.D., and Donald F. Klein, M.D. New York, N.Y., Raven Press, 1978, 340 pp., $25.00.*

A Behavioral Atlas of the Rat Brain, *by Robert Thompson. New York, N.Y., Oxford University Press, 1978, 78 pp., $14.95; $8.85 (paper).*

Living in Prison: The Ecology of Survival, *by Hans Toch. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 310 pp., $12.95.*

War on the Mind: The Military Uses and Abuses of Psychology, *by Peter Watson. New York, N.Y., Basic Books, 1978, 522 pp., $20.00.*

The History of Psychology and the Behavioral Sciences: A Bibliographic Guide, *by Robert I. Watson, Sr. New York, N.Y., Springer Publishing Co., 1978, 241 pp., $11.95.*

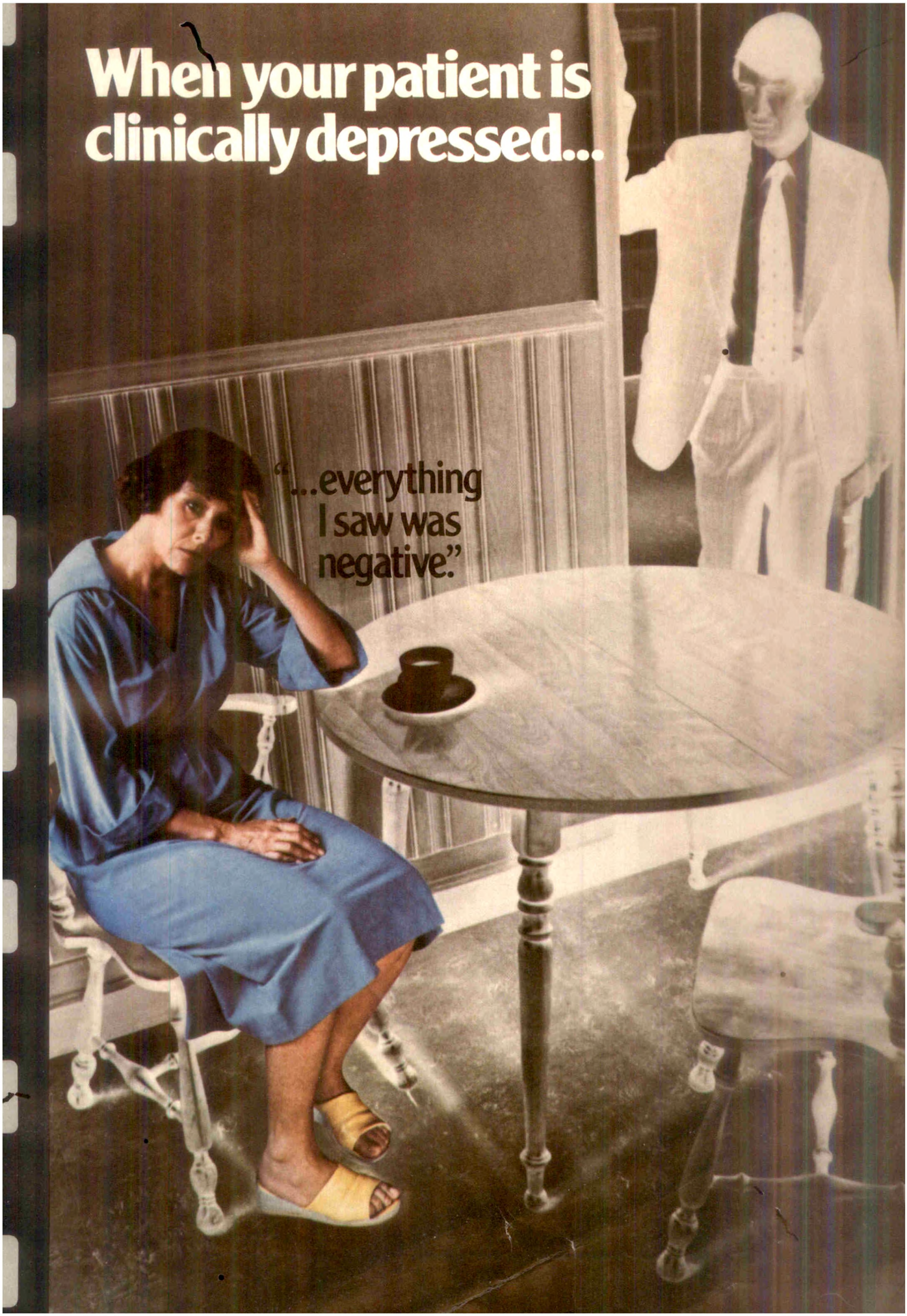
The Language of Change: Elements of Therapeutic Communication, *by Paul Watzlawick. New York, N.Y., Basic Books, 1978, 166 pp., $11.00.*

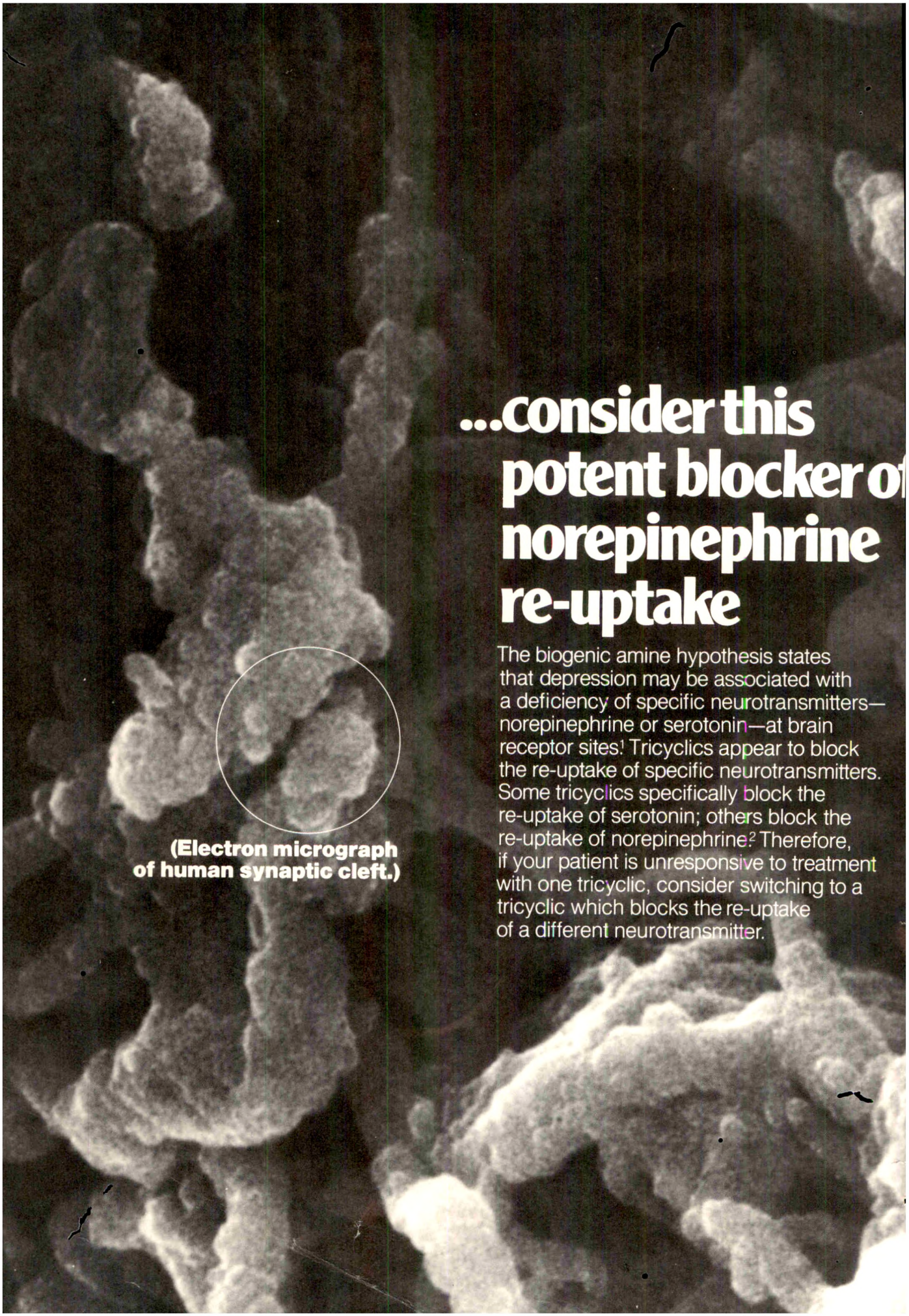
A Clinician's Manual of Hysterical Conversion Reactions, *by Michael I. Weintraub, M.D. Pelham Manor, N.Y., Interdisciplinary Communications Media (Flushing, N.Y., Scholium International, distributor), 1978, 118 pp., $10.50; $6.50 (paper).*

Psychopathy: A History of the Concepts: Analysis of the Origin and Development of a Family of Concepts in Psychopathology, *by Henry Werlinder. Stockholm, Sweden, Uppsala (Almqvist & Wiksell International, distributor), 1978, 218 pp., no price listed (paper).*

The Nature of Schizophrenia: New Approaches to Research and Treatment, *by Lyman C. Wynne, Rue L. Cromwell, and Steven Matthysse. New York, N.Y., John Wiley & Sons, 1978, 714 pp., $45.00.*

Normal Psychology of the Aging Process, revised ed., *edited by Norman E. Zinberg, M.D., and Irving Kaufman, M.D. New York, N.Y., International Universities Press, 1978, 277 pp., $15.00.*


When your patient is clinically depressed...
"...everything I saw was negative."



(Electron micrograph of human synaptic cleft.)

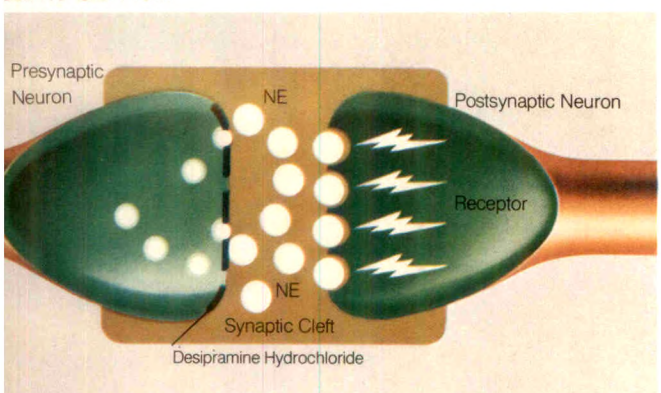
...consider this potent blocker of norepinephrine re-uptake

The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

Norpramin®
(desipramine hydrochloride tablets NF)
25, 50, 75, 100, 150 mg.

Potent blocker of norepinephrine re-uptake

According to current theory[1,3] some tricyclic antidepressants may fail in certain types of depression because they are not sufficiently potent blockers of norepinephrine re-uptake. Laboratory studies have shown that desipramine is the most potent inhibitor of norepinephrine re-uptake.[4] Norpramin may work in some patients when other tricyclic antidepressants do not.



Consider it now, consider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

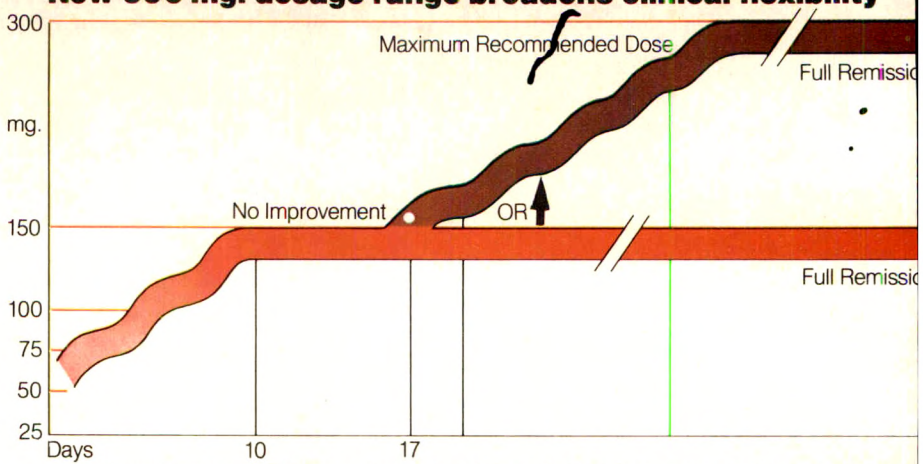
References:

1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Now 300 mg. dosage range broadens clinical flexibility



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed for Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of April, 1978

Merrell

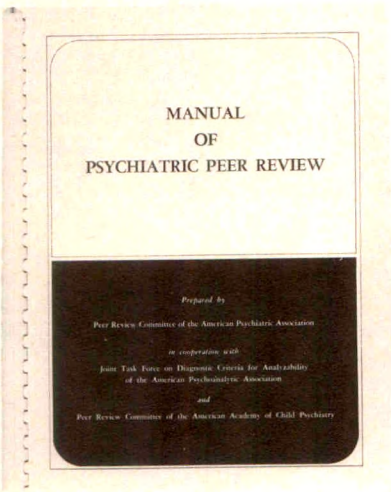
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MANUAL OF PSYCHIATRIC PEER REVIEW

by the Peer Review Committee of the American Psychiatric Association, Donald G. Langsley, M.D., Chairperson in cooperation with the Joint Task Force on Diagnostic Criteria for Analyzability of the American Psychoanalytic Association, William Offenkrantz, M.D., Chairperson, and the Peer Review Committee of the American Academy of Child Psychiatry. Larry B. Silver, M.D., Chairperson.



"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

Published by the American Psychiatric Association, Washington, D.C., September 1976. 114 pages. Spiral binding. Single copies, $6.00. For orders of 10 or more, $5.00 per copy.

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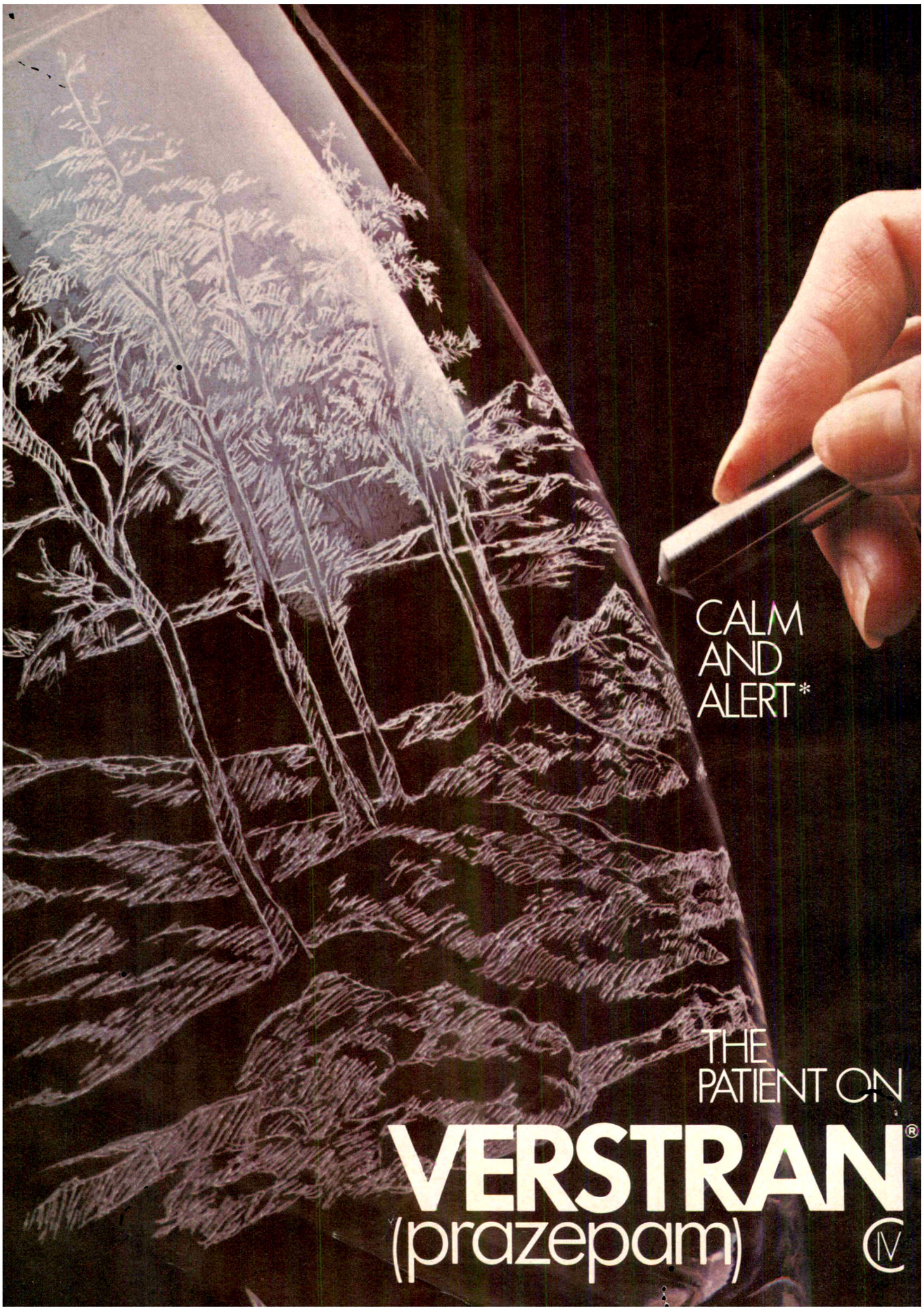
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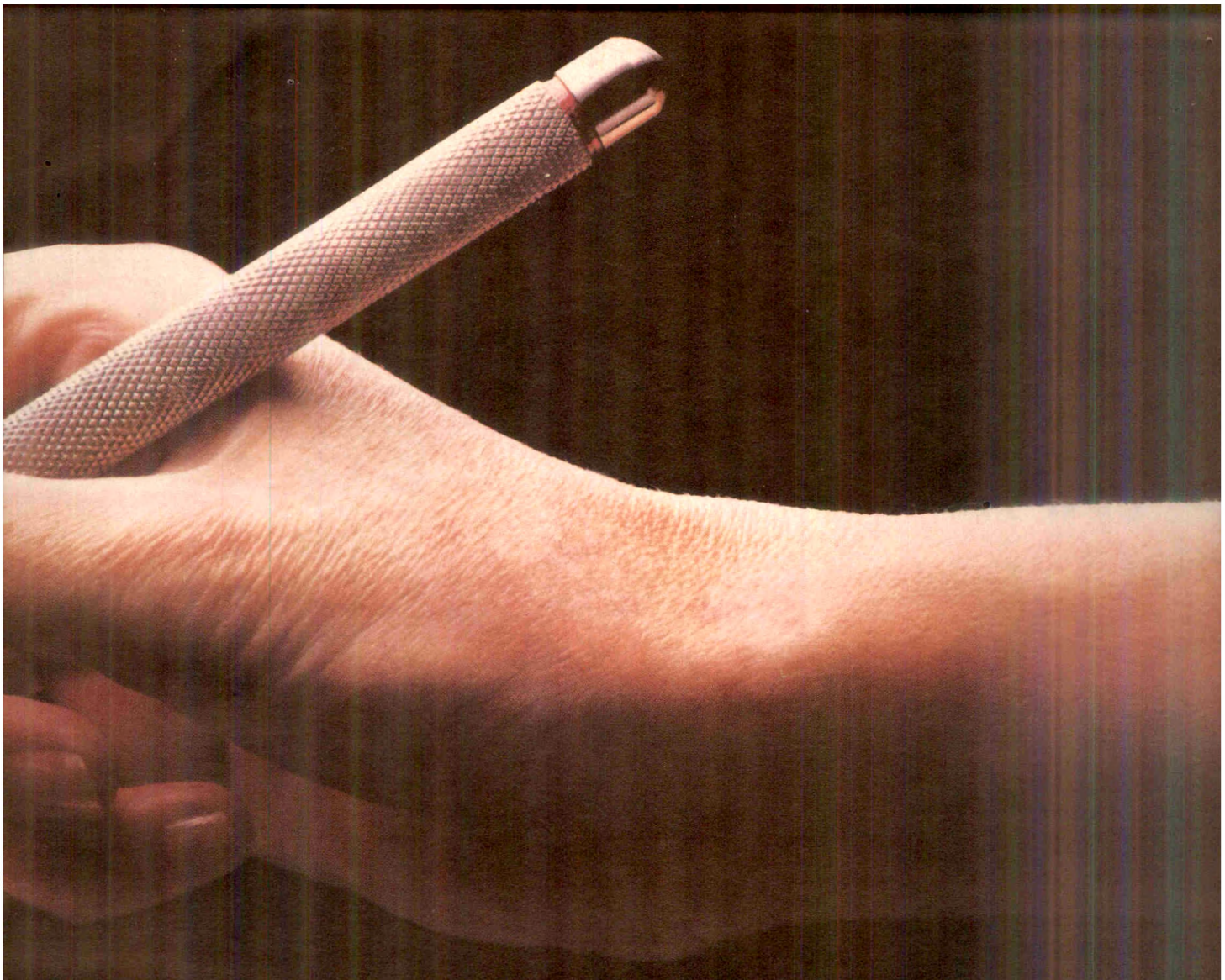
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- Other side effects, if present, are minimal†
- Verstran is appropriate concomitant therapy in most somatic disorders‡
- No accumulation after steady state is reached

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- The option of a *b.i.d.* dosage regimen, gradually adjustable within a range of 20 to 60 mg/day



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†Data on file, Warner/Chilcott Medical Department.

‡See package insert.

§At bedtime.

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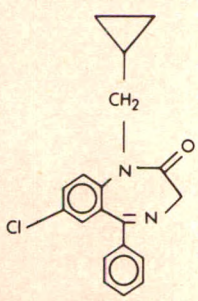
VERSTRAN®

(prazepam) Ⓒ IV

VERSTRAN® (prazepam) Ⓒ IV

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg b.i.d. for one week was 63 (±15 s.d.) hours before and 70 (±10 s.d.) hours after multiple dosing — a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

The effectiveness of Verstran (prazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51) and 1000 (N 0047-0276-60).

STORE BETWEEN 59°–86° F (15°–30° C).

VE-GP-85

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

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Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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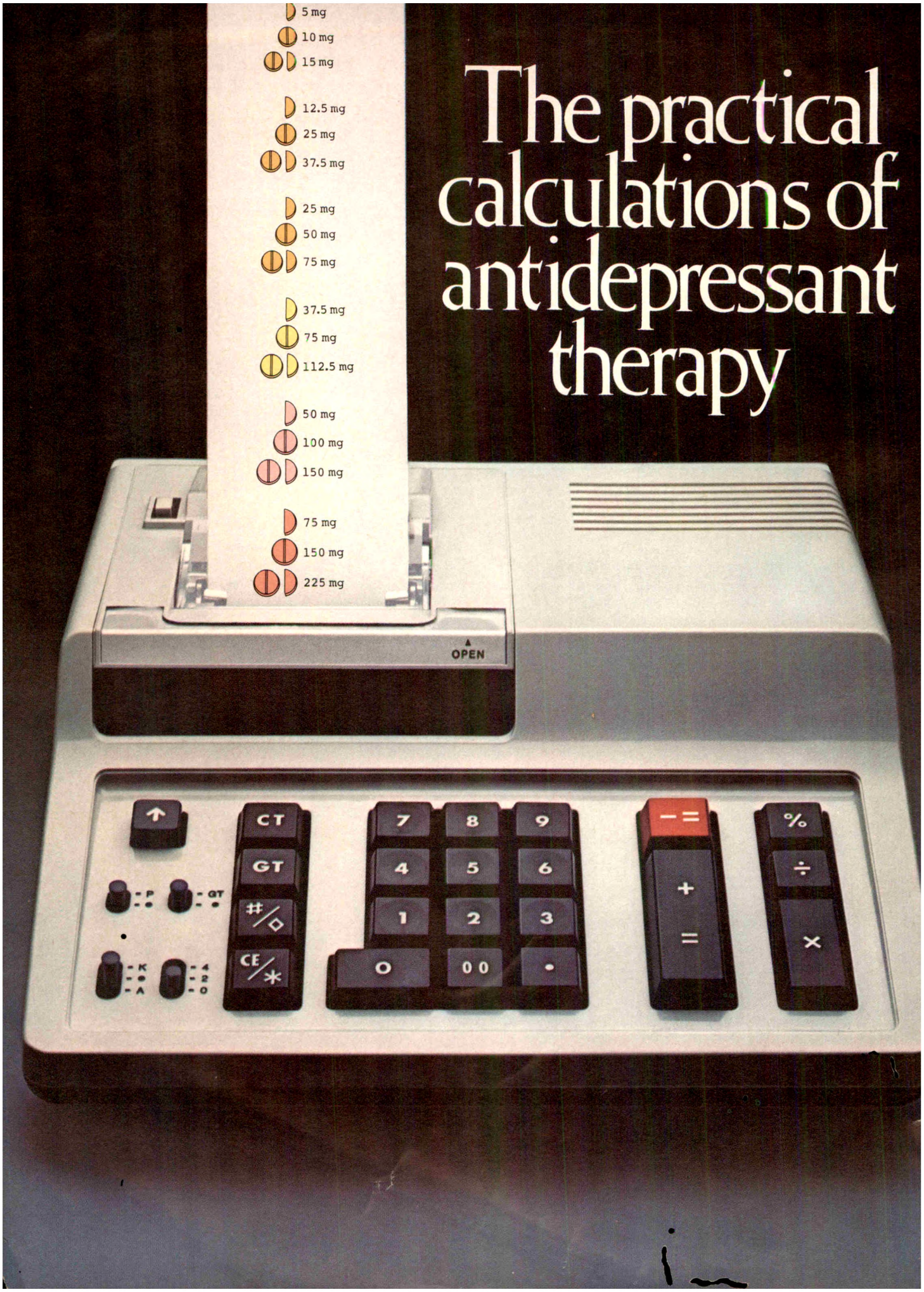
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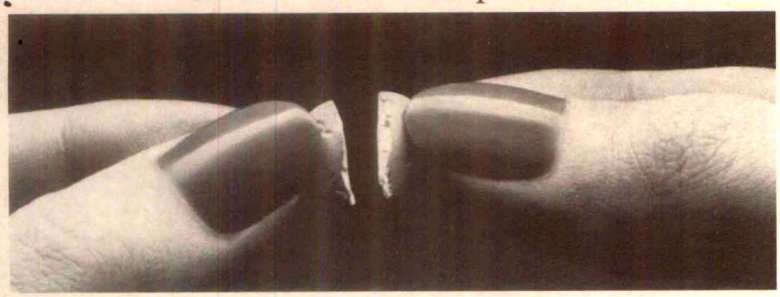
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Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

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Molindone HCl

Lidone™ treats the classic target symptoms



with less likelihood of certain troublesome effects

Prescribing Information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats Oral Dose —
20 mg/kg/day — 10 days, no adverse effect
40 mg/kg/day — 10 days, no adverse effect

Pregnant Mice Oral Dose —
20 mg/kg/day — 10 days, slight increase resorptions
40 mg/kg/day — 10 days, slight increase resorptions

Pregnant Rabbits Oral Dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg/day.
- — Increase to 100 mg/day in three or four days.
- — Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- — An increase to 225 mg/day may be required in patients with severe symptomatology.
- — Some chronic, treatment-resistant patients may require up to 400 mg/day; (however, the long-term safety of 400 mg/day has not been established).

Elderly or debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four times a day.
2. Moderate — 10 mg to 25 mg three or four times a day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone hydrochloride) capsules are supplied in bottles of 100 in the following dosage strengths and colors:

5 mg (**NDC** 0074-5542-13) blue and cream
10 mg (**NDC** 0074-5543-13) red and cream
25 mg (**NDC** 0074-5544-13) brown and cream

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*Benadryl — Trademark, Parke Davis and Co.
*Artane — Trademark, Lederle Laboratories
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The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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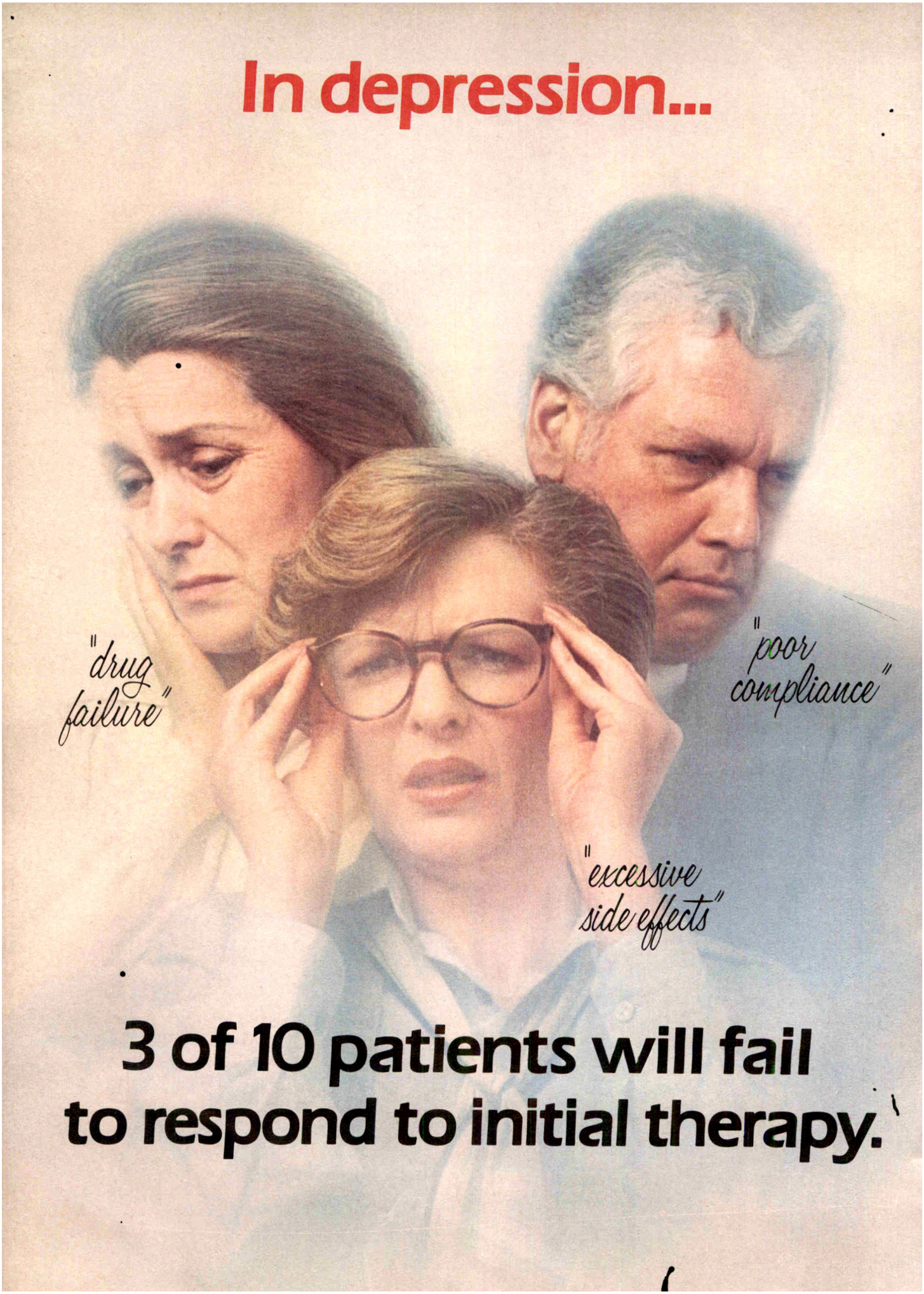
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Please see next page for brief summary of prescribing information and references.

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Indication: For relief of mental depression. **Contraindications:** Do not use MAO inhibitors concomitantly or within 2 weeks of the use of this drug. Hyperpyretic crises or severe convulsive seizures may occur with such combinations; potentiation of adverse reactions can be serious or even fatal. When substituting Pertofrane (desipramine HCl) in patients receiving an MAO inhibitor, allow an interval of at least 14 days. Initial dosage in such patients should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated following recent myocardial infarction and in patients with a known hypersensitivity to tricyclic antidepressants. **Warnings:** Activation of psychosis may occasionally be observed in schizophrenic patients. Due to atropine-like effects and sympathomimetic potentiation, use only with the greatest care in patients with narrow-angle glaucoma or urethral or ureteral spasm. Do not use in patients with the following conditions unless the need outweighs the risk: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (may lower seizure threshold). This drug may block the action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents. Hypertensive episodes have been observed during surgery. The concurrent use of other central nervous system drugs or alcohol may potentiate adverse effects. Since many such drugs may be used during surgery, desipramine should be discontinued prior to elective procedures. The potentiation resulting from excessive use of alcohol may increase the danger inherent in a suicide attempt or overdosage. Caution patients on the possibility of impaired ability to operate a motor vehicle or dangerous machinery. Do not use in women who are or may become pregnant, or in children under 12 years of age, unless the clinical situation warrants the potential risk. Because of increased sensitivity to the drug, use lower than normal dosage in adolescent and geriatric patients. **Precautions:** Potentially suicidal patients require careful supervision and protective measures during therapy. Prescriptions should be limited to small quantities. Discontinuation of the drug may be necessary in the presence of increased agitation and anxiety shifting to hypomanic or manic excitement. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents). Prescribe cautiously in hyperthyroid patients and in those receiving thyroid medications; transient cardiac arrhythmias have occurred in rare instances. Periodic blood and liver studies should supplement careful clinical observations in all patients undergoing extended courses of therapy. **Adverse Reactions:** The following have been reported: *Nervous System:* dizziness, drowsiness, insomnia, headache, disturbed visual accommodation, tremor, unsteadiness, tinnitus, paresthesias, changes in EEG patterns, epileptiform seizures, mild extrapyramidal activity, falling and neuromuscular incoordination. A confusional state (with such symptoms as hallucinations and disorientation), particularly in older patients and at higher dosage, may require discontinuation of the drug. *Gastrointestinal Tract:* anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea. *Skin:* skin rashes (including photosensitization), perspiration and flushing sensations. *Liver:* rare cases of transient jaundice (apparently of an obstructive nature) and liver damage. If jaundice or abnormalities in liver function tests occur, discontinue the drug and investigate. *Blood Elements:* bone-marrow depression, agranulocytosis, thrombocytopenia and purpura. If these occur, discontinue the drug. Transient eosinophilia has been observed. *Cardiovascular System:* orthostatic hypotension and tachycardia. Carefully supervise patients requiring concomitant vasodilating therapy, particularly during initial phases. *Genitourinary System:* urinary frequency or retention and impotence. *Endocrine System:* occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement, and decreased libido and estrogenic effect. *Sensitivity:* urticaria and rare instances of drug fever and cross-sensitivity with imipramine. **Dosage:** All patients except geriatric and adolescent: 75 to 150 mg./day in divided doses or as single daily dose. Dosage may be increased up to 200 mg. daily. Geriatric and adolescent patients should usually be started with lower dosage (25 to 50 mg. daily) and may not tolerate higher doses. Dosage may be increased up to 100 mg. daily. Lower maintenance dosages should be continued for at least 2 months after obtaining a satisfactory response. Therapy may be given in divided doses or as a single daily dose. **How Supplied:** 25 mg. capsules (pink), bottles of 100 and 1000. Also, 50 mg. capsules (maroon and pink), bottles of 100 and 1000.

References:

1. Barringer, T.J.: Desipramine (Pertofrane) in the treatment of depression, Psychosomatics, Third Symposium: Anxiety and Depression 6:326, 1965. 2. Bondurant, W.W. III and Ford, H.: Depression and manic depression in Current Therapy 1976 (Conn, H.F., ed.), Philadelphia, Saunders, 1976, p. 841. 3. Ayd, F.J., Jr.: Central anticholinergic activity and tricyclic antidepressant efficacy, Int. Drug Ther. Newsletter 10:21, 1975.

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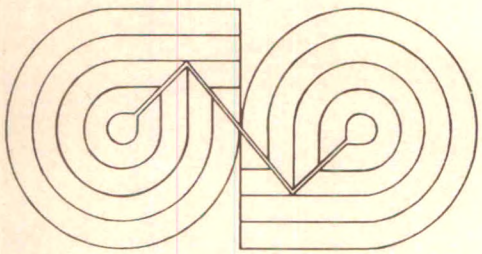
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They thought Ed Sloan was just tired...

maybe he had narcolepsy

A narcoleptic can fall asleep almost anywhere, anytime. The results can be at least embarrassing...at most devastating.

Narcolepsy is often unrecognized

As a primary sleep abnormality narcolepsy is characterized by sleepiness, inappropriate sleep episodes, and cataplexy (episodes of muscular weakness precipitated by strong emotion). These are often accompanied by hypnagogic hallucinations, automatic behavior and/or disturbed nocturnal sleep. Sleep apnea may be a severe life-threatening complication of narcolepsy.

Besides its medical implications, narcolepsy may have drastic social consequences.

"All too often, the patient with disabling hypersomnolence is ignored by physicians for years and even decades, with the consequent tragic toll on his ability to learn and to work."[1]

Ritalin can help in narcolepsy

For narcoleptic sleepiness and sleep episodes, the cited authorities[1,2] most frequently use Ritalin (methylphenidate).

References:

1. Dement WC, Carskadon MA, Guilleminault C, Zarcone VP: Narcolepsy: Diagnosis and Treatment. Primary Care 3:609-623, December 1976.

2. Daly DD, Yoss RE: Narcolepsy, in *Handbook of Clinical Neurology*: Vol 15, *The Epilepsies*, Magnus O, Lorentz De Haas AM (Eds.), Amsterdam, North-Holland Publishing Company, 1974, pp 836-852.

Ritalin®

C II

(methylphenidate)

For the 200,000 narcoleptics who can't stay awake

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Ritalin® (methylphenidate) C II

For the 200,000 narcoleptics who can't stay awake



Ritalin® hydrochloride C II
(methylphenidate hydrochloride USP)
TABLETS

INDICATION
Narcolepsy.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times daily, preferably 30 to 45 minutes before meals. Dosage will depend upon indication and individual response.

Average dosage is 20 to 30 mg daily. Some patients may require 40 to 60 mg daily. In others, 10 to 15 mg daily will be adequate. The few patients who are unable to sleep if medication is taken late in the day should take the last dose before 6 p.m.

HOW SUPPLIED
Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500 and 1000.

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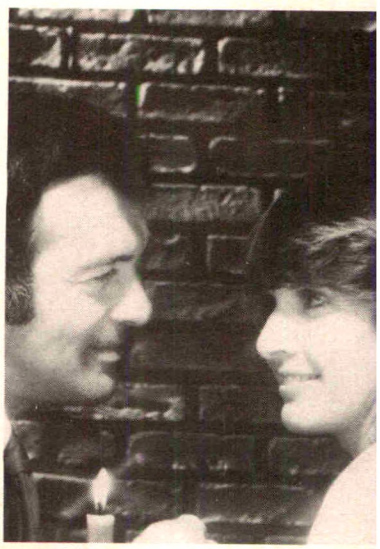
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TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving. The drug may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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For a wide range of patients with marked agitation and depression

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TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

J8TR30 (DC6613215)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486.

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ASSISTANT SUPERINTENDENT FOR PROFESSIONAL SERVICES

New Hampshire Hospital, Concord, N.H., an 800 bed JCAH-Accredited psychiatric hospital, is seeking an Assistant Superintendent for Professional Services to be responsible for the direction and coordination of clinical programs for the treatment, care and management of mentally ill patients. New Hampshire Hospital, the only public psychiatric hospital in N.H., is located in Concord, a city of approximately 30,000 people, 75 minutes north of Boston, MA. Salary ranges from $30,740-$38,690, plus an additional $3,000 per year if board certified. Possibility of faculty appointment to major university. Housing and other fringe benefits are available. Candidate must be a physician-psychiatrist licensed or eligible for licensure as an M.D. in New Hampshire and certified or eligible for certification by the American Board of Neurology and Psychiatry.

Interested applicants should contact C. Clifton DuBois, Ph.D., Superintendent at New Hampshire Hospital, 105 Pleasant Street, Concord, New Hampshire 03301. Telephone (603) 271-2205.

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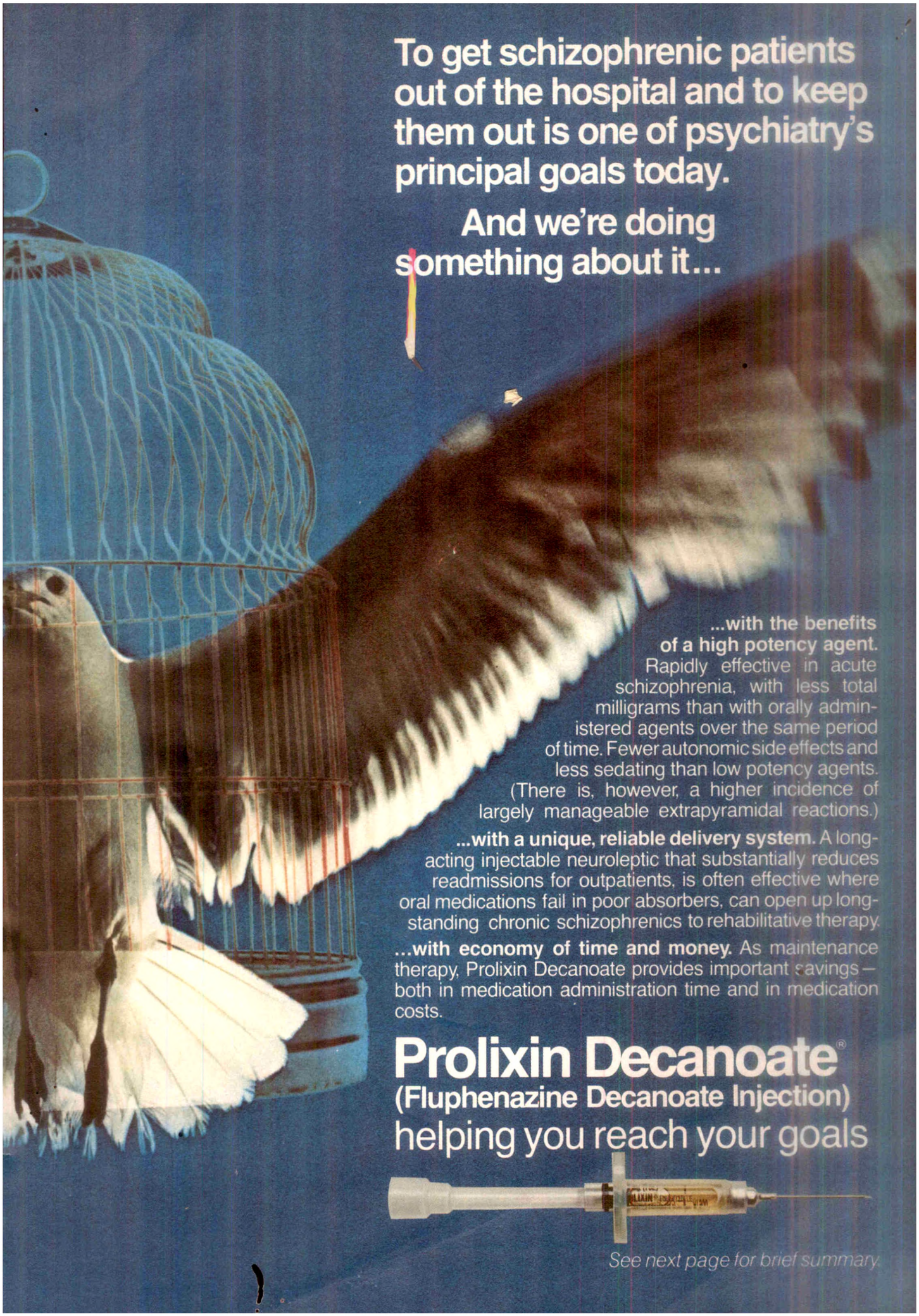
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Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2%(w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

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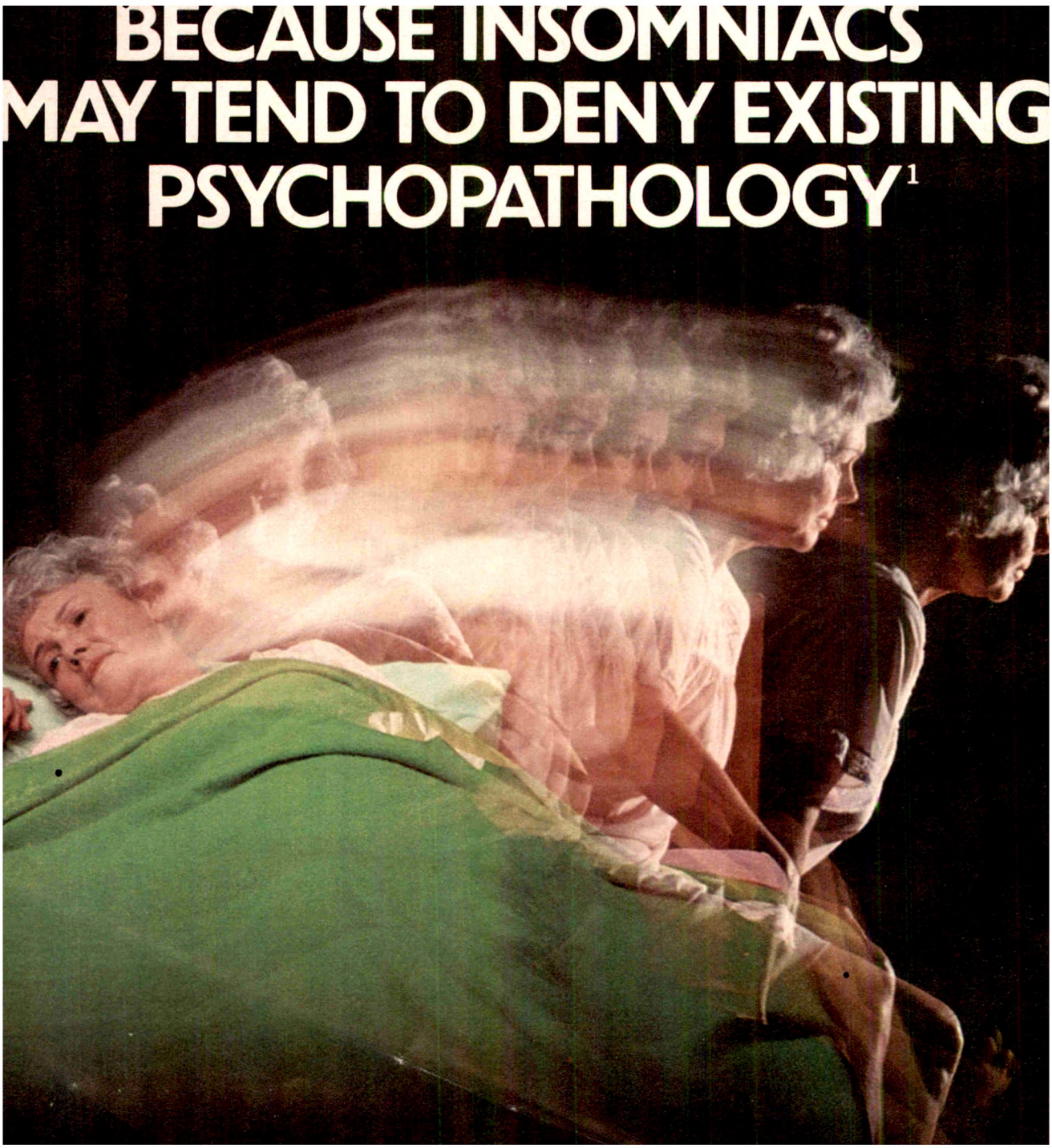
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BECAUSE INSOMNIACS
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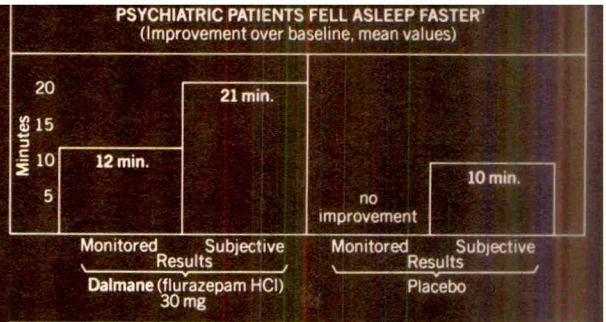
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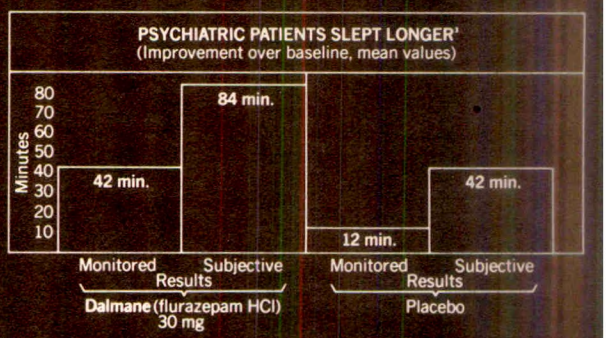
for symptomatic relief of insomnia during psychotherapy

As you know, many insomniacs focus on somatic complaints rather than face the emotional problems that are the basis of their illness. When denial presents a block to effective therapy, pharmacologic intervention may be indicated. In such cases Dalmane, with sleep laboratory proof of efficacy, may prove especially useful as adjunctive treatment.[1]

proved effective for insomniac psychiatric patients[2,3]

In a placebo-controlled study, Dalmane 30 mg *h.s.*, administered for 7 nights to 49 hospitalized psychiatric patients, reduced the time required to fall asleep and increased the sleep duration time. These data, recorded by special monitors, agreed with subjective estimates made by patients on daily post-sleep questionnaires.





proved effective for 28 consecut nights in the sleep laboratory[4-6]

Dalmane (flurazepam HCl) is the only hypnotic proved effective for a 28-night period.[4-6] In sleep laboratory stud of chronic insomniacs, Dalmane has demonstrated a sig nificant reduction in the time required to fall asleep and in total wake time, during short-, intermediate- and longe term administration.[4] Where a hypnotic agent is needed for more than a few nights, Dalmane appears to be relatively safe as well as effective.[6] Although prolonged adm istration of Dalmane is seldom necessary, with repeated use periodic blood counts and liver and kidney function tests are recommended.



Please see following page for a summary of product information.

ROC

EFFECTIVE ADJUNCTIVE THERAPY IN PSYCHIATRIC PRACTICE

DALMANE®
(flurazepam HCl)

One 30-mg capsule *h.s.*—usual adult dosage
(15 mg may suffice in some patients).
One 15-mg capsule *h.s.*—initial dosage for elderly or debilitated patients.

- ☐ sleep within 17 minutes, on average, proved in sleep laboratory studies[3]
- ☐ sleep for an average of 7 to 8 hours proved in sleep laboratory studies[3]
- ☐ no need to repeat dose or increase dosage[2-6]

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES: 1. Kales A, Kales JD, Humphrey FJ II: Sleep and dreams, in *Comprehensive Textbook of Psychiatry—II*, ed 2, edited by Freedman AM, Kaplan HI, Sadock BJ. Baltimore, The Williams & Wilkins Company, Vol 1, 1975, p. 114. 2. Jacobson A, *et al: Psychophysiology 7:* 345, Sep 1970. 3. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ. 4. Kales A, *et al: Clin Pharmacol Ther 19:* 576-583, May 1976. 5. Kales A, *et al: Clin Pharmacol Ther 18:* 356-363, Sep 1975. 6. Dement WC, Guilleminault C, Zarcone V: Progress in clinical sleep research. Scientific exhibit at the American Medical Association, Atlantic City NJ, Jun 14-18, 1975

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INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

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Psychiatry and the Fiscal Third Party

By Paul Chodoff

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Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

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Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

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Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or obscure diagnosis of conditions such as intestinal obstruction, brain tumor, and Reye's syndrome (see Warnings). Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.



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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26

2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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The November 1978 issue of

The American Journal of Psychiatry

will feature

- **Psychodynamics and Developmental Psychology of the Borderline Patient: A Review of the Literature**

  By Edward R. Shapiro



Hallucinations... delusions... thought disorders... controlled

Navane (thiothixene) rapidly controls hallucinations as well as the agitation and hostility patterns they frequently generate.

Rapid control of psychotic symptoms on admission

Navane provides effective, rapid control of the major symptoms of psychosis including hallucinations, delusions, agitation, hostility, and thought disorders.[1-5] Navane promotes rapid progress, usually without the over-sedation or cardiovascular problems that can impede the therapeutic course.

Continued long-term improvement

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Navane allows patients to remain alert and active, seldom causing over-sedation or drowsiness.[7] Hypotension and other cardiovascular reactions[5,6,8] are seldom reported and the occurrence of unpleasant anticholinergic side effects such as dry mouth or constipation is rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled through dosage adjustments or antiparkinson agents.

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BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane* (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, 1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. Denber HCB, Turns D: Double blind comparison of thiothixene and trifluoperazine in acute schizophrenia. *Psychosomatics* 13:100-104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: The place of thiothixene in the treatment of schizophrenic patients. *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, 1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 130th Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlrabe J, Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, et al: Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients. in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

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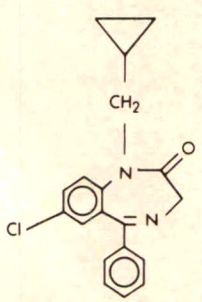
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Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (±15 s.d.) hours before and 70 (±10 s.d.) hours after multiple dosing — a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

The effectiveness of Verstran (prazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

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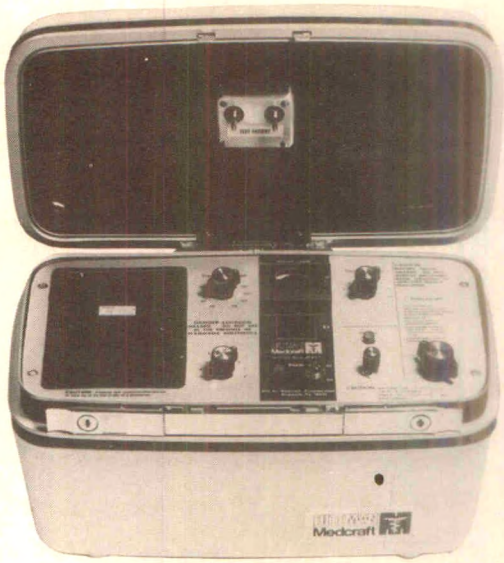
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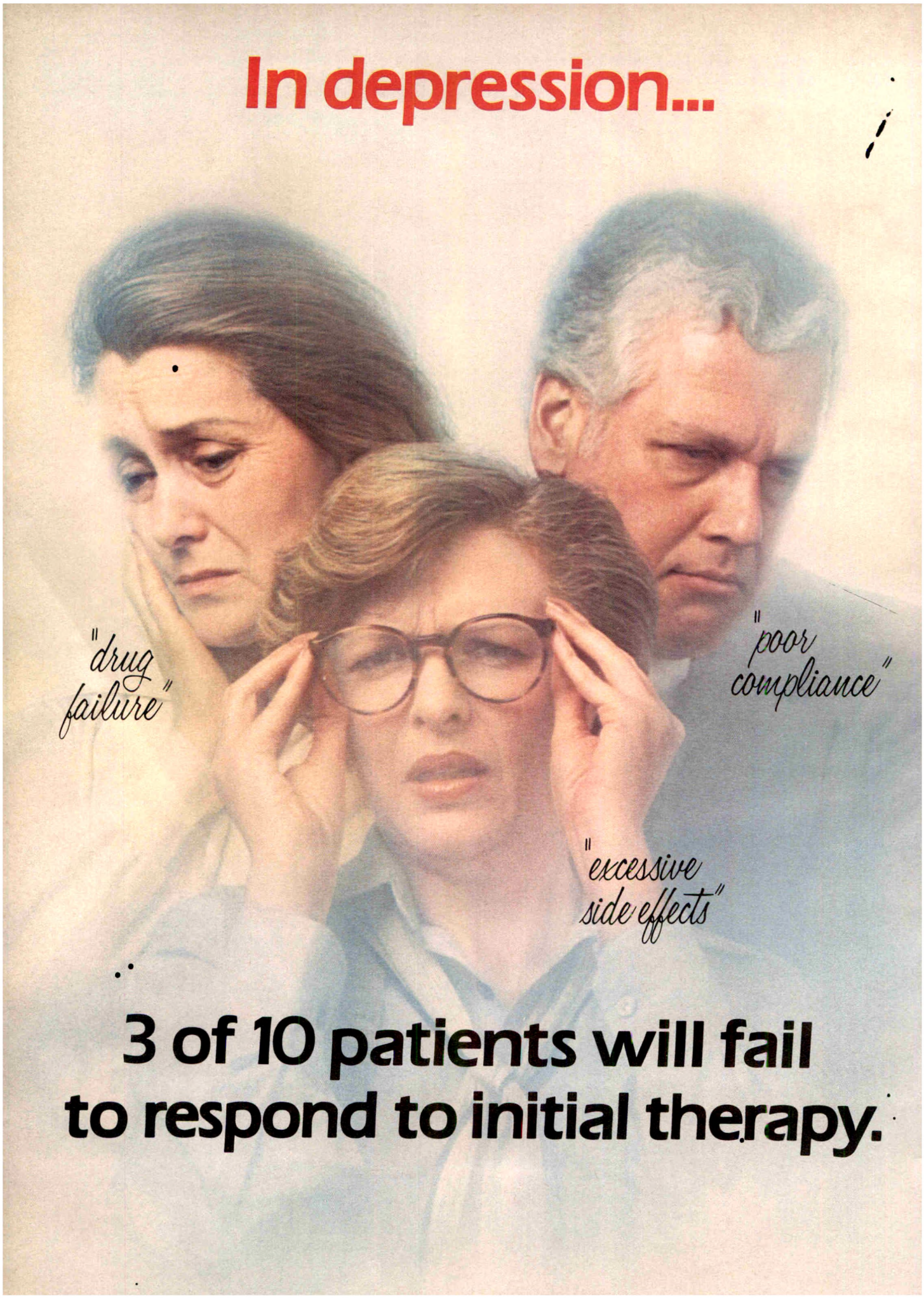
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Do not use in patients with the following conditions unless the need outweighs the risk: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (may lower seizure threshold). This drug may block the action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents. Hypertensive episodes have been observed during surgery. The concurrent use of other central nervous system drugs or alcohol may potentiate adverse effects. Since many such drugs may be used during surgery, desipramine should be discontinued prior to elective procedures. The potentiation resulting from excessive use of alcohol may increase the danger inherent in a suicide attempt or overdosage. Caution patients on the possibility of impaired ability to operate a motor vehicle or dangerous machinery. Do not use in women who are or may become pregnant, or in children under 12 years of age, unless the clinical situation warrants the potential risk. Because of increased sensitivity to the drug, use lower than normal dosage in adolescent and geriatric patients. **Precautions:** Potentially suicidal patients require careful supervision and protective measures during therapy. Prescriptions should be limited to small quantities. Discontinuation of the drug may be necessary in the presence of increased agitation and anxiety shifting to hypomanic or manic excitement. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents). Prescribe cautiously in hyperthyroid patients and in those receiving thyroid medications; transient cardiac arrhythmias have occurred in rare instances. Periodic blood and liver studies should supplement careful clinical observations in all patients undergoing extended courses of therapy. **Adverse Reactions:** The following have been reported: *Nervous System:* dizziness, drowsiness, insomnia, headache, disturbed visual accommodation, tremor, unsteadiness, tinnitus, paresthesias, changes in EEG patterns, epileptiform seizures, mild extrapyramidal activity, falling and neuromuscular incoordination. A confusional state (with such symptoms as hallucinations and disorientation), particularly in older patients and at higher dosage, may require discontinuation of the drug. *Gastrointestinal Tract:* anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea. *Skin:* skin rashes (including photosensitization), perspiration and flushing sensations. *Liver:* rare cases of transient jaundice (apparently of an obstructive nature) and liver damage. If jaundice or abnormalities in liver function tests occur, discontinue the drug and investigate. *Blood Elements:* bone-marrow depression, agranulocytosis, thrombocytopenia and purpura. If these occur, discontinue the drug. Transient eosinophilia has been observed. *Cardiovascular System:* orthostatic hypotension and tachycardia. Carefully supervise patients requiring concomitant vasodilating therapy, particularly during initial phases. *Genitourinary System:* urinary frequency or retention and impotence. *Endocrine System:* occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement, and decreased libido and estrogenic effect. *Sensitivity:* urticaria and rare instances of drug fever and cross-sensitivity with imipramine. **Dosage:** All patients except geriatric and adolescent: 75 to 150 mg./day in divided doses or as single daily dose. Dosage may be increased up to 200 mg. daily. 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References:

1. Barringer, T.J.: Desipramine (Pertofrane) in the treatment of depression, Psychosomatics, Third Symposium: Anxiety and Depression 6:326, 1965. 2. Bondurant, W.W. III and Ford, H.: Depression and manic depression in Current Therapy 1976 (Conn, H.F., ed.), Philadelphia, Saunders, 1976, p. 841. 3. Ayd, F.J., Jr.: Central anticholinergic activity and tricyclic antidepressant efficacy, Int. Drug Ther. Newsletter 10:21, 1975.

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FJ:97:34

They thought Ed Sloan was just tired...

maybe he had narcolepsy

A narcoleptic can fall asleep almost anywhere, anytime. The results can be at least embarrassing...at most devastating.

Narcolepsy is often unrecognized

As a primary sleep abnormality narcolepsy is characterized by sleepiness, inappropriate sleep episodes, and cataplexy (episodes of muscular weakness precipitated by strong emotion). These are often accompanied by hypnagogic hallucinations, automatic behavior and/or disturbed nocturnal sleep. Sleep apnea may be a severe life-threatening complication of narcolepsy.

Besides its medical implications, narcolepsy may have drastic social consequences.

"All too often, the patient with disabling hypersomnolence is ignored by physicians for years and even decades, with the consequent tragic toll on his ability to learn and to work."[1]

Ritalin can help in narcolepsy

For narcoleptic sleepiness and sleep episodes, the cited authorities[1,2] most frequently use Ritalin (methylphenidate).

References:

1. Dement WC, Carskadon MA, Guilleminault C, Zarcone VP: Narcolepsy: Diagnosis and Treatment. Primary Care 3:609-623, December 1976.
2. Daly DD, Yoss RE: Narcolepsy, in *Handbook of Clinical Neurology*: Vol 15, *The Epilepsies*, Magnus O, Lorentz De Haas AM (Eds.), Amsterdam, North-Holland Publishing Company, 1974, pp 836-852.

Ritalin® C II
(methylphenidate)

For the 200,000 narcoleptics who can't stay awake

Please turn page for brief prescribing information.

CIBA

Ritalin® C II
(methylphenidate)

For the 200,000 narcoleptics who can't stay awake



Ritalin® hydrochloride C II
(methylphenidate hydrochloride USP)
TABLETS

INDICATION
Narcolepsy.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times daily, preferably 30 to 45 minutes before meals. Dosage will depend upon indication and individual response.

Average dosage is 20 to 30 mg daily. Some patients may require 40 to 60 mg daily. In others, 10 to 15 mg daily will be adequate. The few patients who are unable to sleep if medication is taken late in the day should take the last dose before 6 p.m.

HOW SUPPLIED
Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500 and 1000.

C76-16 (Rev. 7/76) 665381

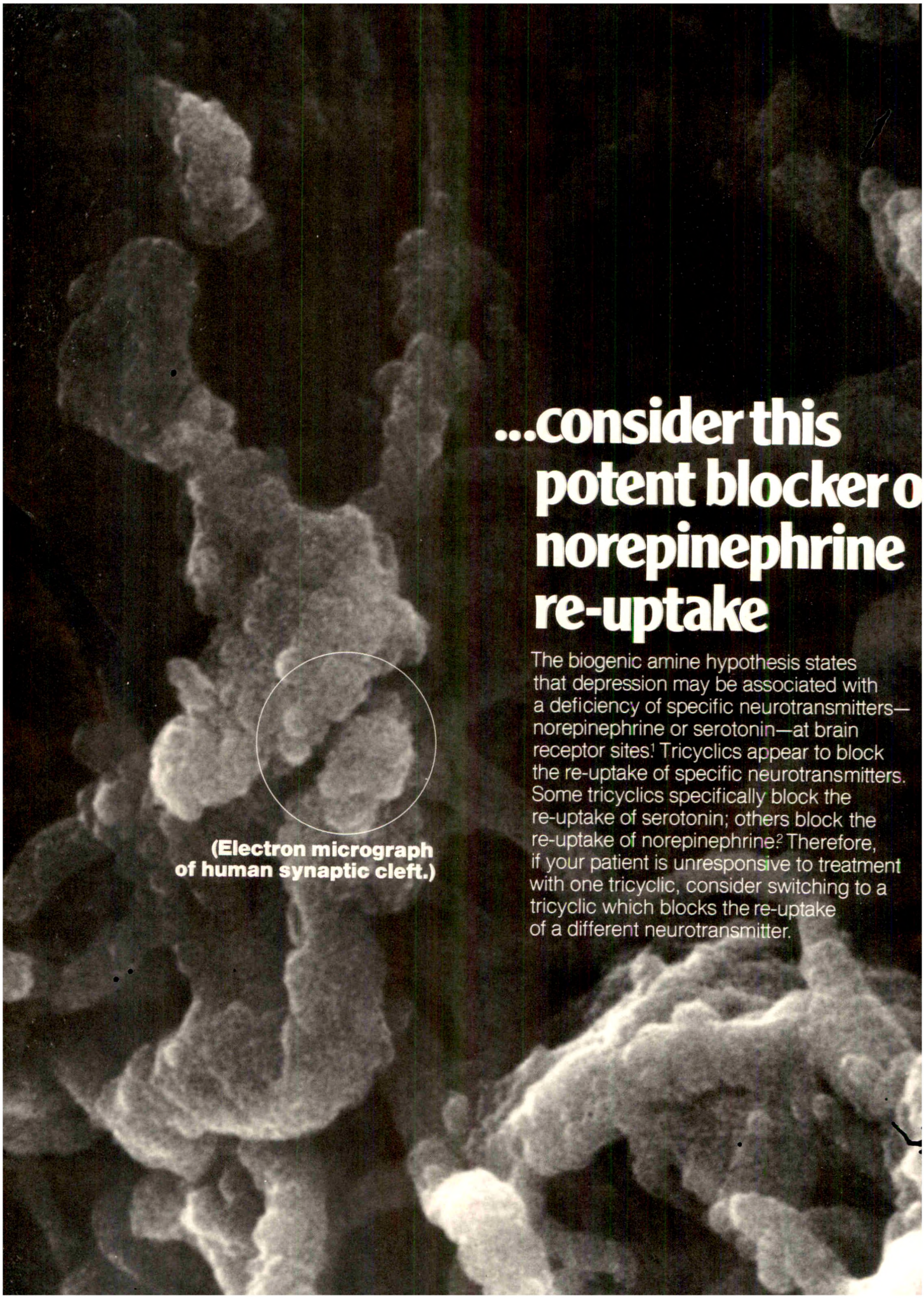
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When your patient is clinically depressed...
"...everything I saw was negative."



...consider this potent blocker o norepinephrine re-uptake

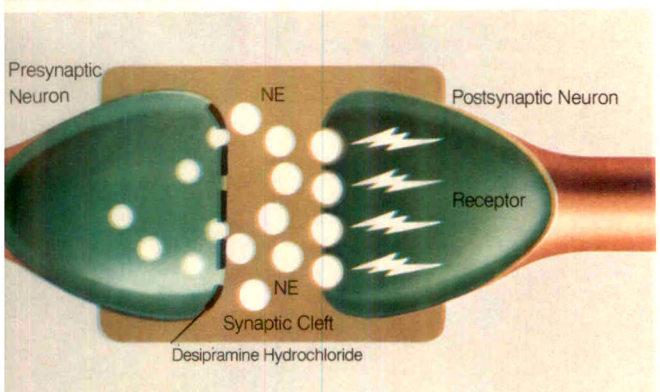
The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

(Electron micrograph of human synaptic cleft.)

Norpramin®
(desipramine hydrochloride tablets NF)
25, 50, 75, 100, 150 mg.

Potent blocker of norepinephrine re-uptake

According to current theory[1,3] some tricyclic antidepressants may fail in certain types of depression because they are not sufficiently potent blockers of norepinephrine re-uptake. Laboratory studies have shown that desipramine is the most potent inhibitor of norepinephrine re-uptake.[4] Norpramin may work in some patients when other tricyclic antidepressants do not.



Consider it now, consider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

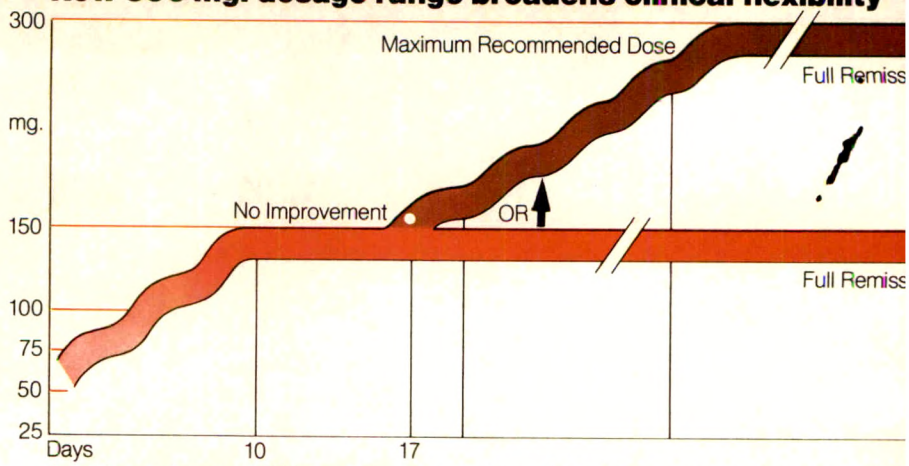
References:

1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Now 300 mg. dosage range broadens clinical flexibility



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed fo Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: **1.** Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage. **PRECAUTIONS:** **1.** It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

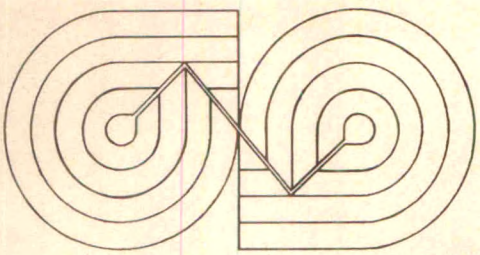
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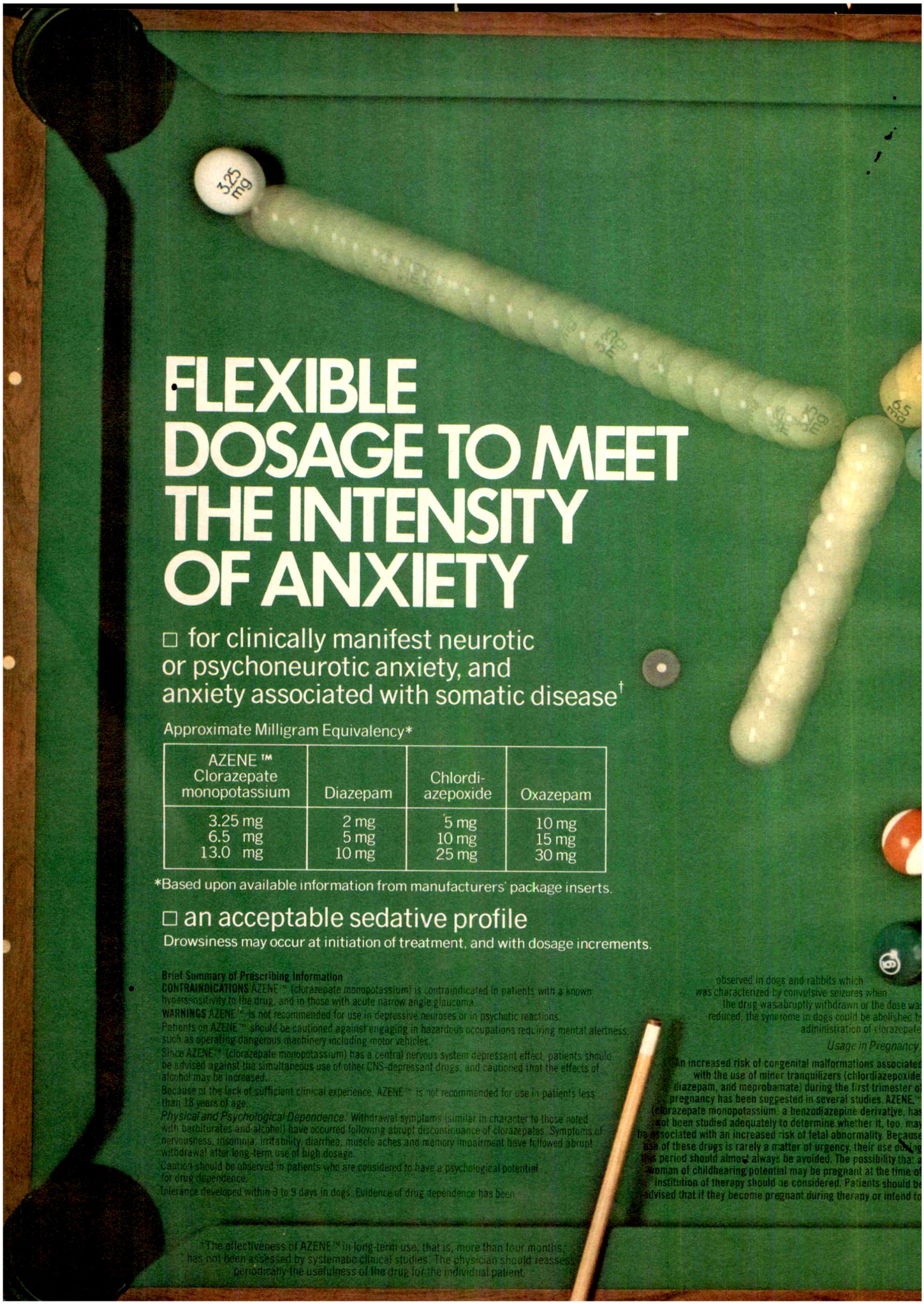
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FLEXIBLE DOSAGE TO MEET THE INTENSITY OF ANXIETY

□ for clinically manifest neurotic or psychoneurotic anxiety, and anxiety associated with somatic disease†

Approximate Milligram Equivalency*

AZENE ™ Clorazepate monopotassium	Diazepam	Chlordi-azepoxide	Oxazepam
3.25 mg	2 mg	5 mg	10 mg
6.5 mg	5 mg	10 mg	15 mg
13.0 mg	10 mg	25 mg	30 mg

*Based upon available information from manufacturers' package inserts.

□ an acceptable sedative profile

Drowsiness may occur at initiation of treatment, and with dosage increments.

Brief Summary of Prescribing Information

CONTRAINDICATIONS AZENE™ (clorazepate monopotassium) is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS AZENE™ is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on AZENE™ should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since AZENE™ (clorazepate monopotassium) has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, AZENE™ is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence: Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

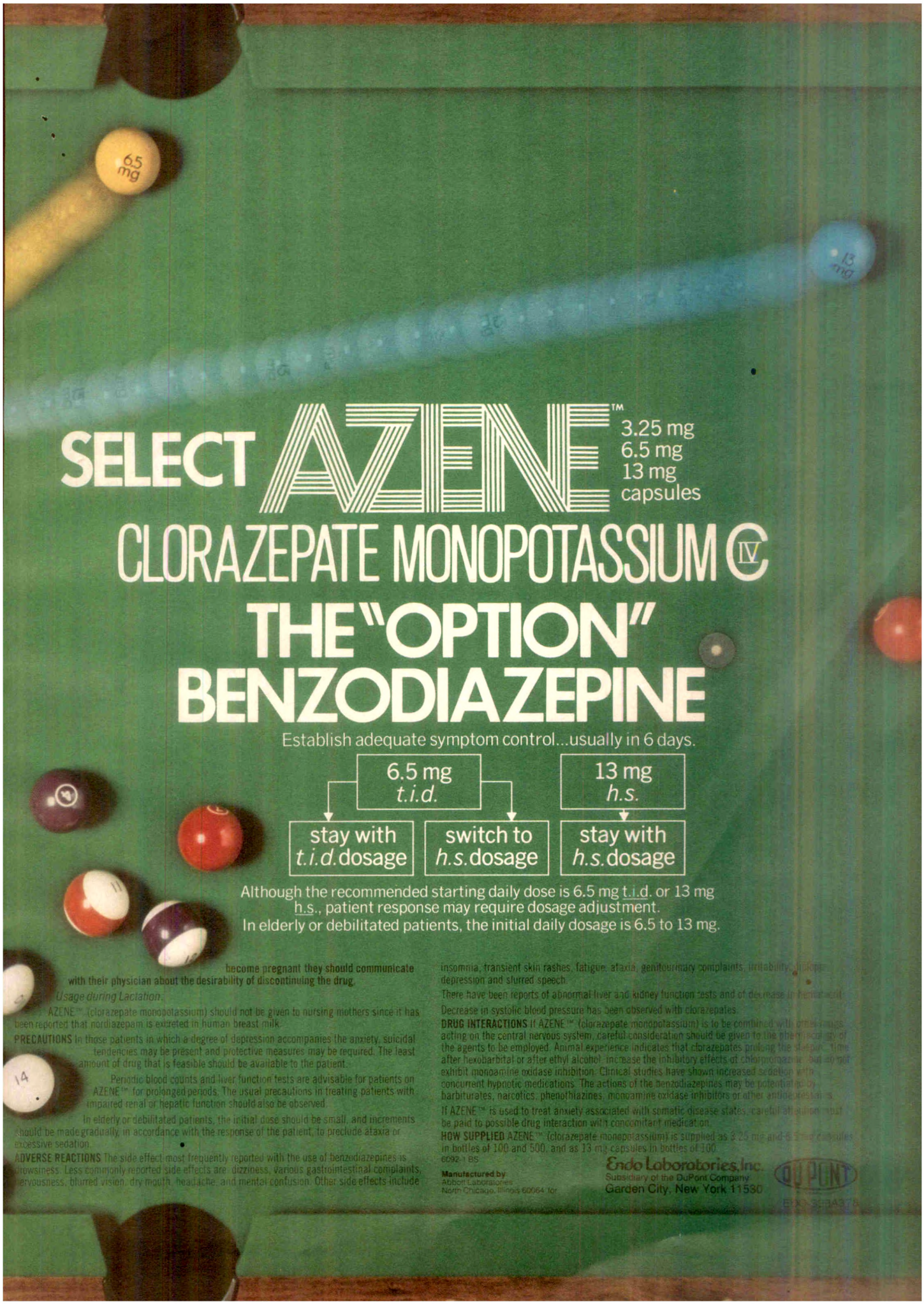
Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Tolerance developed within 3 to 9 days in dogs. Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. AZENE™ (clorazepate monopotassium), a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to

†The effectiveness of AZENE™ in long-term use, that is, more than four months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.



SELECT AZENE™

3.25 mg
6.5 mg
13 mg
capsules

CLORAZEPATE MONOPOTASSIUM C IV

THE "OPTION" BENZODIAZEPINE

Establish adequate symptom control...usually in 6 days.

6.5 mg *t.i.d.*

- stay with *t.i.d.* dosage
- switch to *h.s.* dosage

13 mg *h.s.*

- stay with *h.s.* dosage

Although the recommended starting daily dose is 6.5 mg t.i.d. or 13 mg h.s., patient response may require dosage adjustment.
In elderly or debilitated patients, the initial daily dosage is 6.5 to 13 mg.

become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation.

AZENE™ (clorazepate monopotassium) should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Periodic blood counts and liver function tests are advisable for patients on AZENE™ for prolonged periods. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS The side effect most frequently reported with the use of benzodiazepines is drowsiness. Less commonly reported side effects are: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects include insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, initial depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed with clorazepates.

DRUG INTERACTIONS If AZENE™ (clorazepate monopotassium) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that clorazepates prolong the sleep time after hexobarbital or ethyl alcohol, increase the inhibitory effects of chlorpromazine, but do not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If AZENE™ is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

HOW SUPPLIED AZENE™ (clorazepate monopotassium) is supplied as 3.25 mg and 6.5 mg capsules in bottles of 100 and 500, and as 13 mg capsules in bottles of 100.

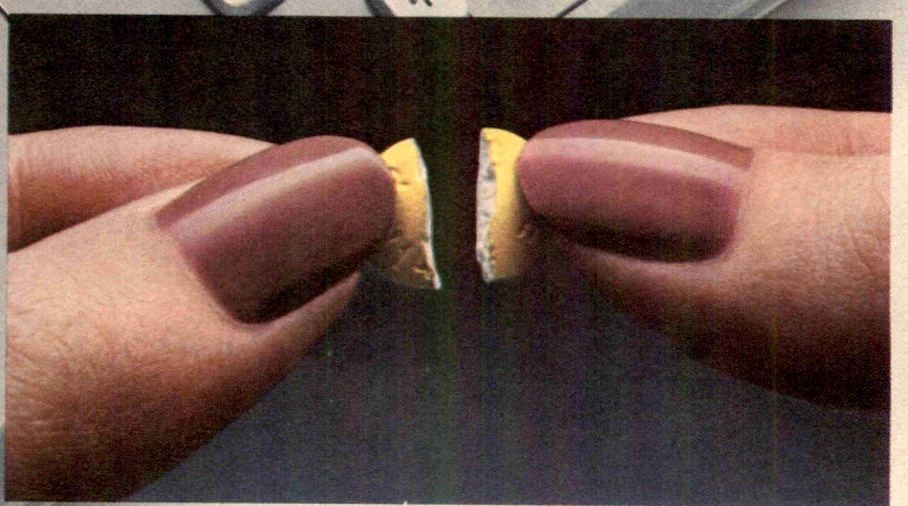
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Abbott Laboratories
North Chicago, Illinois 60064 for

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Subsidiary of the DuPont Company
Garden City, New York 11530

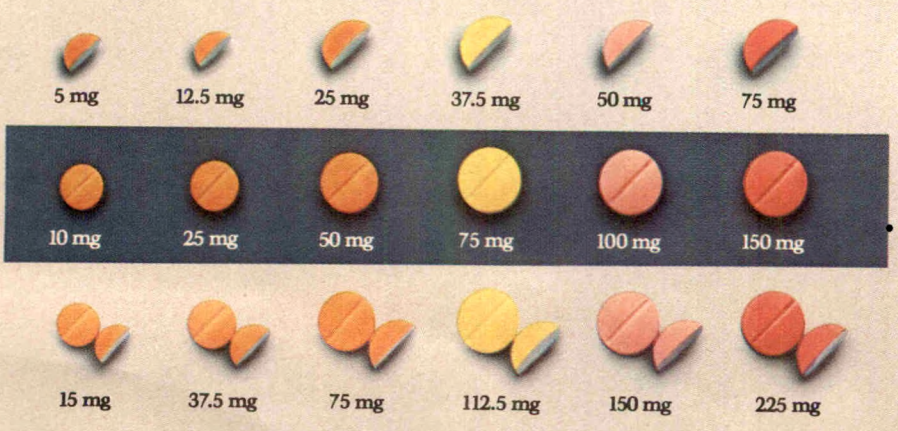
DUPONT

The easiest way
multiply and divide



Adds better dosage control

(+) Long term treatment of depression with amitriptyline requires careful dosage titration to meet the individual needs of each patient. Because all six strengths of Endep (amitriptyline HCl) are scored, you can easily change the dose by half-tablet steps, without waiting for the patient to fill a new prescription.



to add, subtract, amitriptyline...

Subtracts some of the drawbacks of antidepressant therapy

(−) Scored Endep (amitriptyline HCl) tablets may eliminate the need for a new prescription each time the dosage needs to be changed, saving time for you and money for your patient. And, by keeping the patient on the same strength tablet, there's less chance he'll become confused, as he might if changed to another size or color of amitriptyline.

Multiplies therapeutic options

(×) The pharmacokinetic properties of Endep (amitriptyline HCl) are such that its clinical effect is the same whether prescribed *t.i.d.* or as a single daily dose. Thus, once you have established the proper dosage, you can prescribe the entire dose *h.s.*, minimizing anticholinergic and sedative side effects and helping to assure patient compliance with this simple regimen.

By prescribing the higher strength tablets *h.s.*—secure in the knowledge that the dosage can be adjusted in half-tablet steps without resorting to a new prescription—your patient will economize further on a per milligram basis.

Endep®
amitriptyline HCl/Roche
The only amitriptyline scored for easy division

Endep
amitriptyline HCl/Roche

Before prescribing, please consult complete product information, a summary of which follows:

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

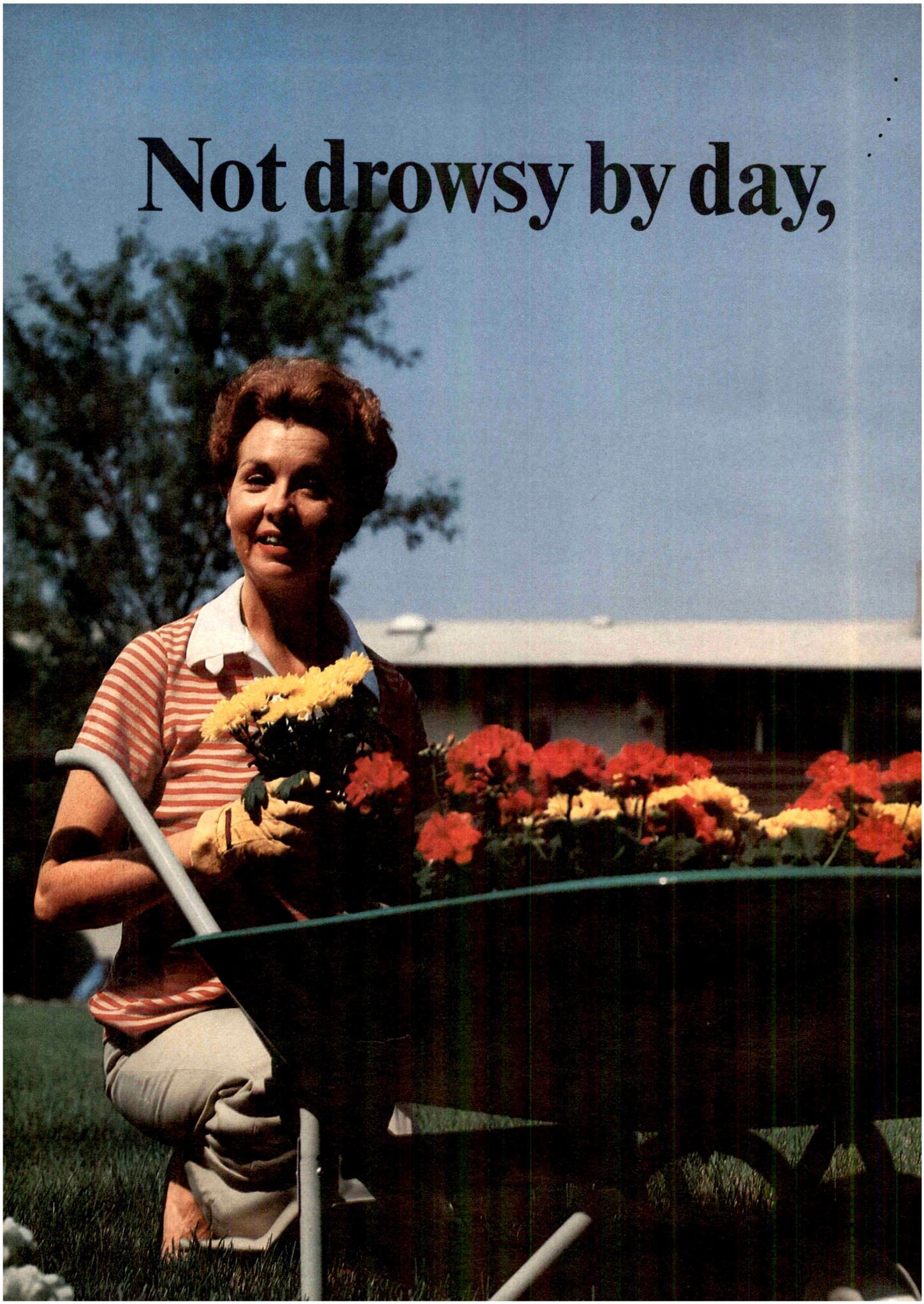
Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110


Not drowsy by day,

yet anxiety controlled

Troublesome drowsiness uncommon

Tranxene lets you manage anxiety in most patients without problem sedation. Any initial drowsiness is usually transient.

Calming without impairment

Patients not oversedated can function better on the job* or at home. By avoiding excessive sedation, Tranxene helps you calm patients without compromising their capacities.

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Tranxene®

(CLORAZEPATE DIPOTASSIUM)

C IV

4306CB

CAPSULES: 3.75, 7.5, 15 mg; TABLETS: 11.25, 22.5 mg.

*Of course patients should be cautioned against hazardous tasks requiring mental alertness. See overleaf for prescribing information.

8103278

Tranxene® (CLORAZEPATE DIPOTASSIUM) 4306CB

CAPSULES,
SINGLE DOSE TABLETS
Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

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now dual-action Triavil® 4-50

containing 4 mg perphenazine and 50 mg amitriptyline HCl

For even more dosage versatility in the treatment of marked agitation with depression



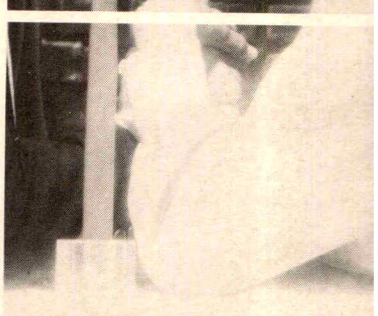
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to simplify therapy
five dosage strengths now available



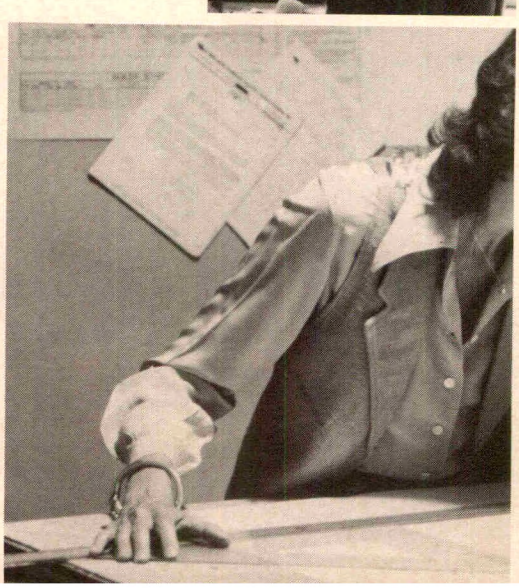
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to improve compliance
for initiating therapy
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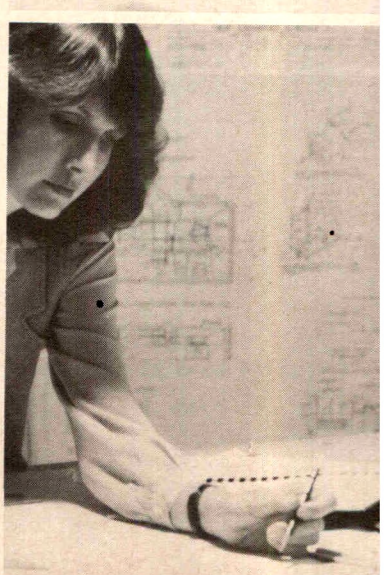


NEW unit-of-use package
bottles of 60 for 30 days of therapy, where appropriate









When TRIAVIL is part of the program, you may anticipate these therapeutic benefits for many patients:

Rapid antianxiety effect
The tranquilizer component can alleviate symptoms of anxiety and agitation within a few days. Hypnotic effects from the tranquilizer component appear to be minimal, particularly in patients permitted to remain active.

Highly effective antidepressant action
The antidepressant component can help relieve symptoms of depression such as poor concentration and feelings of hopelessness as well as early morning awakening; adequate relief of symptoms may take a few weeks or even longer.

...to aid the psychotherapeutic process
Patients often become more accessible and cooperative; symptomatic relief may enable the patient to function more effectively in his daily activities.

Treatment with TRIAVIL—a balanced view:
TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving. The drug may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

MSD
MERCK SHARP & DOHME

Please see following page for a brief summary of prescribing information.









For a wide range of patients with marked agitation and depression

dual-action Triavil®

containing perphenazine and amitriptyline HCl

now offers more dosage strengths than any other formulation containing a tranquilizer and an antidepressant

dual-action Triavil®

containing perphenazine and amitriptyline HCl

Available:
TRIAVIL® 2-25: Each tablet contains
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TRIAVIL® 2-10: Each tablet contains
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TRIAVIL® 4-50: Each tablet contains
4 mg perphenazine and 50 mg amitriptyline HCl.
TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

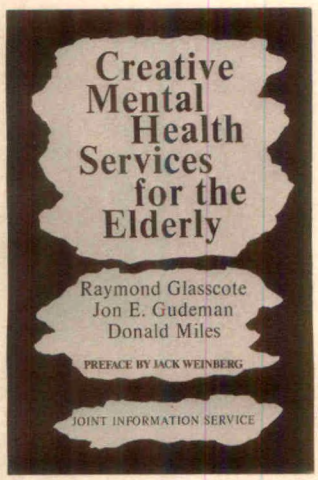
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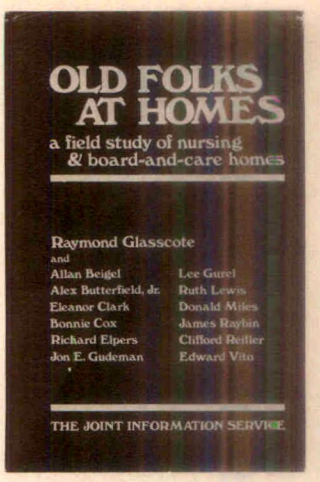
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Fluphenazine	High
Acetophenazine	Moderate
Trifluoperazine	High
Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2–6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170–171.

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Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.
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Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.
It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Psychiatry and the Fiscal Third Party

BY PAUL CHODOFF, M.D.

Third-party payment has profound effects on such areas of psychiatry as confidentiality; diagnosis; the therapeutic relationship; psychiatry's identification with the rest of medicine and the role of nonphysician mental health professionals; psychiatric education; and the availability, type, location, and quality control of treatment. As third-party payment becomes increasingly frequent, psychiatrists will have to come to terms with its good and bad effects in hospitals and community mental health centers as well as in private practice.

SYSTEMS FOR SUPPLYING psychiatric care can be defined and differentiated by the methods which they are funded (1). There are essentially three such methods: direct out-of-pocket payment by the recipient of care to the psychiatrist; payment in whole or in part by a third party, almost always a provider of health insurance otherwise not involved in the treatment; and public funds from federal, state, county, or city sources. Thus, psychiatric care in the Veterans Administration and the military is funded entirely by the federal government. State mental hospitals operate principally with state money but also receive some insurance and out-of-pocket payments. Health maintenance organizations are financed entirely through prepaid insurance, and the community mental health centers depend largely on public funds, although they may receive third-party and out-of-pocket payments.

The private practice of psychiatry today is a system whose payment method appears to be in a state of transition. Replacing direct out-of-pocket payment by the patient (or an interested party) to the psychiatrist is an increasing reliance on insurance funds. An essentially two-party transaction thus is being converted to one involving three parties.

The principal theme of this paper will be an examination of the effects on private practice of a third party operating as a payment agent between the recipient of care and the psychiatrist. Some comparisons will be made between two-party (out-of-pocket) and three-party practice and also between fee-for-service private practice on the one hand and the organized systems of psychiatric care on the other. Just as casting a stone into a still pond has a spreading ripple effect, so the interposition of the fiscal agent between the two directly involved participants has widespread and profound consequences for the psychiatric transaction. Third-party payment not only produces new conditions but also, and possibly even more importantly, makes unavoidable a confrontation with certain aspects of the practice of psychiatry that have not heretofore demanded attention in so urgent and persistent a manner. An analogy might be the increased complexity which results when diadic relationships become triadic in the course of infant-child development.

THE MEDICAL MODEL

One of the previously latent issues that the introduction of third-party payment has brought to the surface is the so-called medical model problem. The "medical model" is a term that is employed widely and generally rather loosely. It has been equated solely with organicity—one is not sick unless he or she has a demonstrable organic lesion. In a broader and more inclusive definition, "sickness" describes a form of deviant behavior with the following characteristics: it is negatively valued by society, it is involuntary—the individual displaying it is not responsible for his or her "sick" behavior—and it falls under the professional purview of physicians (2). Any individual satisfying these criteria assumes what has been called the "sick role" (3) and becomes a "patient." An organic ciol

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ogy for the behavior is often present but is not essential.

The definition of the medical model can provide material for an interesting academic debate with scholastic overtones, but the issue becomes of immense practical importance when the livelihoods of a whole profession are affected by attitudes about this abstract concept. In fact, I submit that possibly the most urgent question facing psychiatry as a profession today is the medical model question—to what extent are psychiatrists physicians and the people they deal with professionally, patients?

How are these questions about the medical model affected by the transition of practitioners from two- to three-party payment? The companies and government programs that provide the benefits for their subscribers, being in the health insurance business, must operate on the basis of some definition of illness even though the definition is likely to be covert rather than clearly formulated. In practice, the model they are familiar with works most easily with such diseases as pneumonia and appendicitis, conditions with medical diagnoses, more or less demonstrable pathology, and obvious degrees of functional disability. It is true that such criteria may be lacking in substantial portions of the caseloads of nonpsychiatric physicians; however, it is their absence in many psychiatric cases that particularly seems to give rise to confusion, uncertainty, and even suspicion in health insurance circles.

Therefore, the cost of long-term psychiatric care, both inpatient and outpatient, is not the only reason that most insurance companies balk at providing full benefits for the patients of psychiatrists. There is also a reluctance to do so because insurance people do not understand how and why certain psychiatric patients can be considered "sick." They complain that they "can't get a handle" on psychiatry for the purpose of planning, projecting, and actuarializing the claims they will be required to pay.

Difficulty in Diagnosis

One specific way in which these medical model uncertainties become manifest is in the transmission of *DSM-II* diagnoses to insurance carriers in connection with claims for psychiatric services. It is accepted that diagnosing illness is a medical function par excellence. Why, then, is there such a furor about submitting psychiatric diagnoses in connection with insurance claims? There are two reasons. First, and very important, is the confidentiality issue, which I will deal with later as one of the consequences of the change from the two- to the three-party payment system. However, I believe that a legitimate fear that confidentiality will be breached in a damaging way is not the only reason for psychiatric reluctance to make and transmit diagnoses. It must be acknowledged that some psychiatric patients, particularly but not exclusively those treated in office practices, simply do not lend themselves very well to the medical diagnostic process. In general, psychiatric patients seen in various settings include a very wide continuum of troubled human beings (4). At the unequivocally "medical" end of this spectrum are individuals with various forms of organic brain disease. Next are the major psychotic disorders, followed successively by the symptomatic neuroses and the personality disorders included and described in *DSM-II*. However, psychiatrists also treat some patients with more or less serious character deformations resulting in persistent maladaptive disturbances that do not conform specifically to the *DSM-II* categories (this is also true, although possibly to a lesser extent, of the broader and more inclusive *DSM-III*). Legitimate differences exist as to where along this continuum the division should be made, but it is clear that the patients at one end comprise a very different population from the "patients" at the other end and that the latter can be included under even an elastic definition of the medical model only with a good deal of Procrustean manipulation. Thus, even leaving out considerations of confidentiality, the difficulty in finding appropriate medical diagnoses for patients who fall at the nonmedical end of this spectrum accounts for some of the reluctance of psychiatrists to transmit diagnoses to insurance companies who are reimbursing their patients.

Psychiatrists' Reactions

Since, however, the diagnostic procedure is necessary for reimbursement, psychiatrists must conform or run the risk of losing their patients. They cannot avoid dealing with the ambiguous aspects of their profession's medical identification. Like most people faced with ambiguity, they may react with anxiety and defensiveness; thus, some of the resentment felt and expressed by psychiatrists toward the insurance carriers may contain elements of projection. I would even suggest that the emphasis on breaches of confidentiality as a barrier to transmission of information, when present to an exaggerated degree, may be a rationalization for the uneasiness of a psychiatrist confronted with the need to make a medical diagnosis in cases in which the medical diagnostic procedure is not particularly applicable. Practically speaking, when he or she is unwilling to make an accurate diagnosis because of considerations of confidentiality or is unable to do so for the reasons described above, a psychiatrist is likely to take refuge in writing bland diagnoses such as anxiety neurosis or depressive neurosis. This device satisfies formal requirements and is usually generally descriptive of patients' conditions. Such diagnoses are unlikely to have an adverse effect on the therapeutic relationship or to damage patients should they be revealed. However, in the long run, this practice can have only negative effects both on the status of psychiatry as a discipline with at least some scientific aspirations and on the attitudes of the insurance carriers, whose suspicions that they may be paying not for medical illnesses but for self-improvement or education will be encouraged. Furthermore, the delicate fabric of the psychotherapeutic relationship, which depends on the honesty and integrity of the therapist, may be damaged in

the atmosphere of collusion between the participants in making such diagnoses only to secure reimbursement.

The situation I am describing is by no means the rule in psychiatric practice but, rather, is one that applies particularly to office psychiatrists engaged primarily in psychotherapy and then only with a segment of their patients. Those psychiatrists who deal with a more clearly medical population may be concerned about breaches of confidentiality with reference to the insurance requirements, but they will usually have no serious difficulty in making diagnoses. Thus, this insurance requirement may be promoting a potential schism in the profession of psychiatry between the "physicians" and the "psychotherapists" since it is the latter whose medical identification is most threatened. It is also clear that these problems are not present in two-party out-of-pocket private practice where payment does not depend on the furnishing of a medical diagnosis to someone other than the participants in the therapeutic transaction. Furthermore, they are much less obvious in the salaried, organized systems for providing psychiatric care. In these—community mental health centers, the military, the VA, and state hospitals—the funding agency is much less likely to key payment to the demonstration of the existence of a distinctly medical illness. The model of social distress or societal disequilibrium may provide an entrée as acceptable to these funding agencies as a "medical" disability (1).

An interesting by-product of the influences I have described is the tendency of individual psychiatrists and of psychiatric organizations to replace a rather casual attitude toward organized medicine with a firmer reidentification and involvement with it. Clearly, when psychiatrists mend their medical fences and reassert their identities as physicians, they are partially motivated by their need to have the services they render included among the benefits dispensed by health insurance plans and future national health insurance programs. Furthermore, the tremendously increased involvement by psychiatric and psychoanalytic organizations in guild and economic issues undoubtedly stems from the same cause.

RELATIONSHIPS WITH OTHER MENTAL HEALTH PROFESSIONALS

Another important consequence of the introduction of insurance funding into psychiatric practice is the uncovering and accentuation of the competitive aspects of the relationship between some psychiatrists and members of other mental health professions (principally but not exclusively clinical psychologists). Like the medical model problem (to which it is certainly related), such competition has always been at least potentially present. Although there are already rumblings that the dispute about who is qualified to treat mental and emotional disorders is moving even into the hospital setting, the chief locus of concern about this issue has been among private practitioners of psychotherapy. Although the question of what kind of training—medical or other—qualifies one as a psychotherapist involves major unresolved substantive issues, there is little doubt that economic considerations also play a significant role (5).

The extension of health insurance coverage has considerably increased the pool of individuals now able to take advantage of the services of private practitioners for psychotherapeutic treatment and thus has created competitive tensions among these practitioners. In this competition, psychiatrists, by virtue of being physicians, are in the favored position to secure health insurance payments for their patients. As noted above, this advantage is motivating psychiatrists to emphasize that they are physicians. At the same time it is also motivating other mental health professionals to assert with some success their right to receive direct reimbursement for psychotherapy in spite of the fact that they have not been medically trained. They may in fact be theoretically uncomfortable with the medical model to which they have to pay at least lip service in order to come under health insurance aegis. In addition, the fact that nonphysicians contend that they are qualified to offer the same services as psychiatrists sharpens the debate over whether this form of treatment is medical and whether it should be funded through health insurance. Pandora's box may be opening here, since the future may bring similar pressures to receive insurance payments from psychotherapists with increasingly diverse (including nonprofessional) training backgrounds.

This issue may exert a divisive influence not only between psychiatrists and mental health professionals but within the psychiatric profession itself, since the strains I have described above apply almost exclusively to those psychiatrists who practice privately and independently and who have little collaborative contact with psychologists or social workers. Academic and institutionally based psychiatrists, on the other hand, must deal every day with colleagues in these and other mental health professions. Thus, they are much more strongly motivated than office practitioners to accommodate the latter group. Furthermore, since these institutional psychiatrists receive their income through salaries rather than in the essentially entrepreneurial manner of private practitioners, they are not so likely to experience the activities of mental health professionals as economically competitive.

HOSPITAL PRACTICE

Up to this point I have been dealing with third-party payment effects principally as they apply to outpatient, predominantly psychotherapeutic practices. However, there are also important consequences for psychiatric practice in hospitals. Since insurance programs pay more readily for inpatient than outpatient

medical services, there may be some tendency in psychiatry (although probably not so much as with other medical specialties) for patients to be admitted for workups and treatment regimes that might otherwise be handled on an outpatient basis. Also, day hospitals that offer an alternative to hospitalization are hampered in their efforts to compete with the latter by reason of less favorable insurance coverage. In general, however, the coverage available for inpatient units in general hospitals encourages acute care in such institutions and cuts down on the necessity for municipal or state mental hospitalization. This is not an insignificant factor in the marked reduction in the populations of such hospitals and can be considered to play a role in discouraging the development of chronicity.

The availability of insurance coverage also promotes the successful development of certain specialized psychiatric hospitals that provide acute and intermediate care essentially through the team approach. These hospitals, while often innovative in their program, are likely to emphasize services that will qualify for reimbursement. In accordance with the old adage that he who pays the piper calls the tune, it is becoming clear that the carriers are demonstrating an increasing power to control the kinds of services offered in institutions dependent for their survival on the income derived from insurance sources.

RECIPIENTS AND PROVIDERS AND TREATMENT PLANNING

The most clearly beneficial effect of widespread insurance coverage for psychiatric conditions is to open up the private practice option to a socioeconomic population for whom it previously would have been unavailable. Prospective patients are no longer dependent solely on charity or on publicly financed systems. Because in our culture private practice is highly valued and attracts effective and well-trained practitioners, there is at least theoretically a resultant overall improvement in the care available to a wider stratum of the population. This broadening of the socioeconomic mix of patients may also make it possible for certain theories of psychopathology and personality development to be less culturebound than they were, for instance, when Freud was basing his ideas on a rather narrow sample of upper-middle-class Viennese.

In areas with good insurance coverage for outpatient private practitioner services, there has been a considerable increase in the demand for them. It is not clear whether this indicates the existence of a previously unmet need or whether the ancillary payment method may also be encouraging a broadening of the concept of what sorts of personal distress are indications for psychotherapy in our society. At any rate, it has been estimated that there are 31.6 psychiatrists per 100,000 population in the Washington, D.C., area, with its excellent third-party program for federal employees, a figure equalled only by New York City (32.8 per 100,000) and in contrast to an average of less than 10 per 100,000 in the rest of the country (6). This is dramatic evidence of the efficacy of insurance programs in increasing the supply of psychiatrists (and also of other mental health professionals). In the Washington, D.C., area, where the Federal Employees Health Benefits program is a very important source of payment for most practitioners, an incidental effect has been a tendency for the fee psychotherapists receive to be standardized at the level for which the insurance carrier will make an 80% reimbursement. Consequently (and rather unexpectedly) it is possible for the incomes of neophyte therapists quickly to reach equality with those whose experience is considerably greater. This occurs, of course, only if the field does not become so overcrowded that entrants into practice find difficulty filling their schedules.

Besides increasing the supply of both practitioners and patients, the insurance money also influences the modalities, intensity, duration, and goals of treatment planning. Thus, the decision whether to offer long-term intensive or short-term return-to-function psychotherapy to a patient obviously depends on many factors but certainly includes as an important consideration the state of the prospective patient's finances. The knowledge that insurance help can be counted on frees the therapist to exercise his or her best clinical judgment about the treatment modality best suited to the case. On the other hand, the availability of funds has the potential for encouraging malutilization of both outpatient and inpatient treatment. Thus, a hospital stay may be terminated not solely for clinical reasons but because the insurance entitlement has run out. The somewhat fuzzy indications for certain psychotherapeutic modalities may be stretched because insurance coverage is available and because the therapist prefers these modalities. The same may be said of the proliferation of psychotherapy groups, sometimes without adequate patient selection or clinical indications. Again we note the contrast with two-party private practice where lack of out-of-pocket funds may limit treatment options but also may curtail such looseness. With the salaried systems for providing psychiatric care, financial incentives to prolong treatment do not exist. However, in these systems, particularly the HMOs, the funding method may have the opposite effect of encouraging unduly short-term and limited treatment rather than the more intensive but more expensive methods that may be indicated in some cases.

PSYCHIATRIC EDUCATION

As with other medical specialties, the trend toward vastly increased health insurance coverage among the general population has affected undergraduate and resident education in psychiatry because charity patients are no longer as available to students and residents for teaching purposes. Also, the assurance required by the insurance carriers that the "specialists"

whom they are paying for their services have proper credentials adds motivation for taking national Board examinations. Such third-party pressures, combined with the movement for continuing education, are also generating an interest in various credentialing mechanisms among subspecialties. For instance, the major psychoanalytic organizations have already moved toward specialty certification in order to establish a mechanism that would indicate which practitioners are qualified and thus eligible to have their patients receive benefits.

PEER REVIEW AND STANDARDS

Review and monitoring of the professional activities of their colleagues by peers within the specialties of medicine clearly seems to be an idea whose time has come. In psychiatry today we see a rich ferment that includes both concurrent and retrospective review of in-hospital and outpatient treatment practices. The American Psychiatric Association has moved far in this field; in fact, when questions are raised about including full coverage of psychiatric treatment under the aegis of insurance and national health insurance, APA presents its panoply of peer review activities with the claim that peer review in the hands of the profession itself will be effective not only in eliminating abuses but also in controlling costs.

Another important reason for the current interest in peer review, besides these economic pressures, is the public enthusiasm for and adaptation of the ethic of accountability. Accountability requires that whoever dispenses goods or services to the public must be able to assure the consumer that the product is what it is stated to be, that it is being offered in good faith, and that the consumer has recourse for complaints and questions. The burgeoning consumer movement has been adopted and encouraged by third-party insurance funders for their own purposes, which are to protect their subscribers from abuse, to keep their costs down so that they can successfully compete with other carriers, and to make projections and plans on the basis of reasonably consistent actuarial data. These aims have been accepted as legitimate by organized psychiatry, which has offered its cooperation by instituting peer review. Incidentally, the tendency for psychiatric peer review to be conducted under the aegis of medical societies rather than independently is additional evidence of the reidentification of psychiatry with the rest of medicine.

How effective psychiatrists will be in monitoring their own profession through peer review remains to be seen. Skepticism has been expressed (7) about whether it is possible for the members of any profession to forego their own guild and economic interests sufficiently to protect the public. The stakes are very high in this endeavor since failure to convince the public and the third parties that these efforts at self-control are effective is likely to lead to the introduction of extraprofessional monitoring with all the threat of loss of professional autonomy this would pose. I am inclined to question whether peer review is an effective cost control mechanism, but I believe that it can be a potent means of dealing with abuses and will patterns of malutilization of certain services (8).

I mentioned previously that insurance pressures generally are felt more strongly in private practice than in the organized, salaried systems. This is a matter of peer review. A mechanism to account for whether subscriber money is being spent properly and prudently appears to be of greater concern to insurance funders than to the funders for the military and the VA. In these systems the relationship between the source of funds and the providers of care is not nearly so direct as that between insurance carriers and private practitioners of psychiatry. Consequently, VA and military psychiatrists are not being influenced to establish peer review committees by these economic considerations, although they may do so for other reasons (9).

Quality control measures also become a source of tension between third-party payers and practitioners in the establishment of standards for various aspects of psychiatric treatment. In order to process claims and to project and control their operations in general, the carriers seek relatively simple, hard and fast criteria for such treatment concerns as length of stay in hospital, average duration of psychotherapeutic treatment for various diagnostic entities, the usual number of members in a psychotherapeutic group, the optimum number of electric shock treatments, etc. Specific answers to these and many other questions are sought so that they can be translated into guidelines to provide nonprofessional claims review personnel with an easy and efficient way of deciding whether a claim should be paid immediately or held back for further scrutiny.

The nature of the examples I have given will immediately arouse discomfort and frustration in psychiatrists, most of whom will agree that the diagnostic categories and treatment modalities of their profession lend themselves poorly to rigid rules. This conflict between insurance carrier needs for standards and the reluctance and inability of psychiatrists to supply them is responsible for some of the antagonism that exists between the two groups. It affords a sobering example of the power that their financial control gives to the carriers to shape and influence the kinds of services that can be offered to patients. It puts psychiatrists in a dilemma between the wish to operate free of such external and nonprofessional constraints and the knowledge that they cannot do this and also protect their patients' ability to receive reimbursement.

In the course of discussion about the reimbursement of certain treatments the question is sometimes raised by third parties (insurance or governmental) about evidence of their effectiveness. This confronts psychiatrists, in an embarrassing way, with the paucity of reliable outcome studies, particularly with regard to the psychotherapeutic modalities. Among the reasons for trying to remedy the present unsatisfactory state of re-

search in this area is the need to be able to lay before third-party carriers and national health insurance planners evidence that psychotherapy in the hands of competent therapists is a valid mode of treatment.

TREATMENT RELATIONSHIPS

That someone else is (as it were) peering at the treatment transaction, no matter how distant the vision or dark the glass, is bound to influence the relationship between the two participants. This is true whatever the diagnosis or the modality, but it is likely to be more troublesome and have potentially graver consequences for those forms of psychotherapy in which scrutiny of the relationship itself is a therapeutic instrument.

Confidentiality

Just as the wider availability of treatment relatively free of financial constraints is the most distinct advantage of insurance coverage, so its greatest disadvantage is the inevitable intrusion of the payer into the therapeutic relationship. Granted the legitimacy of the third party's requirements for certain information to process claims and to protect the financial interests of other subscribers, this need is still felt as a wrench. It interferes with the therapeutic benefit derived from the assurance to the patient that anything he or she says will be absolutely inviolate. There are bound to be disturbing questions about possible misuse of the information transmitted. Thus, psychiatrists will disgorge the information required to justify payment of claims with a degree of reluctance that varies according to the amount requested and the sensitivity of the information.

It is in this matter of confidentiality that one must acknowledge the inestimable advantage of two-party treatment, where, in an increasingly impersonal and bureaucratized culture, there still exists an oasis of assurance that one's secrets will be safe.

A sobering instance of the widespread consequences of the breaching of confidentiality in connection with insurance reimbursement occurred in the Washington, D.C., area when a new mental health report form, which required a great deal more information in regard to federal employee subscribers, was introduced by Blue Cross/Blue Shield. Especially since most psychotherapists felt that they had to reveal the form and their answers to its questions to their patients, a whole gamut of complicated reactions ensued, ranging from refusal of some patients to start treatment and disruption or serious interruption of treatment in a few instances to apparently neutral responses and even, occasionally, to effects that could be described as beneficial. The form generated a very marked negative reaction among the psychiatrists involved, most of whom indignantly regarded it as a kind of latterday plague; their efforts (plus the threat of legal intervention) led to the withdrawal of the form. Whether or not all the fears expressed were entirely realistic, the episode served to demonstrate that insurance carriers do deal differently with psychiatrists than other medical specialists. It also highlights the resentment among the psychiatric community at giving up its cherished autonomy and conforming to the requirements of a fiscal third party.

Allegiance

Although a psychiatrist cannot ensure a patient the absolute confidentiality that exists in two-party transactions, the psychiatrist whose patient accepts insurance reimbursement is unlikely to have serious internal conflicts about whose interests he or she is serving. The psychiatrist will certainly opt for the interests of the patient rather than those of the payer in any conflict between the two in which he or she has a choice. In private practice in general, whether two or three parties are involved, there is not likely to be the kind of conflict between the interests of the patient and the psychiatrist which may occur in salaried organized systems—for instance, in the military, where the importance of the mission may supersede the needs of the individual patient in treatment (1).

Transference-Countertransference Relationships

How does the presence of the third party affect those subtle and shifting currents in the relationship between the two participants that play so important a role in insight-oriented psychotherapy and psychoanalysis? Does it constitute an interference so grave as to hamper seriously the overall effectiveness of such treatment? As with other questions concerning psychotherapy, we do not have the benefit of satisfactory outcome studies but must rely on overall impressions, anecdotal material, and individual case reports. On some matters, there appears to be agreement. It seems clear that in some cases the limitation of the patient's own financial responsibility for treatment encourages acting out through missing appointments with a frequency that would be unlikely if his or her own pocketbook were more seriously affected. This phenomenon, incidentally, raises the question of whether rules about charging fees for missed hours should be altered when payment comes not exclusively from the patient but ultimately from other subscribers to an insurance plan. Some therapists hold that the situation is unchanged by the source of payment since their relationship is solely with the patient. Other therapists, however, are troubled and see the situation in terms of an ethical issue not present in two-party contracts. Also, as with the diagnostic practices previously described, it is possible that an atmosphere of collusion with the patient that is inimical to treatment might develop when such instances are frequent.

Although some influence on other transference-countertransference manifestations would be expected because of the third-party intrusion in such an important aspect of the therapeutic relationship as payment, the nature of this effect has not been explored thor-

oughly. There are reports about problems arising from insurance payment, such as the encouragement of a feeling of omnipotence in the patient who is getting treatment for little or no cost (9). Whether such problems are frequent is not clear; it is possible, of course, that they could be used productively as grist for the psychotherapeutic mill. Psychoanalysts who use the treatment model of the resolution of the transference neurosis through interpretation might be concerned that patient-therapist collaboration in dealing with the third party could interfere with analytic neutrality by emphasizing the real aspects of the analyst. Also, if the analyst adheres to the "sacrificial fee" theory that successful treatment depends on regular, somewhat painful disgorgements of money from the patient, problems might be anticipated in cases where insurance has made payment easier (10). In response to these caveats, it can be stated only that no firm evidence has been adduced which suggests that treatment results have been substantially downgraded by these potential obstacles to transference-countertransference maturation (11).

DISCUSSION

The introduction of a third-party funding agent into the treatment transaction between patient and psychiatrist has been associated with important and unexpected consequences for many aspects of the practice of psychiatry. Some of these consequences are unequivocally positive, notably the greater availability of the private practice option and the freeing of treatment planning from financial constraints. There have also been unequivocally unfavorable effects—the breaching of confidentiality in the treatment relationship, a tendency toward mandating types of treatment by funding agents who are primarily concerned with cost control, and an increased amount of record-keeping and bureaucratic wrangling. Other consequences of the third-party influence are less easy to characterize; their effect varies depending on point of view or usage.

The third-party impact has also brought to the surface with fresh urgency certain questions about the extent to which psychiatrists can properly wear the mantle of physicians. This has created tension between psychiatric practitioners and other mental health professionals. It has also augmented a potential for conflict between psychiatrists whose chief or only treatment modality is psychotherapy and those who have more varied practices and between psychiatrists who earn their living through fees for service and those whose income is derived from salaries within organized systems of psychiatric care.

Because insurance payment for psychiatric services has had such mixed effects, it should not be surprising that the reaction of those psychiatrists whose practices have been heavily influenced by insurance payment has likewise been a mixture of satisfaction, irritation, and uneasiness. The relative freedom from financial constraints in treating patients has been a ... the threat to professional autonomy from ... agencies uninvolved in treatment who appear to view psychiatrists with suspicion and to treat them differently from other physicians.

However, no matter whether the effects ... bad, or indifferent and no matter what the ... of reluctance and acceptance with which they are greeted, there seems to be little question that payment for psychiatric services through a third-party intermediary is bound to play an increasingly ... role. This is true not only for private practice as ... been discussed here, but also for some of the ... systems for supplying psychiatric care, such as community mental health centers and state hospitals. It is probable that sooner or later all psychiatric services in the private sector will be paid for largely through some combination of commercial insurance and national health insurance programs. Thus, unless we take the somewhat idiosyncratic course of withdrawing from such a system entirely in order to preserve two-party values, private practitioners of psychiatry will have to come to terms with the advantages and disadvantages, problems, questions, ambiguities, and ironies resulting from this change in the payment method. The ... of organized psychiatry has reacted to insurance pressures with endless conferences, plans, proposals, ... negotiations, out of which has come the establishment of peer review mechanisms. These responses have thus far been largely on an ad hoc basis. I believe that the time for our profession to take an objective look at the many implications of the changes resulting from the new payment methods so that it can formulate policies that will be reasoned and comprehensive.

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On Editing the *Journal*: *Ave Atque Vale*

BY FRANCIS J. BRACELAND, M.D.

Dr. Braceland discusses his 13-year tenure (1965–1978) as Editor of the American Journal of Psychiatry, *noting that he took office during a period of social turmoil that in many ways resembled the decade of the* Journal*'s founding 134 years ago. He describes the advantages and disadvantages of the peer review system he introduced to the* Journal *and states that this approach is essential in a time of ultra-specialization. He reviews the crafts of editing and writing for medical journals, both of which are brightened by clarity and brevity. As to the future of medical journals, he considers reports of their demise premature, while acknowledging the problems they face. Dr. Braceland briefly considered a "rocking chair" approach to his retirement from the Editorship, but the Editor Emeritus will instead continue to provide whatever assistance he can to his colleagues.*

I HAVE BEEN ASKED by the Program Committee to discuss my involvement with the *American Journal of Psychiatry*, which I have had the honor of editing for the past 13 years. I was asked also, if possible, to predict its future, as well as the future of medical journals in general. To carry out this mission, I must first ask your indulgence as I glance back a bit, for when I became the *Journal*'s 9th editor it was already 121 years old and had remained alive through times of war, famine, and pestilence. As I discuss this broad span of years, you must realize that unlike the scholarly mandarin I cannot paint them delicately with an exquisite Chinese brush; my canvas must be broad and my brush wide.

Like many things and people later destined to become great, the *Journal* had a modest beginning. Its birthplace was the Utica Lunatic Asylum, the time was July 1844, and its given name was *The American Journal of Insanity*. None of this sounds very promising, but in its favor was the fact that its sire was Amariah Brigham, a gracious, capable physician, a scholar, a teacher, and a dedicated man.

The decade in which he founded the *Journal* was later to become known as the "Fabulous Forties." The whole nation was awakening and astir; the crude conditions of pioneer life were lessening on the Eastern Seaboard, and there was leisure enough to think of plans for human betterment. Schools, academies, hospitals, and libraries were being opened, and several hospitals for the insane were under construction.

We are justified in looking at this period, for neither institutions nor publications can exist in a vacuum; all are influenced by the social, political, and economic currents that swell around them. The period is best described by Meade Minnigerode, and excerpts from his book, *The Fabulous Forties 1840–1850* (1), give us some idea of the incredible happenings in those times. Speaking of the decade in general Minnigerode said it was

> an unfinished, unstabilized company taking part in a national pageant which there had been no time to rehearse. . . . the apotheosis of banality. . . . A time when females were treated with utmost courtesy and very slight consideration. An indecent dance, the polka, was coming in to complete the demoralization of the younger set. Tight lacing was being blamed as a cause of insanity. . . .

An unbelievably large number of people were enthusiastically awaiting the end of the world on April 23, 1843, a date arrived at by the biblical computations of an itinerant preacher named Miller. Millerites were filling the mental hospitals on the East Coast and ascension robes were selling like hotcakes. Brigham later said that numerous suicides were caused by Millerism and that thousands had their health impaired to such a degree as to unfit them for life in the future.

This was the milieu in which the *Journal* first saw the light of day. Brigham had been drawn to Utica by the enthusiastic directors of the institution then under construction. They assured him it would be an institution second to none, and this appealed to him. A first-class alienist who had been the third superintendent of the Hartford Retreat, he was also a prodigious worker, and he sought every opportunity to educate the public in the intricacies of mental disorders, welcoming interested individuals and teaching those who were well disposed. His restless energy soon took a more important turn; he decided to found a journal.

On May 18, 1844, Brigham wrote to Pliny Earle, Physician to New York's Bloomingdale Asylum, as follows: "I am about starting an *American Journal of Insanity*, quarterly, Octavo, ninety-six pages edited by officers of the Asylum" (2). Two months later the

Revised version of a special lecture presented at the 131st annual meeting of the American Psychiatric Association, Atlanta, Ga., May 8–12, 1978.

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Journal appeared, the first publication of its kind in the English language. Most of the articles were written by Brigham himself. In order to make the publication widely available the price was kept at $1.00 per annum. It was printed and paid for by Brigham alone, and he continued to be its main source of articles. It is of historical interest that in the same year three other psychiatric journals were launched—one English, one German, and one French—and all three are in some form still extant.

The new *Journal* served to keep the few alienists, who lived in widely scattered areas, in touch with one another at a time when the field of psychiatry was practically a wilderness. There was a need for communication that the *Journal* fulfilled. Also, it was undoubtedly a factor in the formation of the Association of Medical Superintendents of American Institutions for the Insane in October 1844, four months after the *Journal* was founded.

The *Journal* proved to be a prodigious task, and Brigham, never robust, was often temporarily disabled by illness. His letters referred frequently to his health, and in 1846 he wrote and asked Pliny Earle to take over, noting, "I am getting old and averse to labor and cannot take charge of the *Journal* another year." At that time he was 48 years old. He carried on, however, until September 8, 1949, when he died at the age of 51 of an attack of dysentery.

Although it is tempting to trace the *Journal*'s further history, I must endeavor to be brief. I should, however, mention the fourth Editor, G. Alden Blumer, who served from 1886 to 1894. It was his unpleasant duty to dispose of the *Journal*, which was still the property of the Utica State Institution and was coveted by the New York State politicians. Fortunately for all, the American Medico-Psychological Association (the name the lineal descendants of our founding fathers had taken) bought the *Journal* for $944.50. Dr. Richard Dewey became Editor and in 1894 the *Journal* was moved to Chicago.

The *Journal* had several additional Editors, all of them interesting and worthy of note, but we have time only to mention one of them, Clarence B. Farrar, the 8th editor. A distinguished, cultured gentleman, Dr. Farrar had been Editor of the *Journal* for 33 years and had run the *Journal* from Toronto when, in 1964, the Council of the American Psychiatric Association decided to consolidate all of its offices and centralize its operations in Washington. Dr. Farrar retired in July 1965.

Dr. Farrar had been Editor over an incredibly important period in history. It covered the Great Depression, pre-war build-up, World War II, and all of the political dislocations of that holocaust. His tenure also saw the beginnings of the greatest recorded advances in psychiatric therapy, including the start of the modern physical therapies and the appearance of the new psychopharmacologic agents. His was a hard act to follow.

A search committee was formed under the chairmanship of Howard P. Rome and the hunt [illegible] Editor was on. There were few applicants [illegible] present incumbent was invited to undertake the task. He accepted even though he knew that the Editor's job was not the best way to make friends. He [illegible] that if one were ever to find a popular editor he would be neither sitting nor standing—he would [illegible] down surrounded with flowers.

The new Editor met with the Council and [illegible] carte blanche in all editorial matters and [illegible] only that the *Journal* was the organization's official organ and that all aspects of the discipline merited consideration. Modest space was found for the *Journal* [illegible] in the Washington headquarters, and by a great stroke of good fortune, Ms. Evelyn Myers, who had editorial experience at the National Institute of Mental Health and elsewhere, was made Managing Editor. She proved to be a "gem of purest ray serene." Nearly I began to worry about the situation facing us. We were not to become "official" until July 1, 1965, and were expected to get out a July issue! This was [illegible] for neophytes, but, fortunately, neither of us realized the enormity of the task before us.

I will pass over quickly the unpleasant things that had to be faced first, namely, returning a number of previously accepted manuscripts that were unacceptable and notifying the distinguished Editorial Board, who had served faithfully for many years, that a new Board would be appointed.

In some fashion not yet understood, the July issue appeared on time. Dr. Farrar wrote me [illegible] hand a note that is my proudest possession and he said he did not believe it could be done. However it was; it looked good and it was on time.

The early issues, without our quite realizing it, gave evidence that the *Journal* was indeed under new management and was "open." In the first and second issues there were articles on psychoanalysis, a subject that had not appeared often in earlier issues. There was also a disquisition by Mike Gorman in which he gave everybody hell all around in an article entitled "Psychiatry and the Public Policy." One sentence, addressed to all psychiatrists, gives an idea of the paper's general tenor: "You can no longer hide in the isolation comfort of your private offices appropriately furnished with an overstuffed couch and a picture of Freud visiting Worcester in 1909."

If there was any doubt that the *Journal* was under new direction, it was now dispelled. Gorman [illegible] repeated the observation of the Angel Gabriel in Connolly's *Green Pastures*: "Everything is all down is coming loose." He was right, for we began our term of office in a period roughly comparable to the Fabulous Forties. The nation was again astir: a [illegible] war was going on, drug use was widespread, "flower children" were running away from home, and college campuses were in turmoil.

Young folks let it be known they were going to take over a tired old world and show those over 30 how to eliminate poverty, racism, prejudice and ev[illegible]

in general. A small irresponsible segment took over universities, presented their nonnegotiable demands, occupied the dean's office, smoked his cigars, Xeroxed his papers, and wrote home for money on his stationery. The virus of discontent spread to medical schools, and Arnold Mandell, a first-class teacher, later wrote,

> Increasingly, bright students, with good academic records, are refusing to accept the challenge of describing character in terms of the dynamics of personality; they tell me that such balanced, lyrical efforts are the masturbatory activity of the passive during this time of social change. (3, p. 529)

The delusional systems of this period were not as tightly reasoned as the Millerism of the 1840s, but they were reinforced in some cases by hallucinogenic drugs, and it all seemed like a bit of a throwback to the time when the *Journal* was born.

There were editorials by two distinguished colleagues in our first issue. Dana Farnsworth wrote about unrest in our colleges. Francis Gerty wrote of the Silver Pitcher, a token of office given originally by a grateful Association to Dr. Dewey and later by his daughter to Dr. Gerty to be passed from Editor to Editor. In the same pages, I wrote an appreciation of the retiring Editorial Board and solicited help from all and sundry in my new job as Editor. Editorials became scarcer as the years went by, not because the Editor was modest or mute but because the *Journal* was soon inundated with manuscripts. There is an old saying that when you are up to your nether end in alligators, it's not the time nor the place to teach the children to swim. Well, we were up to there in manuscripts, and there was no space to spare for the Editor's musings.

BEING PAID WHILE LEARNING

What have we learned through the years? Too much to detail here, and frequently we learned through trial and error. Early on we opted for the referee system, i.e., sending our manuscripts out for peer review. We felt that this was the fairest and the wisest course and we firmly believe in it now. In this procedure, we joined with many other specialty journals, and we agree wholeheartedly with Franz J. Ingelfinger, former Editor of the *New England Journal of Medicine* and the most outstanding contemporary American medical editor, who said,

> Editors who depend on referees to differentiate that which is publishable from that which is not, some would argue are a pusilanimous lot. If they do print an article which turns sour the mishap can be blamed on anonymous reviewers. Editors of course reject this mean spirited charge. But there is no doubt that the average editor, confronted by the products of today's rarified research, lacks the confidence to go it alone. He needs the help of the ultraspecialists to judge the ultraspecial articles so that his journal will print the best of what is submitted and will disseminate material that has the imprimatur of authority. (4, p. 686)

We are fully aware of the system's defects, which include some inadequate reviews, bias on the part of an occasional referee, and unrealistic expectations on the part of the Editor. The costs, time, and effort involved in this system are enormous. Also, there is the possibility of violation of confidence and even the occasional suppression of a novel advance. One study showed that concurrence between two referees shown the same paper was only moderately better than chance, but as yet we know of no better way to operate. On occasion, we have sent the same paper to three, four, or more reviewers, particularly if the subject is controversial. In some cases, when these various opinions from experts have led to a rejection, we later see the paper in another journal.

Unfortunately, there are times when referees fail to view the manuscript fairly or objectively. Some academic groups hold tightly to certain ideas or methods and believe that anything written outside of their boundaries is ipso facto wrong. Occasionally authors ask us not to send their paper to a certain well-known group. This puts the editor in a bit of a quandary, particularly if this group contains a number of the known experts in the subject. Not possessed of the wisdom of Solomon, the Editor searches his roster for the wisest person available to get his advice; he often discusses such problems with members of the Editorial Board.

There are many other problems with the referee system. Some referees hold papers for an unconscionably long time, requiring numerous communications from the editorial office, while we attempt to sooth the justifiably ruffled feathers of the author. Some prospective referees never do answer us. However, as far as we can determine, the referee system in an operation as large as ours is "the only game in town."

Occasionally there have been objections from different groups of our readers about our accepting drug advertisements. Brush fires on the subject are lighted in various sections of the country at intervals. This same problem plagues other journals. Let us turn to Ingelfinger, for we are again in complete agreement with him. After asking what would happen if the *New England Journal* gave up drug advertising, he appended another question: "Would such a solitary, perhaps idealistic, gesture improve either the quality of patient care or the character of the *Journal*?" He answers: "The intensity of the 'no' I hear exceeds that of a sonic boom." He elaborated on his statement:

> Without pharmaceutical advertising, its [the *NEJM*] subscription price would increase about $25 per year. The suggestion that other advertising be sought lacks substance, for enterprises selling to a general market see no reason for placing advertisements in a medium read by only one restricted segment of that market. Similarly fanciful is the cry, "Price does not matter—doctors can pay!" Of course they can, but will they? . . . In spite of its

exceptional qualities, the *Journal* would be threatened by an ominous prognosis. Circulation would drop, and the subscription price would correspondingly rise even more. In other words, the *Journal* might well achieve a moral but pyrrhic victory, for it would be as pure and free of taint as a corpse. (5, p. 529)

We read also in that same dissertation (p. 528) that "Several journals here and abroad have been threatened with loss of pharmaceutical advertising unless they were willing to interdigitate text with advertising pages. Such pressure originating, I would hope," he said, from the "unconscionable competition of advertising agencies rather than the pharmaceutical houses themselves."

Our experience with the staffs of various of our advertisers has been felicitious. We have told them what we would publish and what we would not publish. An ad hoc in-house committee passes on the appropriateness of all ads, and three "outside" psychiatrists are used as consultants when questions arise. This procedure has worked out to our satisfaction. We operate on the principle that our colleagues are mature, intelligent, professional people, and we know that simply because they read an ad does not mean they will start dispensing the substance to all and sundry. We have too much respect for them to think that.

THE CRAFT OF EDITING

Richard M. Hewitt (6), distinguished former editor of the *Mayo Clinic Proceedings*, divided editors into professionals, those who are full-time and of long experience, and amateurs, whom he described as individuals who are part-time, expert in some branch of medicine, and likely to know the authors who are writing in the field. The editor also should know what has been done and what needs to be done so that he can solicit papers in areas that have not received sufficient coverage. Few medical editors are born to the purple; most of us come in the plebian entrance, the back door if you will.

The editor must also possess dogged independence. An editor of a journal owned by a professional society could be in an awkward position. If the editor is complaisant, the journal might become little more than a house organ reflecting the views and wishes of a group or a faction of it. Such a journal might well serve these groups, but it would not be a good scholarly publication and would eventually lose ground and disappear. Fortunately, those of us on the *Journal* staff have never encountered interference from any source during our tenure of office.

Medical journals in general are said to be in poor health at present, and the future of a number of them is in doubt. There is evidence of Cheyne-Stokes breathing in a few. Others, while still alive, are stodgy, dull, and a bit pedantic. Actually, the function of a journal is to give information to its readers, "some of it new, some of it old, some rearranged, some [illegible] some publishable because of their logic, [illegible] and departure from the commonplace" [illegible]

As is the case with many other journ[illegible] number of papers we receive are much too [illegible] lix, and discursive. Some are more peri[illegible] publishable. Some are larded with unnece[illegible] it is not necessary for the readers to know [illegible] pig's temperature at 4 a.m. on a given Thu[illegible] see every detail presented and re-presente[illegible] tables. The question is what were the result[illegible] bearing do they have on the purpose of the [illegible] regularly return papers and ask authors to [illegible] and eliminate some of the myriad of tables [illegible] This is not done in pique—there just is [illegible] *Journal* space to say things two of three [illegible] Theodore Fox (8), former editor of *The J*[illegible] said,

> The editor's real task is to make every w[illegible] to do this he must criticize, revise and abo[illegible] This is not the best way to make friends but [illegible] way for the editor to serve both his authors [illegible] ers.

The fact that psychiatry arrived late a[illegible] specialty (although it treats one of the olde[illegible] known to man) sometimes presents a spec[illegible] for the editor hesitates to reject a paper t[illegible] bit far out lest it really have some therap[illegible] bility. The referees are often ambivalent [illegible] sponsibility rests squarely on him. Une[illegible] head that wears the editorial crown! Dec[illegible] the paper and add to his backlog, or reje[illegible] chance miss something that might bring bra[illegible] the future? Then, too, he must remember [illegible] the author's brain child, his labor of love. [illegible] jor responsibility of the editor is to his [illegible] must be the thumbs up or thumbs down of [illegible] or more gently, the binding or loosing of [illegible]

The editor oftimes must serve not only [illegible] a mediator but also as a translator. When [illegible] referee writes as his opinion that "this p[illegible] unpleasant odor and could do with some[illegible] ener," the editor's task is to avoid decapit[illegible] the [illegible] pectant author by softening the harsh [illegible] writes to the author that, "sadly, this pape[illegible] for us. . . ." Ingelfinger calls this "exce[illegible] around the rosy" (9). "Although your [illegible] pressed me with its many admirable featur[illegible] ments of the readers are such as to make [illegible] for us to publish it." The editor can give [illegible] sons for the rejection but must be careful [illegible] courage a writer who should be encourag[illegible] ue and refine his work or encourage one [illegible] not.

Any decision by an editor and his st[illegible] volves numerous considerations. First, [illegible] are read by many individuals outside of [illegible] fession. Sometimes a paragraph of an arti[illegible] out, amplified, and disseminated far and [illegible]

news media. Some statements that are harmless on the surface can be completely misunderstood and turn up in a Congressional hearing considering changes in medical practice. Others may be used as fuel in the ever-increasing number of malpractice suits. The attentive reader of the daily press will encounter many denigratory comments about physicians that are taken from articles in which the doctors have found fault with themselves and their colleagues. Such articles may even be the basis of legislative actions. Medicine is one of the few professions that pillories itself mercilessly in print and in public, and the complainer may later read his own words quoted back to him in a lawsuit. He may have tossed off some frilly *bon mots* in a moment of enthusiasm and years later find these ill-conceived utterances prominently displayed in public print and enjoying a vast circulation. Medicine today is the stuff of the news media—miracle cures, the carcinogenic potential of many everyday substances, and, especially, the untold wealth of physicians are all grist for the media mill, and the editor must regularly recall these facts to writers.

There are a number of situations that raise the editor's hackles—the most annoying is to find that an article he published had been published elsewhere, either in large part or in toto. Then there are some individuals who have one idea or one particular approach to a subject and they "milk it" for all it's worth—and sometimes more. It might be good for four or five papers on the same study to be distributed to journals at home and abroad. Personally, I do not mind being sent a reprint of an article that I turned down which was published elsewhere. In that case, I am happy for the author.

All editors receive a number of preliminary reports accompanied by an urgent letter suggesting immediate publication to establish priority of an idea. Caution flags should go up and sound advice should be solicited in these cases. One fears publishing misleading information that may have to be corrected later. The situation is especially difficult when an author's presentation at a convention has been given widespread publicity—the editor then gets the paper and a mass of Xeroxed newspaper articles showing how this new discovery will do some miraculous thing. The half-life of some of these well-publicized discoveries is very short, and the editor must be concerned about misleading his readership. When we attempt to cool the author's ardor we sometimes only convince him that we are morons who stand stuffily in the way of great discoveries.

As we began editing the *American Journal of Psychiatry* in 1965, the Association had 13,800 members: today there are 25,000. More of our colleagues are writing than ever before, and for a variety of reasons—building up a bibliography for promotion purposes, acquiring continuing education credit, and, perhaps most often, because they have something important to share with their colleagues. Some authors see the editor and his staff as helpers; others are sure they are obstructionists in the pipeline of true knowledge. The editor, kinder people say, is a checkpoint the writer has to pass—a frontier past which he must make his way before he goes out into the literary world. Some feel simply that the editor's parents were not properly married.

Sir Theodore Fox finds worst of all are those editors to whom rules of grammar are one of the fixed points in an otherwise unstable universe and who are emotionally affronted if an author breaks a rule. The editor, he says, is mostly right but sometimes wrong. "Nevertheless, I stick firmly to my opinion that the quiet subliminal below stairs labour of editorial staffs does a great deal to improve communication among doctors by making the average article clearer and less unreadable" (8, p. 10). I would add to that, "by detecting bloopers that the referee and the editor might have missed to someone's embarrassment."

This Editor agrees that the staff's work is invaluable, and they manage to keep many barbarities of language out of the *Journal*. It is true that some authors will not allow their pristine prose to be tampered with no matter what the *Journal* rules might be, but this is infrequent. We deal with such instances by carefully going over the segment in question again; if we are right we send the author a Xeroxed copy of our authority for our action; if we are wrong we admit it and make amends.

WRITING FOR MEDICAL JOURNALS

No paper by an editor would seem to him to be complete without some comment about writing, even though his own productions might not be on the level of the King James version. Writing is said to be at least a craft and at best an art. All can agree that writing should be beautiful if possible. It is said that it needs taking only a little more trouble in order to make it pleasing to writer and reader. Pickering observed that "since we have each but a brief spell on earth, why not make that experience enjoyable?" Unfortunately, however, we all sometimes feel like Gustave Flaubert, whose writing irritated him. "I am like a violinist whose ear is true," he said, "but whose fingers refuse to reproduce the sounds he hears within."

Critics with literary sensibilities often comment wittily about the outrageous style of some such scientific writing. Scientists, they say, use jargon because they are obsessed with the desire to show that their work really does belong within a reputable context. Hayakawa commented that some doctors seem to hold to a tradition that goes back to the Chou Dynasty in China and to ancient Greece—the assumption that you can never be respected as a man of learning if everyone understands what you say. In past ages, intellectuals talked to each other in Latin, Sanskrit, or Classical Chinese, always making sure these languages were no longer spoken. The dead languages performed the extremely useful function of keeping the peasants in awe-

struck reverence before mysteries they could not hope to understand.

"We no longer encounter Sanskrit, but we do occasionally encounter jabberwocky. If one wants to try to make his paper read well, let alone glow, one does not have to perspire over it; one simply says what he means in simple declarative sentences. It is, however, desirable to get away from the "plop plop" style we so frequently encounter in medical writing. Some writers take a simple statement, choke it to death with adjectives, put as much as they can in the passive voice, and then embalm it further in polysyllables, making it more un-understandable.

The editor of a medical journal has some idea of what his readers want, and he can even try to find out for certain in readership surveys and by careful attention to his correspondence from readers, but even these facts must not overwhelm him. He must proceed carefully, for the specialties do not stand still, and it is the editor's job to recognize the advances. He must therefore present the material in a manner that will appeal to as many of his readers as possible. It was Alan Gregg who called to our attention the fact that easy writing was generally damn hard reading. The editor, therefore, cannot be overwhelmed by the name of the author: his reputation may be great, but we are publishing his work, not his biography.

The title and first paragraph of an article are important, as is the blessed précis. Doctors will not be enticed by an article whose title and early habiliments are dull, for there is too much reading material available and too little time to read it. Also, titles must lend themselves to proper categorization if they are to appear in the various medical index journals. The golden rule is to write, rewrite, rewrite again, and polish. Plato is said to have written the first paragraph of *The Republic* 12 times, and few of us are of the calibre of Plato.

We of the *AJP* have only occasionally encountered difficulties with titles. Other journals are not so fortunate, and we may be excused for appropriating the report of one instance. Robert A. Day (10), the outstanding Managing Editor of the publications of the American Society for Microbiology, wrote on the necessity for care in the syntax of titles. Most errors in titles, he pointed out, are due to faulty word order. One article he quoted as an example was entitled "Preliminary Canine and Clinical Evaluation of a New Anti-Tumor Agent, Streptovitacin." Day observed that "When that dog gets through evaluating Streptovitacin I've got some work I'd like him to look over." Incidentally, he said, the abstract of that article revealed that only male puppies were used. He thought perhaps a better title might have been "Evaluation of Streptovitacin in Sons of Bitches."

Just one or two further comments about doctors writing before I desist. William Bean warns us in talking about patients and in writing about them we should "avoid the good old zoological labels, 'male' and 'female,' . . . It is just as easy to say man or woman, girl or boy. . . . We don't refer to our children as [illegible] year-old white males, we call them little boys [illegible] devils). Few of us would introduce our mother [illegible] Maggie from Dubuque as a charming fifty [illegible] seventy-year-old white female" (11, p. [illegible]). Dr. Bean goes a bit further than I would when he states, "A patient may properly be on a bed or on a bed [illegible] but not on digitalis or on a high-fat diet [illegible] into the soup."

There is a barbarity that distresses me greatly [illegible] curs when physicians describe "cases" (to which [illegible] so object), saying "The patient *presents*. [illegible] sents"—that's what female baboons do in those [illegible] pictures we see of simian colonies in Africa.

I will spare you further examples of execrable [illegible] language, but I would like to note that there [illegible] papers which have very little to say and yet say it [illegible] Sometimes writers object to our returning [illegible] for "rewriting, tightening, and pruning." However, once an article is printed, it remains forever [illegible] on paper, and in the future it might not be [illegible] encounter early reminders of one's handiwork [illegible] probably should not have been written in the first place.

Brevity is the elixir that makes editors' lives pleasant. Clearly, briskly writing is a joy to read. [illegible] no courses and no "how-to-do-it" books [illegible] writer. It has to be done by ordinary folks [illegible] ing and rewriting and plugging away, practicing [illegible] criticism, striking out unnecessary words [illegible] writing it over again. Remember, too, that [illegible] nals are painfully short of space, but one must be careful of "overabbreviating." "S.O.B." [illegible] for "shortness of breath," but it also has [illegible] notations.

If you will pardon these professional observations, I will tell you modestly that I learned most [illegible] learned editing. Our own *Journal* is less particular about grammar than it is in asking that the author be clear in the message he intends to get across. The copy editors can put papers in the Queen's English, but they cannot tell what a man is thinking.

READING—HOW DOES ONE GET THE TIME?

In Boswell's *Johnson* it is written that the greatest part of a writer's life is spent in reading. [illegible] turned over half a library to write one book. The best way for a modern medical writer undoubtedly [illegible] familiarize himself with the thoughts and words of the finest members of the human race down through the ages. This can be done by getting into the habit of reading briefly every day in some field, even in the face of the overwhelming burden of study, and by a profound determination to attain excellence.

We as physicians are all awash in a sea of [illegible] literature. How can we handle this and make our reading effective? In our time there will always be too much to read; we must read selectively, wisely, and

despair not. Richard Orr (12) states that while there is no magic way, one secret is to follow several rules:

1. Speed—the ability to read very quickly is common among practitioners who are on top of the literature rather than under it.
2. Selection—sorting out printed materials that arrive into three piles: 1) requiring only a quick glance, 2) deserving of at least 5 or 10 minutes of rapid scanning, and 3) meant for more extensive study and reading only the parts that promise maximal return (read the summary first).
3. Optimum use of time—everyone of us has a few moments of free time during the day.

Felix Marti-Ibanez was a master at using these moments—he called himself "a ragpicker of time," and he read and wrote a prodigious amount. However, there is no habit which one can more easily take up or more easily put aside as other duties press upon one.

THE FUTURE

What of the future of journals like our own, which are inundated with manuscripts? Some feel that they have little future, for doctors will soon prefer newer techniques. Sir Theodore Fox believes that most of the scientific periodicals will disappear and be replaced by computerized material that could be made available through great centers which the seeker could tap at will. Morris Fishbein, an excellent editor, thought that we are in an evolutionary period and still have time before the computers take over. Actually Sir Theodore's idea is that there would be "recorder journals," "newspaper journals," and an intermediate group trying to be both.

It is my belief that journals as we know them will continue for many generations. They remain lying quietly until they are needed, but in them are recorded the thoughts, dreams, and hopes of those who for centuries have gone before us. Our own *Journal* gives a good example. In it one can trace the history of modern psychiatry from its beginning in this country to the present. We see various ideas emerge and methods tried, disappearing after a time only to rise again later or, perhaps one step higher in the scale. Then, too, the modern researcher wants to have a look at all of the reports of colleagues in his particular field, and good scientific libraries make it easy for the researcher to peruse what he wants to, when he wants to.

One serious concern is the great mass of material being presented to the *Journal* for possible publication. A long lag time before publication is worrisome, and when it extends to a year it becomes distressing to everyone, for many articles are time-limited. At present a committee of our Editorial Board is examining our particular problem and all hope fervently that some answer will be found.

As I leave the editorial chair I do so knowing that the *Journal* is in excellent hands. An editorial office furnishes a marvelous perch from which to observe the overall scene in a medical specialty and I am grateful that I was part of an excellent office in a time of transition. A great deal happened in my 13 years as Editor. Old age and death were "discovered," and these subjects interested me for obvious reasons. A plethora of papers on these subjects and on drugs, licit and illicit, appeared—some good, some not so good. We watched the incredible advances in psychophysiology and psychopharmacology with great pleasure, and we watched disconsolately the failure of some of the community mental health centers to live up to expectations.

From the vantage point it was my privilege to occupy, it has seemed for the last decade that psychiatry was slowly making its way back into medicine, the discipline that nurtured it and from whence it sprang. As it has developed potent therapeutic tools, psychiatry's drift toward medicine has become more apparent. Actually, there are large numbers of psychiatrists who never felt they had left medicine's protective boundaries at all. Nevertheless, the return of the prodigal psychiatry as a specialty to medicine should be a felicitous one.

Personally I am indebted to many people. I have already written and spoken of my affectionate regard for my colleagues on the *Journal* staff and for the Editorial Board. The Board was carefully selected and consists of colleagues who are expert in some aspect of our field. I am deeply indebted to each one of them—they have been helpful always and patient with my calls for help. I shall never forget the kindness, helpfulness, and capabilities of all of them, and I shall not worry about what the future holds for me as long as I can take my memories of all of these wonderful people with me.

As to my future plans, a scheme outlined by Edward A. Reinhard (13) did appeal to me—but only briefly. He spoke of retiring in a few years and getting a big rocking chair. He said, "I shall rock very slowly, read what I want to read and watch the younger lemmings try to avoid getting pushed off the cliff and drowning in an ocean of abstracted, translated, computerized dehumanized data." I thought of that possibility for a while, but then I became afraid that perhaps legislatures, judges, lawyers, and civil libertarians might push our own young colleagues off the cliff; they are working at it at the moment. It was then that I decided not to rock; instead I shall stay on the beach and try to be lifeguard for any of ours that do come over the cliff. I was a lifeguard before I went into medicine and I may as well go out that way. I'll be slower to the rescue, mind you, but I'll be just as intent and just as interested while I retain any faculties at all.

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Is Modern-Day Schizophrenic Outcome Still Negative?

BY MARTIN HARROW, PH.D., ROY R. GRINKER, SR., M.D., MARSHALL L. SILVERSTEIN, PH.D., AND PHILIP HOLZMAN, PH.D.

To evaluate formulations about more positive outcome in modern-day schizophrenic patients, the authors assessed 132 young patients 2.7 years after hospital discharge. Despite modern treatment techniques, only 14%–17% of the 79 schizophrenic patients in the study group were functioning effectively without relapses; 50% had adjusted very poorly. The schizophrenic patients were functioning significantly more poorly than the nonschizophrenic patients in all areas ($p<.01$), showing poor adjustment, high symptom levels, and high rehospitalization rates. The two major acute subgroups (schizo-affective and acute schizophrenic patients) were functioning better than those with paranoid and chronic schizophrenia. Overall, the authors conclude, diagnosis carries prognostic implications. Schizophrenic outcome is more favorable today than it was in Bleuler and Kraepelin's era, but it is still relatively negative.

REALISTIC GUIDELINES on schizophrenic outcome are relevant to theories about the nature of psychosis and are of value to the practicing psychiatrist. Detailed information in this area is all the more important in light of reports of changes in prognosis for schizophrenic patients over the last decade. Traditional beliefs about negative outcome in schizophrenia have been challenged by several recent trends.

One of these trends involves the widespread use in the last 20 years of more effective pharmacological treatments (1–4). The original hope was that the use of phenothiazines would drastically alter the negative outcome traditionally associated with schizophrenia. The traditional view of an extremely negative prognosis was believed to be a characteristic of schizophrenia as recently as 1941, when Kant (5) observed that the recovery rate for schizophrenia was only 1%–7%.

Clinical observations about improvement in outcome for schizophrenia since the advent of more modern treatment techniques have been buttressed by the findings of Strauss, Carpenter, and Hawk (6, 7) in an important program of follow-up research. Their results showed a more positive outcome for schizophrenia than would have been anticipated by most theorists. Similarly, in a 1974 follow-up study of schizophrenic and nonschizophrenic patients (8), members of our research team also reported smaller differences than anticipated between schizophrenic and nonschizophrenic patients, although there were significant differences in favor of nonschizophrenic patients. Although several recent studies have cast some question on the traditional belief in a negative schizophrenic outcome, two recent studies of ours (9, 10) found a surprisingly high degree of symptomatology in schizophrenic patients after the acute phase of their illness.

The reports about a more positive outcome in schizophrenia pose questions concerning our standard concepts of this disorder. A central belief associated with the diagnosis of schizophrenia involves ideas about a negative outcome. This belief is important historically in concepts of dementia praecox and is still prominent in current theory about schizophrenia.

In the research reported on here, part of a larger project studying the long-term course of schizophrenia (10–14), we attempted to resolve some of these issues about outcome for modern-day schizophrenic patients. In particular, we focused on the following four questions related to schizophrenic functioning during the postacute period: 1) Do schizophrenic patients function more poorly than nonschizophrenic patients during the postacute phase? 2) What percentage of schizophrenic patients have a very poor outcome, and what percentage of schizophrenic patients can be expected to have a very favorable outcome? 3) If schizophrenic outcome is negative, which specific areas of functioning are affected, or is the poorer functioning spread across all areas? 4) Do certain schizophrenic subgroups function more poorly than other subgroups? Put another way, does schizophrenic subdiagnosis make a difference?

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METHOD

Patient Sample

To study young schizophrenic patients during relatively early phases of their disorder, we assessed, at the time of hospitalization, 132 inpatients at Michael Reese Hospital (median age=23.5 years, range=17–34 years) and then reassessed them an average of 2.7 years after hospital discharge. The patients were almost entirely from social classes I, II, and III according to the 5-point Hollingshead and Redlich system. These patients were part of the ongoing multidisciplinary schizophrenia project that has been conducted at Michael Reese Medical Center and the University of Chicago since 1970 (10–13).

The sample included 79 schizophrenic patients and 53 nonschizophrenic patients diagnosed by the consensus of two senior clinicians using *DSM-II* (15) criteria. The schizophrenic population included subsamples of 7 patients with schizo-affective schizophrenia, 11 with acute schizophrenia, 22 with paranoid schizophrenia, and 28 young patients with chronic schizophrenia. The nonschizophrenic sample was composed of 28 patients with depressive disorders, 16 with severe personality disorders, and 8 with other diagnoses.[1] There were no significant differences between the schizophrenic and nonschizophrenic patient samples in age, education, and intellectual level.

At the time of follow-up, 46% of the schizophrenic patients were on phenothiazines. During the postacute period schizophrenic patients on phenothiazines were functioning significantly more poorly than schizophrenic patients not on phenothiazines on the major indexes of outcome ($p<.01$, t test). In many cases, however, their poorer clinical conditions may be the reason they were being treated with phenothiazines. The schizophrenic patients receiving psychotherapy at follow-up did not differ in overall outcome from those not receiving psychotherapy. Caution in interpreting the results comparing different medication and psychotherapy groups is warranted because patients were assigned to the medication groups or to psychotherapy on the basis of clinical condition and/or other factors rather than on a random basis.

Posthospital Adjustment

The follow-up assessment of the 132 schizophrenic and nonschizophrenic patients included a structured interview (8, 17), a series of performance tests and projective tests, and several questionnaires, including the Katz Adjustment Scales (18). The follow-[illegible]ule involved detailed evaluation of the follo[illegible]g 1) social, work, and family functioning, 2) the pa[illegible]s's subjective evaluations of their own mental he[illegible]h 3) the patients' intellectual level, 4) their use of [illegible]cation and posthospital treatment, 5) their poten[illegible]e depressive, and psychotic symptomatology [illegible]schotic symptomatology was assessed by means of [illegible]3) form of the Present State Examination (1[illegible]6) hospitalization. The present research focu[illegible]n the patients' level of functioning as assesse[illegible]e evaluating five specific areas of adjustmen[illegible]f adjustment, work or instrumental functionin[illegible]n outcome, rehospitalization, and suicide).

Two scales of overall adjustment were [illegible]. These included a scale developed by Le[illegible]n associates (16) used in the past in seve[illegible]n tions by others (16, 19) and by members [illegible] research group (17, 20). This overall scale [illegible]es the areas of work and social functioning, life [illegible]ns self-support, symptomatology, potential [illegible]e lapses, and rehospitalization. The second [illegible]s was one used by Strauss and Carpenter (6[illegible]in vestigations of posthospital functioning. T[illegible]d vidual scales covering specific areas [illegible]d by Strauss and Carpenter were used to allo[illegible]t rect comparison of the current sample w[illegible]ied by others.

RESULTS

Results for Schizophrenic and Nonschizophrenic Patients on Overall Functioning

Table 1 presents the data for the schiz[illegible]ic and nonschizophrenic patients on Levenstei[illegible]n ates' index of overall functioning (16) for [illegible] of the follow-up period and for the comple[illegible] period. Several major features concern[illegible] level of functioning of the samples can be [illegible] 1.

The schizophrenic patients as a grou[illegible] significantly more poorly than the nons[illegible] patients during the entire follow-up perio[illegible] despite extensive use of phenothiazines [illegible] therapy. A core group of schizophrenic pa[illegible] 50%) had adjusted *very* poorly; these p[illegible] negative outcome in the two major [illegible] symptomatology and a low level of func[illegible] sidered as a group, the schizophrenic pat[illegible] a high rate of rehospitalization; 62% o[illegible] phrenic patients had been rehospitalize[illegible] times since the index hospitalization.

These outcome results are superior to [illegible] years ago during Bleuler and Kraepelin's [illegible] still represent an extremely low level of [illegible] is possible that our modern treatment [illegible] not as effective with certain types of pa[illegible] the core group of patients with severe c[illegible] cox who had an extremely negative outc[illegible]

[1]The results on outcome for the 132 patients include data for 7 patients who committed suicide during the follow-up period. The results on these 7 patients are included in the data on overall outcome in the major measure used (the Levenstein-Klein-Pollack Outcome Scale [16]) to provide a representative picture of overall outcome. However, since they could not be interviewed, data for these 7 patients are not included in the results on such individual areas of functioning as social and symptomatic functioning. They are also not included in the results using the Strauss-Carpenter total outcome score, in accordance with Strauss and Carpenter's system of calculating these results (6), and to allow for comparison of results across samples using the same techniques.

TABLE 1
Overall Outcome for Schizophrenic and Nonschizophrenic Patients*

	Good						Poor						
	Very Good		Equivocally Good		Equivocal		Poor Functioning and Severe Symptoms		Suicide				
Outcome	N	%	N	%	N	%	N	%	N	%	Chi Square	df	Significance
Complete follow-up period													
Schizophrenic patients (N=79)	11	14	5	6	21	26	36	46	6	8			
Nonschizophrenic patients (N=53)	22	42	7	12	22	42	1	2	1	2			
Analysis**											36.43	2	p<.001
Last year of follow-up													
Schizophrenic patients (N=75)	13	17	3	4	24	32	33	44	2	3			
Nonschizophrenic patients (N=53)	28	53	5	9	18	34	1	2	1	2			
Analysis**											33.39	2	p<.001

*According to the Levenstein-Klein-Pollack Outcome Scale scores (16); 1–2=good, 3–6=equivocal, 7–9=poor.
**Good versus equivocal versus poor outcome/schizophrenic versus nonschizophrenic patients.

of Kraepelin. In contrast to these patients, it may be the large group of middle-outcome schizophrenic patients who profit more from modern treatment techniques, who now function somewhat more effectively, and who are no longer placed on back wards or "continuous treatment units." These middle-outcome schizophrenic patients now seem to experience mild to severe difficulties in functioning and sporadic rehospitalizations rather than severe deterioration. Thus, middle outcome schizophrenic patients are still functioning far below the level of the typical "normal" person in our society.

When one looks for the schizophrenic patients who were functioning adequately in all three areas during the postacute phase (functioning at a high level socially and occupationally, with no relapses or rehospitalizations and no symptomatic difficulties), one finds that in our sample only 14%–17% fit into this category. This low rate is, at best, only slightly superior to the rate 70 years ago.

The large significant differences we found between diagnostic groups are not completely concordant with Strauss, Carpenter, and Hawk's data (6, 7), which suggested smaller (but still significant) differences between schizophrenic and nonschizophrenic patients. When the same indexes of functioning used by Strauss and Carpenter (6) were used, our 73 schizophrenic patients who were interviewed were found to be functioning significantly more poorly than our nonschizophrenic patients ($t=5.88$, $p<.001$, two-tailed). Our schizophrenic patients were functioning more poorly than the Strauss-Carpenter sample (the mean score on the Strauss-Carpenter index for our schizophrenic patients was 9.70). In addition, our nonschizophrenic patients were functioning at a slightly higher level than the Strauss-Carpenter nonschizophrenic sample (the mean on the Strauss-Carpenter index for our nonschizophrenic patients was 13.38). Although our schizophrenic patients had poorer scores than the Strauss-Carpenter sample in most individual areas of functioning, our patients had a slightly lower rate of rehospitalization.

The nonschizophrenic patients as a group showed a different pattern of postacute functioning. In contrast to the schizophrenic patients, almost none of the nonschizophrenic patients had a very poor outcome. Over half of them showed a favorable outcome, and most of the others showed an equivocal outcome. The nonschizophrenic patients with an equivocal outcome experienced some difficulty in terms of continuous symptomatology, relapses and/or rehospitalization, or life disruptions and difficulty in functioning. Thus, although the nonschizophrenic patients' outcomes were more favorable, a large number of nonschizophrenic patients experienced difficulty in one or two areas (as opposed to experiencing considerable difficulty in all three major areas, as was found for many of the schizophrenic patients).

Differences Between Schizophrenic and Nonschizophrenic Patients in Various Areas of Functioning

Tables 2–4 show the differences between the schizophrenic and nonschizophrenic groups in various areas of functioning during the posthospital period. The poorer posthospital adjustment of the schizophrenic patients in comparison with the nonschizophrenic patients is not limited to one area of functioning. The differences cut across all major areas at statistically significant levels. This includes significant differences in the social area, in instrumental performance (or functional work role), in extent of psychotic symptomatology, and in rate of rehospitalization.

There were significant differences between the schizophrenic and nonschizophrenic patients in psychotic features and in certain types of thought pathology (see table 3). However, the schizophrenic dysfunctioning during the follow-up period did not occur only in areas related to psychotic symptoms. Many of the greatest differences between the diagnostic groups oc-

TABLE 2
Outcome in Social and Work Functioning for Schizophrenic and Nonschizophrenic Patients

Item	Good N	Good %	Equivocal N	Equivocal %	Poor N	Poor %	Chi Square*	Significance
Social functioning								
Schizophrenic patients (N=73)	33	45	25	34	15	21		
Nonschizophrenic patients (N=52)	33	63	17	33	2	4		
Analysis**							8.17	p<.02
Work functioning								
Schizophrenic patients (N=73)	26	36	25	34	22	30		
Nonschizophrenic patients (N=52)	35	67	14	27	3	6		
Analysis**							15.78	p<.001

*df=2.
**Schizophrenic versus nonschizophrenic patients.

TABLE 3
Psychotic Symptoms of Schizophrenic and Nonschizophrenic Patients

Item	Absent N	Absent %	Intermediate N	Intermediate %	Present N	Present %	Chi Square*	Significance
All schizophrenic patients (N=73)	26	36	14	19	33	45		
Nonschizophrenic patients (N=52)	33	63	14	27	5	10		
Acute and chronic schizophrenic patients (weighted estimate)**		43		17		40		
Analysis***							18.45	p<.001

*df=2.
**This figure is a composite estimate of psychosis derived from an equal weighting of young acute and chronic subgroups, the two major subgroups that were not primarily paranoid.
***All schizophrenic patients versus nonschizophrenic patients.

curred in nonsymptom areas, indicating that the long-term course of schizophrenic psychopathology after the acute phase is not limited to symptom difficulties.

A drastic decline in social skills and social functioning has been proposed by some theorists as the major factor associated with schizophrenic deterioration. The data on the present sample at the 2.7-year follow-up period showed significant differences in social functioning between the schizophrenic and nonschizophrenic patients (see table 2). These included at least some difficulty in terms of sociability (poorer quality of relationships and less frequent association with friends) and some difficulties with spare-time activities. The social area, however, was not the area of malfunctioning showing the greatest differences between the two groups.

The area showing one of the largest significant differences between the schizophrenic and nonschizophrenic patients during the first 2.7 years after the index hospitalization was that of work or instrumental functioning (see table 2). A large percentage of the schizophrenic patients showed at least some difficulty in instrumental performance. This was very striking among the men: three-fourths of the schizophrenic men showed at least some difficulty, or less than adequate functioning, in performance in their societally expected work role.

TABLE 4
Rehospitalization of Schizophrenic and Nonschizophrenic Patients Since Index Hospitalization

Item	None N	None %	One or More N	One or More %	Chi Square	Significance
Schizophrenic patients (N=73)	28	38	45	62		
Nonschizophrenic patients (N=52)	34	65	18	35		
Analysis**					8.87	p<.001

*df=1.
**Schizophrenic versus nonschizophrenic patients.

The schizophrenic patients also tended to evaluate their own mental health in a less positive light than did the nonschizophrenic patients (p<.01, chi-square), possibly in part because of some realistic evaluation of their difficulties in functioning. Although all but a few patients in both diagnostic groups evaluated their psychological state in less than optimal terms, the majority of the patients did not tend to have a severely negative outlook.

Another area of importance concerns suicide. Six of the 79 schizophrenic patients (7.5%) committed suicide during the follow-up period. This is a high rate. It must be kept in mind that the current sample of schizophrenic patients was a relatively young one, and the possibility exists that some of them had experienced considerable difficulty in adjusting to the disappointments and frustrations associated with their problems in functioning and their intermittent or chronic failures. This may have been a factor in some of the suicides. Other experienced clinicians have noted that one can expect a suicide rate of 5% or higher in young schizophrenic patients, and this is in accord with our previous experience in other settings. It is quite possible that an older, chronic, multi-year schizophrenic sample with lower expectations would have a lower suicide rate.

The data on rehospitalization (see table 4) showed significant differences between the schizophrenic and nonschizophrenic patients and also indicated considerable absolute malfunctioning for the schizophrenic patients. Thus, despite the use of modern treatment techniques and aftercare, 62% of the schizophrenic patients had been rehospitalized at least once after the index hospitalization, and at the time of the follow-up assessment 19% were in the hospital. This high rate of rehospitalization is roughly in accord with data on schizophrenic samples recently presented by other investigators. In contrast to the high rate of rehospitalization among the schizophrenic patients, 35% of the

TABLE 5
Overall Outcome for Major Schizophrenic Subgroups*

Outcome	Good: Very Good N	Good: Very Good %	Good: Equivocally Good N	Good: Equivocally Good %	Equivocal N	Equivocal %	Poor: Poor Functioning and Severe Symptoms N	Poor: Poor Functioning and Severe Symptoms %	Poor: Suicide N	Poor: Suicide %	Chi Square	df	Significance
Complete follow-up period													
Schizo-affective and chronic schizophrenic patients (N=18)	3	17	4	22	4	22	6	33	1	6			
Chronic and paranoid schizophrenic patients (N=50)	5	10	1	2	15	30	24	48	5	10			
Analysis**											6.21	2	p<.05
Last year of follow-up period													
Schizo-affective and chronic schizophrenic patients (N=18)	6	33	1	6	4	22	6	33	1	6			
Chronic and paranoid schizophrenic patients (N=46)	4	9	1	2	18	39	22	48	1	2			
Analysis**											6.83	2	p<.05

*According to the Levenstein-Klein-Pollack Outcome Scale scores (16); 1–2=good, 3–6=equivocal, 7–9=poor.
**Good versus equivocal versus poor outcome/schizo-affective and acute schiophrenic groups versus chronic and paranoid schizophrenic patients.

nonschizophrenic patients had been rehospitalized since the index hospitalization.

When the data on the various areas of functioning are viewed from a broader perspective, the importance of such areas of adjustment as work and social performance become apparent. Although work and social dysfunctioning are often seen as secondary symptoms, during the course of the disorder these secondary features become equally or more important than the primary (psychotic) symptomatic features in terms of overall adjustment. If the poorer functioning in work and social areas are really secondary features, do we yet know what the more important or primary aspects are (14, 21)?

Since the schizophrenic patients' dysfunctioning affects almost every area of their postacute performance, it is not surprising that the index showing the largest significant differences between the schizophrenic and nonschizophrenic patients during the posthospital period should be the overall index. The broadest overall index (the Levenstein-Klein-Pollack Outcome Scale [16]), which reflects a number of difficulties for the schizophrenic patients cutting across a variety of different areas, showed the largest significant difference. This result of maximum difference between schizophrenic and nonschizophrenic patients during the postacute phase on the broadest overall index is in accord with data we reported on a different sample living in a different geographic area (8). In addition, the posthospital adjustment of the present schizophrenic sample is similar in some respects to that found in a sample assessed by Levenstein and associates (16) using the same overall index of functioning.

Differences Between Schizophrenic Subdiagnostic Groups

Table 5 presents the overall results for the major schizophrenic subgroups. There was a trend for the two major acute schizophrenic subgroups (the schizo-affective and the acute schizophrenic groups) to show better functioning on the scales of overall functioning, compared with those with chronic and paranoid schizophrenia ($p<.05$). The trend toward poorer functioning among the chronic and paranoid schizophrenic patients was most prominent in the social area ($p<.05$). In the area of work or instrumental performance, the chronic and paranoid schizophrenic patients also showed slightly poorer functioning than the two more acute schizophrenic subgroups, but the difference in this area was not even close to significant. The chronic and paranoid schizophrenic patients had more psychotic symptoms during the postacute phase, but the differences were not significant.

As can be seen by these results on diagnostic subgroups, the trend toward poorer posthospital functioning among chronic and paranoid schizophrenic patients occurred in all major areas of functioning. In a few areas the poorer functioning of the chronic and paranoid schizophrenic patients was at significant levels, but in most areas the differences were much smaller and not significant. These consistent differences across all major areas suggest that there may be some basic differences among subtypes; however, the interpretation of these results suggests several possibilities.

It should be remembered that our group of chronic schizophrenic patients with poor outcome are young patients who have not had years of hospitalization and who are being studied at a relatively early stage in relation to a possible long-term course of future schizophrenic or psychotic breaks. Some studies that have found poorer outcome among chronic schizophrenic patients have used samples of older patients who already had a history of many years of hospitalization. The latter type of research presents a problem because it involves "findings" of subsequent poor outcome for certain subtypes of schizophrenic patients who have

already had many years of poor outcome and hospitalizations before the study—not a very remarkable finding.

Of considerable interest, the differences between the chronic and acute schizophrenic patients in symptomatology and functioning were greater (more negative for the chronic schizophrenic patients) at the posthospital or follow-up period than at the acute phase and during the hospital period, when the original diagnosis was made.

If the severity of schizophrenia is assessed in terms of a negative outcome, our results on differences in outcome between schizophrenic subgroups that had only small to moderate symptom differences at hospital admission support our previous data and formulations in this area. Our previous data (8, 22) suggested that in schizophrenia, unlike some other disorders, the severity of the disorder should not be assessed in terms of the severity of the symptomatic picture during the early active phases of the disorder (e.g., at the time of hospitalization). Rather, factors such as early onset of the schizophrenic disorder and poor premorbid functioning (at times in the social-sexual area) may be better, although still imperfect, indexes of severity of the underlying schizophrenic disorder.

DISCUSSION

Our outcome results have implications for a number of related issues. The current somewhat pessimistic results on schizophrenic outcome should be considered in relation to the increased optimism that has emerged with the advent of more modern treatment techniques.

We sometimes hear that diagnosis is not important today because overall outcome depends on the particular individual and the particular treatment. The form of treatment involved undoubtedly makes some difference, but this does not tell the whole story. The large significant differences we found, as well as the data of Strauss and Carpenter (6), strongly indicate that diagnosis is an important factor; modern-day schizophrenic patients as a group still show a relatively negative outcome.

The poor outcome associated with schizophrenia applies to a number of different aspects of functioning. Both the symptomatic picture and the functioning level of the schizophrenic patient in other areas are negative. The present data suggest that the posthospital picture of schizophrenia is not just a case of debilitating symptoms. In the current sample the postacute work and social functioning of the schizophrenic patients showed large significant differences in comparison with nonschizophrenic patients.

It is difficult to compare the present negative results on modern-day schizophrenic outcome 2.7 years after hospitalization with those found for schizophrenic patients earlier in this century. Precise comparative figures on earlier samples assessed many years ago are not available to make exact comparison possible. The current-day schizophrenic patient functions better after hospitalization than he or she used to. One type of evidence supporting this contention can be found in the indications that many years ago a much larger percentage of first-break schizophrenic patients never left the hospital after their initial or second hospitalization. Although the present data and other results point to the fact that many modern-day schizophrenic patients do not function as poorly as was the case during the era of Bleuler and Kraepelin, the results clearly show that as a group they do not have a benevolent outcome. Only a small percentage of our schizophrenic patients (14%–17%) functioned at a high level without relapses after hospitalization, and a large percentage (about 50%) tended to have a very negative outcome.

The present results do *not* support the observation made by some investigators that one-third of schizophrenic patients function well after treatment, one-third perform in an equivocal manner, and one-third function poorly. These estimates do not apply to the schizophrenic patients we studied, except in the area of work functioning. Further, the assumption that schizophrenic outcome is similar to that of other psychoses was not supported by our data. The majority of the nonschizophrenic patients showed at least some residual or recurring difficulty in functioning but their levels of both symptomatology and difficulty in functioning were not as severe as those of the schizophrenic patients. The differences between these patients and the schizophrenic patients was very large.

The current results suggest a mixed picture. With our modern treatment modalities most schizophrenic patients fare better than they would have in the days of Kraepelin and Bleuler (they no longer experience continuous or even lifelong hospitalization). However, the outcome for the modern-day schizophrenic patient is still a relatively negative one.

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Diagnosis of Nonpatients in the General Community

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Using a sample of 116 subjects drawn from a large metropolitan area, the authors compared ratings of psychopathology from both structured and clinical interviews. Although the two types of interviews yielded similar findings when there was definite psychopathology or definite signs of health, for some subjects in the middle range between health and sickness, the two interviews did not yield similar findings. The authors suggest the need for a new typology to categorize nonpatients in the community who are in the middle range between frank psychiatric disorder and optimal mental health.

ACCURATE knowledge of the prevalence and distribution of psychiatric disorder in the general population is necessary in order to test a number of etiological hypotheses, e.g., social class as a causative factor in the development of mental illness. To date, at least 58 studies throughout the world have attempted to measure psychiatric disorder in the general population without regard to treatment status (1). The wide variation in reported rates in these studies, of under 1% to over 50%, is due to the contrasting concepts and methods for identifying cases used by different investigators. The focus in this paper will be methodological. We will contrast two different types of research interviews employed and relate them to each other. Some studies employ the usual open-ended clinical interview; others use a structured questionnaire. The main disadvantage of the clinical interview method is that it cannot be replicated by other researchers. The advantage of the structured interview is that the procedures are described in specific detail and are thus capable of accurate replication. Replication of studies is essential if the field of psychiatric epidemiology is to advance beyond its present state, in which varying study results are difficult to evaluate and use for testing hypotheses.

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Clinicians in general are skeptical of the d[illegible]ed from structured questionnaires. One res[illegible] instance, concluded that

> Depending on the population involved, esti[illegible] degree of psychiatric disturbance through qu[illegible]-type interviews administered by nonprofessio[illegible] to overestimation of the degree of illness and to [illegible] incorrect conclusions. Clinical assessment appe[illegible] only currently available method to avoid this [illegible]. p. 537)

Leighton and associates (3), in an attemp[illegible] validity of a questionnaire interview, con[illegible] chiatrists' ratings using clinical methods ve[illegible] obtained from structured interviews. They [illegible] that the two methods "show on the whole r[illegible] ment." These researchers drew their subsa[illegible] for this comparison primarily from the "[illegible] that is, those who were rated originally as [illegible] "almost certainly a psychiatric case" or [illegible] evidence of any symptoms of psychiatric [illegible] and some evidence of good health and a[illegible]." However, three-quarters of the 405 subjec[illegible] ton and associates' total sample were r[illegible] "middle," that is, as "probably a psych[illegible] or "possibly a psychiatric case."

The frequency of severe psychopatho[illegible] tively rare in the general population. The v[illegible] of cases one discovers in random survey[illegible] populations are the less severe types, a[illegible] diagnosed with less reliability. For exam[illegible] and Wilson (4) have shown that the clinica[illegible] of schizophrenia and other more seve[illegible] pathological states are more reliable than [illegible] rosis or personality disorder—the less sev[illegible] psychopathology.

The findings of previous investigators [illegible] the comparison of clinical and structure[illegible] should be done using community respond[illegible] structured interview "case" ratings are u[illegible] who report only mild indications of psych[illegible] This group of respondents is the most nu[illegible] also the group most likely to present proble[illegible] uation. If such a group gave similar resul[illegible] structured and clinical interviews, it would [illegible] support to the convergent validity of struc[illegible] views.

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METHOD

A total of 116 subjects from the community were interviewed three times over a one-year period using the Psychiatric Epidemiology Research Interview (5). This instrument is a structured fixed-response questionnaire derived from previous work and covers a large number of areas of psychological functioning. In addition to including the more usual questions related to psychotic, psychoneurotic, and behavioral disorders and psychosomatic illnesses, it also tries to uncover indications of positive mental health as well as stressful life events.

The sample was randomly drawn in roughly equal proportions from five New York City ethnic groups (white Protestants of European ancestry, Jews, Irish, blacks, and Puerto Ricans) on a strict probability basis. Within each ethnic group an attempt was made to balance educational levels in order to unconfound social class and ethnic status.

Trained nonpsychiatrist interviewers from the National Opinion Research Center interviewed 74 of the subjects. These 74 were matched with 42 subjects interviewed by 8 different psychiatrists, most of whom had had previous experience interviewing community respondents using other structured research instruments. These psychiatrists, unlike the lay interviewers, rated each subject according to the Midtown Manhattan (6) and Stirling County (7) rating systems and made a psychiatric diagnosis if they thought one was indicated.[1] The psychiatrists were trained to reliably use the Stirling and Midtown ratings. No reliability training was done for diagnosis, and the psychiatrists, who generally had diverse training and experience in psychiatry, were asked to diagnose according to *DSM-II* (8).

Two psychiatrists independently reviewed the 42 written protocols of the subjects interviewed in the field by other psychiatrists. The reviewing psychiatrists selected subjects for a clinical follow-up interview based on the following criteria: there were indications of impaired functioning and/or psychiatric symptoms, psychopathology was not gross or severe, impairment in functioning was not marked, and the clinical picture did not conform to a psychiatric diagnosis according to *DSM-II*. These criteria were established in order to select those subjects who would be most likely to present problems in evaluation and would, therefore, presumably be the ones most likely to have discrepant results on the structured and clinical interviews. Leighton and associates (3) chose their subsample mainly from the categories in which there was little question about whether or not a subject was a psychiatric "case," and they were, therefore, more likely to find agreement between the two interview methods. The rationale for our adopting the strategy of selecting the subjects who were most likely to present problems in evaluation was that if we found the two interview methods to give similar results, it would be strong support for the convergent validity of structured interviews.

[1]The Stirling County and Midtown Manhattan rating systems are well known in the field of psychiatric epidemiology. The Stirling rating, developed by A.H. Leighton and associates, has been used for studies in the Maritime Provinces of Canada and in Africa as well as other places. The Stirling rating is made on the basis of whether a respondent is a psychiatric case. No attempt is made to specify which particular diagnostic category fits the respondent, but only whether one or more diagnoses apply. The rating does not refer to the severity of the diagnosis. The Midtown Manhattan rating system, on the other hand, is based on a gradation of severity of symptoms and impairment.

RESULTS

Eleven subjects were selected for follow-up. Those selected for clinical evaluation had been rated in the structured interview as having less severe symptoms and less impairment in functioning, and they were less likely to be psychiatric cases than those not selected (see table 1). There were no differences in the range of diagnoses between the two groups (see table 2). Three subjects refused or could not be located for a clinical interview. Two of the 11 were clinically interviewed immediately after the third structured interview because the interviewer had many questions about the responses given at that time. One of the 11 would not consent to a fourth interview, but 2 different psychiatrists spoke to her at length over the phone. The remaining 5 were interviewed for the fourth time in their homes by a psychiatrist.

Three psychiatrists reviewed the results from the 8 respondents for whom there were sufficient data for a clinical evaluation. On the basis of a consensus rating the 3 made a final evaluation. In making their final evaluation they had access to the results of the clinical and structured interviews plus all other information collected on the respondents. Thus the final evaluation by which the structured interview was to be compared was based on all of the information available on each of the respondents.

As a result of the clinical evaluation, 6 of the 8 subjects received a change in their rating as to whether or not they were a psychiatric case according to the Stirling County rating system. Three became cases, and 3 who were originally judged to be cases were in the final evaluation judged not to be psychiatric cases. The 2 respondents who required no changes in their evaluation were generally functioning at a high level and reported almost no symptoms. However, they were selected for follow-up because there were indications of psychopathology that may have been missed in the structured interview, admission of some suicidal thoughts by 1 subject, and the other subject's fathering of 2 handicapped children, which frequently results in psychiatric symptoms. Clinical interviews did not reveal anything remarkable in these 2 respondents.

Two respondents had answered positively to some items ordinarily indicating psychosis, but after a clinical interview it was determined that they had misun-

TABLE 1
Distribution of Ratings of 42 Subjects from the Community

Rating*	Subjects Selected for Clinical Interview (N=11)	Subjects Not Selected for Clinical Interview (N=31)
Stirling County**		
A	2	11
B	1	6
C	4	9
D	4	5
Midtown Manhattan***		
0	2	6
1	7	10
2	2	2
3	0	7
4	0	2
5	0	4
6	0	0

*All ratings are based on the third structured interview.
**A=is almost certainly a psychiatric case; B=is probably a psychiatric case; C=is possibly psychiatric, borderline; and D=no evidence of any symptoms of psychiatric significance and some evidence of good health and adjustment. The dividing line between being judged a case or not is between B and C.
***0=no significant symptom formation; 1=mild symptom formation but functioning adequately; 2=moderate symptom formation with no apparent interference in life adjustment; 3=moderate symptom formation with some interference in life adjustment; 4=serious symptom formation and functioning with some difficulty; 5=serious symptom formation and functioning with great difficulty; and 6=seriously incapacitated, unable to function.

TABLE 2
Distribution of Diagnoses of 42 Subjects from the Community

Item	Subjects Selected for Clinical Interview (N=11)	Subjects Not Selected for Clinical Interview (N=31)
Diagnosis*		
Psychotic organic brain syndrome	0	1
Involutional melancholia	1	0
Anxiety neurosis	3	6
Hysterical neurosis, conversion type	0	1
Phobic neurosis	0	1
Depressive neurosis	1	10
Neurasthenic neurosis	0	1
Antisocial personality	0	1
Other personality disorder	0	1
Alcoholism	1	1
Psychophysiological disorders	1	4
No diagnosis	4	4
Interviewer's subjective certainty of diagnosis		
Extremely	2	6
Moderately	7	16
Slightly	1	3
Not indicated	1	6

*All diagnoses are based on the third structured interview.

derstood the questions and that no indications of psychosis were present. (For example, to "mind dominated by forces beyond your control?" one respondent said "yes," but in the clinic she explained that she meant "controlled scious forces.") One respondent answere to a few items probing for anxiety, but the did not evaluate her as a psychiatric case viewed her in the usual clinical manner. A cal interview he felt, and the reviewing concurred, that she had evidence of ma and a psychiatric diagnosis was indicated.

In the following 2 cases the respond very little psychopathology in the stru view, but after the clinical interview psychiatrists concurred that a psychiatr present.

CASE REPORTS

Case 1. Mr. A, a 59-year-old man from an nic group, had responded negatively to all item psychopathology with the exception of a few the frequency was "almost never." He was clinical interview because he said that he had the last 12 months.

Mr. A was extremely helpful and cooperat for the interview. In regard to sexual relations, that it was more likely 2 years rather than later in the interview he said that it had been since he had had sexual relations. He told th viewer with obvious discomfort. His wife was and cooperative. The two of them appeared to to get along well with each other. They had t were successful and of whom they were proud, as they showed the interviewer pictu their grandchildren.

The subject had lived in the same neighb since birth. It had originally been middle class slum. His parents were deceased, and all of h sisters and other relatives had long since m more affluent surroundings. His apartment w but pleasant, in marked contrast to the outs hood, which had all the familiar signs of ur poverty.

Mr. A had graduated from high school and showed above average intelligence, but he h able to hold a job much above that of a clerk tried more demanding work, he felt uncomfo He was now earning $125 a week and said th happy with his income and found it easy to liv socialized with other people and rarely saw did not know his brother's occupation bec want to be "nosy" and ask. His parents h and he had been close to his mother, who d years earlier. Paranoid tendencies emerge scribed why he never socialized with anyone people never returned favors, they took adv and they had made his wife sick.

Mr. A smiled constantly. He often went int tail and was irrelevant at times. There w appropriate affect, thought disorder, and pa He had a lifelong pattern of retreating from a resulted in anxiety for him. He had restricted pationally, socially, and sexually. He was di reviewing psychiatrists as having a personalit

Case 2. Ms. B was a 39-year-old woman from a disadvantaged ethnic group. She was moderately obese, had been separated, had never had any children, and had attended college but was now doing work markedly below the level for which her training qualified her. During the structured interview she gave almost no positive responses to questions directed at eliciting psychiatric symptoms.

Ms. B was extremely helpful and cooperative; she was also friendly and intact and acted quite appropriately. Her home was nicely furnished and well kept. When questioned about her marriage, she said that her husband had been "crazy jealous" and she had had to leave him several years before. She had had a miscarriage while married. Before her marriage and at the time of the study she was having an affair with a married man. As she proceeded to talk of her affair, she became progressively more depressed looking and finally started crying. When it was brought to her attention that she was crying she denied it. She terminated the interview after 45 minutes, saying that she had just remembered another appointment. She quickly composed herself and was smiling again as the interviewer left.

How can one evaluate this case? The further Ms. B was questioned, the more difficulties as well as positive strengths emerged. It is reasonable to assume that more pathology would have emerged with further interviewing. It is clear that a great deal of information was revealed in the relatively brief clinical interview, but was the information sufficient to warrant a change in the diagnostic impression? Three psychiatrists reviewed the data and concurred that a psychiatric diagnosis was indicated. Structured interviewing alone had not led to a psychiatric diagnosis. The signs of depression elicited in the clinical interview are consistent with the obesity previously noted. There were signs of many ego assets in this woman, along with decided psychopathology. The two existed side by side without the emergence of symptoms strong enough to propel the subject to seek help.

In the first two cases significant but well defended pathology emerged with the use of a clinical interview. In the following case the clinical interview resulted in changing the evaluation from a psychiatric case to "not a psychiatric case."

Case 3. Mr. C was a 61-year-old man from a disadvantaged ethnic group. The structured interview revealed evidence of depression, anxiety, alcoholism, and psychophysiological symptoms and the fact that Mr. C was taking tranquilizers. He was rated a "case." The clinical examination did not rule out psychopathology, but it did reveal such a high degree of adaptive functioning that a rating based on the subject's psychopathology alone is misleading.

Mr. C had grown up in an agricultural setting. He had always been anxious and fearful and came to New York City hoping to rid himself of the anxiety. He went to college also hoping to make himself secure and less frightened. He got a job in a large corporation and, again out of anxiety, pushed himself. He achieved considerable economic, occupational, and social success. During the time of his three structured interviews he was in a crisis on his job. He had recently been promoted and was involved in "political" and administrative tasks that were unlike his previous technical duties and caused him considerable anxiety. His drinking, depression, and anxiety had been with him all along but were intensified by recent events. His family physician had given him some mild tranquilizers.

He appeared to have a good relationship with his wife and family. He and his wife had discussed her feelings about their children having left home and what they should do. They decided to take in two foster children, whom they both enjoyed. He had found his structured interviews thought-provoking and helpful. He said he had gotten some insight into his problems when he started to think about some of the questions raised in the interviews. He discussed with the interviewer the possibility of entering therapy. In general, he appeared to the interviewer to be a much more alive and vital person than Mr. A and Ms. B, who had admitted to little psychopathology.

CONCLUSIONS

We selected subjects for clinical evaluation partially on the basis that the protocol of the structured interview presented problems in evaluation. Because of the way the subjects were selected, the finding that 6 out of 8 cases required a change in diagnosis does not support the hypothesis that structured and clinical interviews yield essentially dissimilar results. On the other hand, congruent evaluation in the 8 would have been strong support for the hypothesis that structured and clinical interviews result in essentially the same findings.

Two subjects misinterpreted questions in the structured interviews, and the full extent of one subject's anxiety was not revealed in the structured interview. One would expect some subjects to fall into these two categories. Adding probes to some questions might correct for those "errors" resulting from structured interviews.

For other respondents such as the three described in detail above, it is doubtful that improving a structured interview would be sufficient to detect either the subtle signs of pathology exhibited in the clinical interview or the full panoply of positive strengths and human warmth that discount other signs of pathology. In terms of making a final diagnostic evaluation, the clinical interviews also fall short. The cases described here may represent a group of individuals in the community who range from exaggerated but psychologically normal character types to well-compensated personality disorders. They rarely seek treatment, often experience little subjective distress, and establish a stable equilibrium in which mild psychopathology is balanced by strong inner resources and an ability to cope. It is questionable whether conventional psychiatric diagnosis applies in such instances where pathology and health have established a working symbiosis.

Beiser (9) has considered the problems in evaluation that are raised by this middle-range group. He stated, "The more assets a person displays the less likely he is to effect the kinds of negative maladaptations, which we have come to think of as psychiatric disorder, when confronted with stress. This is the quantitative aspect of the relationship between 'assets' and 'liabilities.' Different patternings of this relationship should also emerge, and should enable us to construct new

typologies to describe the kinds of adaptation which fall between optimal functioning and frank psychiatric disorder." (9, p. 250)

We agree with Beiser that a new typology is required to categorize nonpatients in the community who are in the middle range between frank psychiatric disorder and optimal mental health. Structured and clinical interviews yield similar findings when there is definite psychopathology or definite signs of health, but our study suggests that for some respondents in the middle range between health and sickness, the two interviews did not yield similar findings. Greater congruence in evaluation might be achieved by improvements in systems of typology.

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Some Determinants of Maternal Attachment

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The authors conducted prenatal interviews and behavior observation sessions with 46 families in which the woman was pregnant. These families were again interviewed and observed 7 days, 1 month, 2 months, and 6 months after the child was born. The authors found that the most significant variable predicting the variance of maternal attachment was the length of separation of mother and infant: less separation was associated with greater attachment. The next most significant variable was birth experience, followed in importance by the length of labor and prenatal attitudes and expectations.

MOTHER-INFANT INTERACTION has received increasing attention in recent years (1, 2), along with the beginnings of the elucidation of some determinants of maternal attachment (3–5). Ainsworth (6) reviewed infant behavior patterns that seemed characteristic of attachment.

Kennell and associates (7) indicated that a unique period of special sensitivity for bonding may exist immediately after delivery. Rosenblatt (8) presented animal studies indicating that the onset of maternal behavior is based on hormonal secretions and that the postpartum maintenance of maternal behavior depends chiefly on the stimulation that the mother receives from her young. Rosenblatt (9) and Bronfenbrenner (10) presented evidence that disruption of the natural course of animal behaviors either before or immediately after delivery has deleterious effects on maternal behavior. Bronfenbrenner (10) also proposed that a critical period exists for the organization and consolidation of maternal behavior.

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Animal (11, 12) and human (13) studies have provided evidence that bonding occurs through early maternal-infant proximal body contact (skin-to-skin for the human). Behavioral differences have been found in the quality of the mother-infant relationship between human mothers permitted as little as half an hour of skin-to-skin contact followed by 12 hours of separation compared with mothers not permitted this initial contact (5, 7, 13). These effects have held for up to 1 year. Greenberg and associates (14) found more confidence and responsiveness to the infant among mothers permitted more contact with their full-term newborns than control subjects. Rose and associates (15) and Kennell and Rolnick (16) noted the long-term consequences of brief illnesses and perturbations of mother-infant relationships during the first 2 days of life.

In a previous study of the same group of families to be reported on here, Peterson and associates (17) found that the most important predictor variable in determining father attachment was the father's degree of participation in the birth process and his subjective report of the birth experience. In this research we attempted to quantitate the relative effect of a number of birth-related variables on the development of maternal attachment and to consider the implications of these findings for intervention with mothers and families at risk.

METHOD

Subject Selection

We selected 46 families who were not from any ethnic minority and who had middle- or upper-class socioeconomic backgrounds. For purposes of intragroup as well as intergroup comparisons, we subdivided the families into three groups: those in which the woman experienced natural childbirth without anesthesia in the hospital (N=21), those in which the woman experienced natural childbirth without anesthesia in her own home (N=13), and those in which the woman experienced delivery under anesthesia in the hospital (N=12).

The 46 families were drawn from three sources: patients of an obstetrician in private practice in Palo Alto, patients at the Stanford University Obstetrics and Gynecology Clinic, and clients of midwives of the Santa Cruz Birth Center. (The activities of this group of midwives and statistics on their work have been de-

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scribed elsewhere [18, 19].) The names of women interested in participating in this study were obtained from these three sources, and the women were contacted by telephone. The details, protocols, risks, benefits, and purposes of the study were explained and consent was obtained for an initial interview. At the initial interview these issues were again discussed. Anonymity was assured, subjects were told that they had the right to withdraw from the study at any time, and consent to proceed was obtained.

Procedure

The families were interviewed between the 6th and 8th month of the woman's pregnancy. The woman was asked about her desire for the pregnancy and the baby, her projected plans for taking care of the infant (including what social values she would teach, how much emotional nurturing she would provide, and how much of a time commitment it would require), her projected level of emotional satisfaction with the baby and her current level of satisfaction with the pregnancy, her opinion of the importance of the mother to the child as an infant, her expectations for the delivery, her level of psychological awareness of the needs of infancy, the amount of involvement planned for the father at delivery, her expectations of how the child would change her life, her attitudes about feeding an infant, and her projected confidence in herself as a mother. Discrepancies between observed behavior with the children in the home and plans for the new child were noted, along with disagreement between the marital partners. Each of the factors was scored, and the total score represented a prenatal attitude score. We felt that high scores indicated ease of attachment of the mother with her infant. Questions were also asked about behavior with any other children and the family's sociodemographic status.

The mother's behavior during labor and delivery was observed and recorded, as was the length of maternal-infant contact following delivery. After delivery, the mother was asked about her emotion satisfaction with the delivery and her feelings about her child and the birth. A birth experience score was developed on the basis of the mother's statements about delivery, her behavior during labor and delivery, her immediate reaction to her newborn, the degree of father involvement, the amount of maternal-infant contact sought after postpartum affect (e.g., elation, depression) was evident, and observed and reported behavior of the mother with the infant. High birth experience scores indicated that birth was a positive experience for the mother, that she was an active participant in the delivery, and that she sought and maintained (to the extent possible) close interaction with the infant.

Fathers and mothers were interviewed again, usually during the first week after delivery and 1, 2, 4, and 6 months later. During these sessions the mother was asked questions about her feelings of closeness to the infant and her feeling that the baby was hers, her caretaking behavior, her time involvement with the infant, her confidence in her ability to care for the baby, her feelings about caring for the infant in the middle of the night, her behavior in comforting the baby when she cried, and her feelings about the baby's motherhood, and the work of caretaking.

Discrepancies between reported and observed behavior were noted along with discrepancies between the mother's report and the reports of others. The level of agreement between the couple was noted. The behavior of the mother was recorded in a structured fashion. From these factors, an attachment score was developed. A high attachment score indicated that 1) the mother felt the baby was hers soon after delivery, 2) she was confident in caring for her infant, 3) she sought and maintained close contact with the infant for a high percentage of the available time, 4) she was confident caring for the infant in the middle of the night without excessive resentment or anger directed at the infant, 5) she responded to the infant when he or she cried, usually knowing what the cry was a request for, 6) not letting the baby cry for more than 1–2 minutes, 7) she felt reasonably happy and satisfied with the infant and with motherhood, expressing more positive feelings than negative feelings in general, 8) she related easily with her infant during the interview, holding him or her closely on occasion and comforting the infant when he or she cried, and 8) others agreed with the mother's assessment of her behavior.

With the exception of the interview in the immediate delivery, all of the interviews were conducted in the family's home. The interview team usually consisted of one interviewer who knew what kind of information the family had participated in and who had developed a high degree of rapport with the family and a second individual who did not and was present to make additional observations. On a random basis, individuals would not have foreknowledge of the family coming for research interview. This was usually done once for each family.

Data Analysis

Data were coded from recorded tapes of the interviews by three individuals, at least two of whom did not know the history of the couple. All of the individuals coded together until agreement between them reached 85%. Then every 10th tape was coded by two individuals to assure that interobserver reliability was above 85%.

Variables included in the data analysis were race, education, amount of analgesia/anesthesia, duration of mother-infant separation in the first 72 hours after delivery, length of labor, birth weight, time from delivery to the first extended feeding contact, participation in birth environment, and a disappointment score (scored with 0 for the woman who had the kind of delivery planned and 1–5 for the number of steps away from the kind of delivery planned). Positive separation scores indicated decreasing amounts of mother-infant interaction.

Each group was analyzed separately using stepwise forward-directed multiple regression analysis with a

inclusion criterion of $p<.10$ and an exclusion criterion of $p>.12$. We used the Stepreg 1-Statjob program on the Univac 1101 computer of the Madison Academic Computing Center. Variables were added to the regression according to their order of significance in contributing to their increase in the coefficient of determination until no more variables met the inclusion criteria. The program was constructed to select the initial variable as the one that would make the most significant prediction. Then the entire group was analyzed.

Our original biases were that the amount of analgesia/anesthesia, amount of maternal-infant separation, and birth environment would be the most important variables. Of these, only separation was found to be important. From a bias coding, individual interviewers' preferences did not seem to affect the results. The study was designed to assess the impact of pregnancy and birth-related variables on maternal attachment and does not consider the effect of infant variables or the mother's own early experience and parenting.

RESULTS

For the entire sample (N=46), the most significant variable predicting the variance of maternal attachment was the amount of maternal-infant separation: less separation correlated with greater attachment (partial correlation coefficient $[r_p]=.708$, significance of the increase in the regression prediction $[p\Delta r^2]<.00009$). This was followed in significance by birth experience ($r_p=.617$, $p\Delta r^2<.00009$), length of labor ($r_p=.319$, $p\Delta r^2=.0371$) (longer labors were associated with greater attachment), and prenatal attitude ($r_p=.300$, $p\Delta r^2=.0504$).

Sixty-nine percent of the variance of maternal attachment was predicted ($F=22.99$, $p<.00009$). This left 31% of the variance to be accounted for by the unmeasured factors, including infant variables and the mother's own early experience. For the subgroup of families in which the woman experienced delivery in the hospital with anesthesia, only the disappointment factor was predictive of subsequent maternal attachment ($r_p=.520$, $p\Delta r^2=.470$). This factor presumably related to the differences between couples who planned a natural or a home birth compared with those who planned a hospital delivery with anesthesia. We concluded that preparation and commitment to a delivery style in which greater personal control and parent involvement was possible increased attachment even when those plans could not be realized. The mean attachment scores of couples planning nonanesthetized deliveries who then had anesthesia were closer to the mean scores of those who planned to use anesthesia, although they were somewhat higher. We concluded that prenatal preparation had a positive effect on attachment even when plans were not realized and that it more than offset any theoretical negative effects of parental guilt feelings for not actually having the type of delivery planned. This disappointment factor accounted for 27% of the variance.

For the group planning natural childbirth in the hospital, the birth experience was the most important variable in predicting maternal attachment ($r_p=.743$, $p\Delta r^2=.0015$), followed by length of labor (longer labors were associated with greater attachment) ($r_p=.730$, $p\Delta r^2=.0020$), and maternal-infant separation (less separation was associated with greater attachment) ($r_p=-.465$, $p\Delta r^2=.0808$). Sixty-two percent of the variance of maternal attachment was predicted by these three variables ($F=21.25$, $p=.0005$), leaving 38% of the variance predicted by variables not measured.

For the group planning home birth, the birth experience was the only significant predictor variable ($r_p=.697$, $p\Delta r^2=.0056$), with 49% of the variance predicted, leaving 51% unaccounted for. There was no maternal-infant separation in this group, so this did not appear in the regression equation.

The case studies reported below emphasize the interaction of the variables discussed. In all of these cases, the contribution of variables other than the ones we studied were such that one would expect the development of an optimal mother-infant bond.

CASE REPORTS

Case 1. Ms. A, a 26-year-old woman who was employed part-time as a map drawer planned a home birth. Her husband was an anthropologist. Their first child, age 3, had been born without analgesia/anesthesia in a local hospital with the father present.

Ms. A delivered a girl on Christmas day at home; she experienced no separation from her family. When interviewed 3 days after the birth, she expressed the feeling that she was emotionally closer to this baby than she had been to her first child at the same age. She felt no postpartum depression with this delivery, although she had with her first. She attributed her immediate sense of attachment to this second child to the fact that she could hold, nurse, and sleep with her baby immediately after she was born. She said she was able to get more rest in her home than in the hospital and felt that she had healed more rapidly in her home surrounded by helping family members and friends rather than by nurses.

Case 2. Ms. B was a 28-year-old woman who was employed full-time as an airline ticket agent. Her husband worked full-time as an airline machinist. They had a 2-year-old son. Ms. B expected to make use of anesthesia in labor and delivery with this child as she had with her first and expected to return to work full-time when her newborn reached 3 months of age.

When interviewed after the delivery, Ms. B reported that she had not been able to take anesthesia because she did not get to the hospital soon enough. She reported feeling very surprised at her enjoyment of the birth process and expressed the feeling that seeing and feeling her baby being born was a very positive and emotionally strengthening experience for her. She was surprised at how much actually seeing and feeling the birth process meant to her. She reported having a great deal of faith in herself after the birth of this child, her second son, and she was grateful for the unplanned accident of a natural birth.

She held her baby for half an hour after the delivery, after which she experienced a 16-hour separation from her newborn. She said she felt emotionally closer to this baby by the end of 24 hours—sooner than for her first child, from whom she had experienced a greater separation. One month after the delivery she was considering the possibility of part-time work to facilitate breast feeding. At 6 months she was still breast feeding and working part-time.

Case 3. Ms. C was a 25-year-old librarian expecting her first child. Her husband was a graphic artist. Both wanted a home delivery. Due to a prolonged labor and uterine inertia, however, Ms. C delivered in a hospital. She had a forceps delivery and received epidural anesthesia; for 5 days she was separated from her newborn most of the time.

When interviewed soon after delivery, 1 week after delivery, and 1 month after delivery, Ms. C expressed feeling that she did not know her baby very well, that she was confused as to what to do with the newborn, and that she was helpless. She experienced a great deal of depression for the month after the delivery and felt in general less confident about motherhood than she had anticipated. She said it took her longer than she had anticipated to get to know her baby and that the baby cried more than other babies. She also said that she felt her baby was insecure during the first month, which she believed was due to the lengthy separation they had experienced in the hospital. She described the separation as traumatic for her and said she had been prone to many crying spells during this time. She described motherhood as "very trying."

In general, Ms. C experienced the birth process as ego deflating, traumatic, and lonely. She appeared to compensate for her negative experience by adopting an attitude of faith in fate and in her religion.

Case 4. Ms. D was a 32-year-old woman who taught tennis part-time. Her husband was a lawyer. They had a 3-year-old daughter. She planned a hospital delivery with epidural anesthesia, as she had had with her first child. The father planned to be present.

Ms. D delivered her second child in a hospital under epidural anesthesia for both labor and delivery. As she had with her first child, she experienced a separation from her newborn for approximately 18 hours the first day, 18 hours the second day, and 18 hours the third day after delivery.

She described the childbirth experience as very unpleasant but was grateful that this labor had been very short (4 hours). In her interview shortly after delivery she expressed the feeling that she was as close to this son as she had been to her daughter at the same age—1 week. She said she did not know either of them very well at this point and consequently did not feel they were totally hers yet. When interviewed 1 month later she again expressed the feeling that she did not really know her baby yet and did not feel it was really hers. She still felt the baby to be a stranger in the house. However, she felt that closeness would grow as time went on, as it had with her first child. She also stated that because her daughter had been her first child, she still felt closer to her than to her new baby.

DISCUSSION

The findings reported here emphasize the importance of the actual birth experience as an influence on maternal attachment. From this we would [illegible] model for maternal attachment similar to [illegible] of Bronfenbrenner (10) and Klaus and Kennell (3). Maternal attachment is dependent on many f[illegible]. We hypothesize that the factors operative prenatally along with hormonal influences aid in the deve[illegible]ment of maternal behavior, which is organized by birth experience and then consolidated by the presence of the infant and the interaction of mother and infant during the immediate postpartum period. From the results of this study, it would appear that birth experience and separation are important variables. Appendix 1 lists the stages in the development of attachment, along with the influences, disruptions, and types of intervention possible at each step. In all stages, interventions include easy access to family therapy and [illegible] mental health services, home health support for families needing health services, and public education programs.

We envision the mother's experience during labor and delivery as a crucial transition point in the development of maternal attachment—a period of crucial stress or crisis. We hypothesize that the unfolding intrapsychic events of labor constitute the organization of all prenatal influences into a series of distinctive maternal behaviors and affectional beliefs. These psychic associations with labor and delivery will affect the process, which, finally, is consolidated by the presence of the baby and the responsiveness of the baby to the mother's behaviors and feelings. The presence of the baby provides the concrete reality to the fantasized object and, as such, both releases and concretizes maternal attachment.

Bronfenbrenner (10) noted that when the behavior of an animal is disrupted before consolidation has begun, the behavior is absent from the animal's repertoire. When behavior is disrupted during but before completion of consolidation, the behavior will be expressed when the animal is placed in a situation requiring the emission of that behavior, but either inappropriately high frequency or with an inappropriately low threshold. We hypothesize that this effect explains the high occurrence of battered/neglected child syndrome and the overprotected child syndrome among the parents of premature infants (5, 13, 15). The separation of premature infants is extreme. We speculate that these varieties of these conditions may operate in the parents of full-term infants whose behavior has been disrupted before or during consolidation. Therefore the sensitive period that Klaus (13) argued for can be seen as the period of consolidation of behavior. Subsequent to this period, behavior can still be integrated, but it is a more difficult task and does not have the natural mechanisms of the species assisting the process.

During the birth process, the mother is [illegible] vulnerable; she is opening up both physically and mentally. Separation from her infant following delivery may act as a powerful punishment for [illegible] behavior, but the presence of the baby may [illegible]

erful reward. This may explain the beneficial effects of a positive birth experience on maternal self-esteem (20) and on the spontaneous resolution of neurotic conflicts (19). Subsequent experience, unexpected or changing life conditions, and unconscious motives may also have vital influences on the attitudes and values of parents and on the development of attachment.

The assimilation of the baby into the family is affected by projective identification (21), which may be an explanatory factor for the development of psychopathology in a family member (22). The physical presence of the baby diminishes this effect by permitting reality testing. Separation maintains the infant in the status of a fantasy object—the longer the separation, the greater the tendency toward projection.

A negative birth experience in which fear and pain and discomfort predominate may have far-reaching consequences. Fear and pain breed resentment and hostility toward the object associated with the fear, pain, and discomfort. Fathers, in a sense, may act as affect meters for this process. We have heard fathers expressing jealousy and hostility toward their infants because of the pain and discomfort their wives experienced in childbirth. We have observed this ambivalence persisting in the relationship of both parents with the infant. Such a phenomenon has also been observed in clinical psychiatric practice. This is in sharp contrast to fathers and mothers who perceive birth as a positive emotional experience.

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APPENDIX 1
Stages in the Development of Attachment

I. Birth of Mother
  A. Influences
    1. Experience of being nurtured
  B. Disruptions
    1. Genetic factors affecting intelligence, etc.
    2. Caretaker-infant separation
  C. Opportunities for intervention
    1. Prevention of maternal-infant separation
    2. Active social work and nursing intervention for families at risk
    3. Interventions discussed in stages V and VI

II. Childhood experiences
  A. Influences
    1. Parents' development of psychological difficulties with assumption of parental role
    2. Parents' provision of appropriate role models
    3. Sociocultural identification and learning regarding role-specific behavior
  B. Disruptions
    1. Discontinuity in caretaking
    2. Family dynamics
    3. Family crises
  C. Opportunities for intervention
    1. Inclusion of education regarding childbirth in high school and grade school curricula
    2. Involvement of siblings in childbirth whenever feasible
    3. Easy access to mental health services and paraprofessionals in the community for family therapy
    4. Provision of adequate role models
    5. Social intervention (e.g., in the case of poverty)

III. Marriage before children
  A. Influences
    1. Relationship with father of child
  B. Disruptions
    1. Marital conflict
  C. Opportunities for intervention
    1. Couple therapy
    2. Family planning services
IV. Conception and pregnancy
  A. Influences
    1. Desire for pregnancy
    2. Prenatal attitudes toward the pregnancy, childbirth, and the infant
  B. Disruptions
    1. Unwanted pregnancy
    2. Complications of pregnancy
    3. Family crises
  C. Opportunities for intervention
    1. Social work and nursing support for families with complications
    2. Childbirth preparation classes providing new role models for parenting
    3. Provision of information regarding alternatives to childbirth
V. Birth
  A. Influences
    1. Birth experiences
    2. Length of labor
  B. Disruptions
    1. Environmental disruption
  C. Opportunities for intervention
    1. Support and encouragement for low-risk mothers choosing alternative birth environments
    2. Avoidance of disruption of naturally occurring behavior of parents during labor
    3. Provision of support services for high-risk parents
    4. Avoidance of unnecessary obstetrical intervention
    5. Facilitation of friends' and relatives' involvement in labor
VI. Early postpartum period
  A. Influences
    1. Infant's behavior
    2. Father's development of attachment
  B. Disruptions
    1. Maternal-infant separation
    2. Environmental disruption
  C. Opportunities for intervention
    1. Avoidance of maternal-infant and family-infant separation
    2. Avoidance of disruption of naturally occurring parental behavior
    3. Teaching new behaviors and providing new role models for parents at risk
    4. Aggressive community services and support for families at risk
    5. Involving parents in the care of sick neonates

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The Role of Infant Observation in Child Psychiatry Training

BY GEORGE W. GREENMAN, M.D., RONALD BENSON, M.D., LEXINGTON GRAPENTINE, M.D., CECILY LEGG, M.S.W., AND IVAN SHERICK, PH.D.

The authors discuss the role of infant observation in the training of child psychiatrists, emphasizing the overriding importance of the subtle but crucial interactions in the mother-infant relationship. Infant observation is valuable in preparing the student as an observer of behavior, and developmental concepts are taught and learned more easily in programs that provide opportunities for observing normal infants. The infant study program in the Department of Psychiatry of the University of Michigan Medical Center is presented as a model.

BECAUSE OF the numerous reprint requests for articles on infant and early childhood research published in the *Journal of the American Academy of Child Psychiatry*, the editors of that journal published a collection of many of them, entitled (provocatively) *Infant Psychiatry, A New Synthesis*. The editors proposed a new subspecialty of infant psychiatry and argued that child psychiatry training is a logical starting place for such a program (1). An upsurge of interest in the first year of life is evident from the large number of recent books and articles dealing with various aspects of infancy (2–6).

Selma Fraiberg addressed the problem of who should provide diagnostic assessment and treatment to infants and their families (unpublished manuscript). She advocates a subspecialty in infant mental health for all professionals who serve babies and their parents, including psychiatrists, pediatricians, pediatric and psychiatric nurses, psychologists, and social workers.

One of the possible reasons for the call for infant psychiatrists is that most child psychiatry training programs pay scant attention to infancy (7). At best, normal and pathological infant development are presented in didactic lectures. The student rarely sees a normal baby and even more rarely is assigned an infant for evaluation or treatment.

DEVELOPMENT OF AN INFANT OBSERVATION PROGRAM

Because of the need for more experience with infants and their families, infant observation became part of the two-year program in child and adolescent psychiatry at the University of Michigan in 1974. Couples having their first baby are invited to participate in the program on the basis of their own interest, the probability of a stable family situation, the likelihood of at least a year's residence in the area, a delivery date in September, and the absence of overt psychopathology. Preference is given to first-born infants for several reasons. The student has the opportunity to observe and comprehend, in its incipiency, the complex phenomenon of parenting. Inclusion of only single-child families serves as a means of limiting somewhat the number of variables (always large) in the study population. More pragmatically, the single-child family tends to have greater freedom and flexibility for scheduling appointments to synchronize with the trainee's limited availability. An additional advantage in the use of single-child families is that, for beginning observers, it is generally easier to carry out an infant observation without the distractions provided by older siblings. Stable couples give the resident a greater opportunity to observe the unfolding of a relatively normal family and, on a more practical level, tend to be home when home visits are scheduled and to keep appointments for office visits. The expectant couples are referred to us by other participants in the program or are recruited by senior staff in contacts with childbirth preparation classes, such as Lamaze.

PRENATAL AND PERINATAL PERIOD

Each child psychiatry fellow is assigned a family in July at the beginning of his second year of child psychiatric training. He is asked to interview the couple separately and conjointly in the weeks before the baby is born, obtaining a detailed personal history from husband and wife. In addition, an attempt is made during

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the interviews to gather data concerning the myriad hopes, expectations, and worries surrounding the unborn child. These parental fantasies, both positive and negative, give the resident his first revealing glimpse of the elusive ethos into which the infant will be born. We have found, for example, that important expectations exist regarding the sex, intelligence, motor ability, and physical attractiveness of the child. In the months to come, this fantasy material can be compared with the realities of the young child's endowment. The attitudinal changes of the parents in the face of this reality can be followed. Often, from the earliest days parents attempt to shape the child to fit their conscious and unconscious expectations. The student is in a good position to learn and to intervene if indicated because he knows the prebirth fantasies.

The resident is encouraged to obtain permission to be present at the birth. The delivery experience, which usually includes the fathers, affords the resident a unique opportunity to observe the earliest mother-child-father interactions. At the same time, he can be aware of any complications in the birth process and can assess the newborn along several measures, from the Apgar rating to visual tracking. Also, the resident's knowledge of obstetrics is occasionally a source of support to the father. Most important, however, is the direct observation of the initial dialogue between mother and child. The mother's affective state and her comments about the infant can be most revealing, and the newborn's responses when the mother holds him can be an important index of their future interactions (8).

While they are still in the hospital, the mother and infant are visited several times. This gives the resident a chance to explore in depth the mother's attitudes toward the newborn and to contrast them with her earlier wishes and fantasies. At breast or bottle the child is observed in the mother's care, and many of her basic nurturing skills come to light. The resident notes the baby's beginning ability to integrate the complex physiological functions of sucking, swallowing, and breathing that comprise nursing. In the nursery or at the mother's bedside the baby can be directly examined and held by the trainee for the first time, an intimate contact that may be a new and important experience for the trainee. He acquires a "feel" for his subject, which adds to his ability to assess temperament, alertness, and the infant's ability to mold. He can observe how primitive and yet how highly organized is the newborn's behavior. Frustration tolerance, output of activity, contrasts between states of alert inactivity and waking activity (9), and many other characteristics are sometimes precursors of later behavior.

In September, usually soon after the baby's birth, the child psychiatry fellow is joined by a second team member, a nurse enrolled in the master's degree program in parent and child nursing at the University of Michigan School of Nursing. The nurses are on a semester system and therefore miss the beginning and end of the family's year-long participation.

HOME AND OFFICE VISITS

About 2 weeks after delivery, the child psychiatry fellow and nurse make a home visit at a time when the father can be present. This offers both trainees their first opportunity to observe the complete family in its natural surroundings, with the rich variations offered by the ever-changing circumstances of family life, and provides a further chance to sharpen their skills as participant observers. The observers are alerted to note the mother's affective tone, skill, and comfort as she feeds, bathes, diapers, and interacts with the baby. The baby's responses are noted in similar detail. With the mother's consent and approval, each observer holds the baby and makes observations of his social responsiveness, muscle tone, and molding. This routine, to that of the observer. In addition, they ask the parents about the behavioral patterns of the infant and about their attitudes and behavior toward this new family member. The home visit also allows the team to make direct observations of the nature of the fathering and to take a close look at the quality of the interactions between the mother and the father concerning their baby.

The program is devised for teaching rather than service. However, parents occasionally seek to use the study situations to further their knowledge of child development or to seek reassurance on their parenting styles. Appropriate response is made to such requests. Although the observers are not looking for problem situations, when a possible developmental hazard is noted it is shared with the parents.

Home visits continue at monthly intervals throughout the year. The visits are required for the nurse trainee, elective for the child psychiatry fellow, depending on the time available in his crowded schedule.

Each month, after the initial home visit, the mother, father (if he can come), and baby visit the office. An updated history is taken, focusing on such issues as sleeping and feeding patterns, preferred styles of parental interaction with the child, and the infant's increasing awareness of the world around him.

Formal developmental testing is also done (10). The child psychiatry fellow is responsible for testing procedure. He now becomes familiar with the "tools of the trade" of infant developmental assessment—bell, colored ring, wooden cubes—and learns which tests are appropriate for the child's age. Among other things, he tests for the presence of neonatal reflexes and learns when a positive response should no longer be expected. In general, he becomes adept at eliciting and understanding the neurophysiological signs of growth. The test procedure is videotaped and can be followed simultaneously by other trainees from a distant monitor. Thus, other residents gain first-hand look at more than one infant. In this way they begin to see the variations among infants in function and temperament and come to appreciate the norms and regularities in the unfolding of each infant's mental and emotional development. In addition, the trainee

ability to observe accurately and discuss these observations is heightened. As the infant matures the test data include information about such complex phenomena as, for example, his ability to relate to human objects. The attitude of the child toward complete strangers and toward the tester can be documented, and changes with time in the young child's response to being out of direct contact with the mother can be assessed. Perceptual ability, gross and fine motor control, and cognitive style can also be assessed early and followed in detail over several months.

The nurse is a nonparticipant observer during the office visits. The nurse can note and record complex interactions between the mother, infant, child psychiatry fellow, and video technician during the developmental testing that might otherwise escape notice. For example, the mother's reactions to the examiner's "taking over" and managing the baby during the testing are informative. The child psychiatry fellow, as a beginner in infant developmental testing, may be too involved in administering the test to be attuned to these interactions.

Before the monthly office visits, the resident-nurse teams meet with one or more senior staff members for a briefing on the administration of the infant tests and a discussion of expectable developmental phenomena for infants of a particular age. The babies are all close to the same age, so normal variation is emphasized. If the child psychiatry fellow has not made the home visit two weeks earlier, he has the opportunity to read the nurse's written report before the office visit. After the families are seen, the students and teachers reconvene to discuss the results of the morning's observation and to review the videotape, if desirable. Office space suitable for infant observation is limited, so a maximum of three infant-parent pairs is seen on any one morning. Supervision is primarily accomplished in these group briefing and debriefing sessions. Each fellow is assigned an individual supervisor with whom he can meet if a problem arises that cannot be dealt with during the group supervision.

DATA RECORDING

Another facet of the infant study experience is documentation of data. Following each contact, the trainee writes a report outlining the important observations. At the end of the year, each resident gathers together his knowledge of the child he has been following into an infant developmental profile, a modification of Ernest Freud's baby profile (11). The profile requires a detailed awareness of parental history, the pregnancy and perinatal period, the range of parent-child interaction, latent parental attitudes, and neurophysiologic milestones. Preparing the profile allows the resident to formulate his understanding of the infant's maturational unfolding, constitutional strengths and weaknesses, and significant environmental influences. The profile imposes on the trainee a sense of discipline in his attempt to delineate the critical forces that impinge on the first year.

Infant observation dovetailed neatly with existing opportunities for observation of normal children in our toddler groups and nursery school. A basic goal of our training program is to enhance a thorough understanding of the variations and common characteristics of normal human development. The influences and attitudes of the people comprising the environment and the elements provided through maturation are closely scrutinized. The infant observation opportunities provide the trainee with a rare opportunity to follow, from the earliest moment, the unfolding of these maturational and environmental dimensions and to observe their manifestations and interactions.

For many child fellows, involvement with very young children is not new. Several of the residents are parents; some have come to the child psychiatry program from general practice or a residency in pediatrics, and most have dealt with infants to some degree during their medical school years or internship. What is unique in the infant study experience is the chance it affords to observe objectively essentially healthy infants over a prolonged period and to assess in detail the vicissitudes of early development of a particular child.

VIDEOTAPE USE

All office visits and many home visits are recorded on videotape. A first imperative for the use of videotape is a camera operator who is more than a technician. The camera operator must have an interest in and some basic knowledge of the psychological development of the infant. In the hands of a skillful trained video technician who is fully conversant with the aims and goals of the particular study, the video camera is an invaluable instrument for recording and collecting data that would otherwise be omitted from the record or documented only inferentially.

CASE ILLUSTRATIONS

The practical value of the infant observations in child psychiatric training can be underscored by two brief case vignettes.

Case 1. We began following John A, one of our first infants, when he was 6 weeks old. His intelligent, well-educated parents had heard about the infant study from a friend.

John grew and developed exceedingly well in all areas that we could measure. He was breast-fed and ate well; later he accepted solids readily. He was sleeping through the night consistently by 18 weeks. Motor and language development proceeded at or above the expected rate. His most characteristic trait, which charmed all who had contact with him, was his winning and frequent smile. He was a person-oriented baby, and he smiled broadly at anyone who approached,

until "stranger anxiety," which started when he was about 6 months old, made him a bit more wary.

A crisis requiring intervention by the child psychiatry fellow occurred when John was about 7 months old. Mrs. A had taken John to visit her mother in a distant state. The grandmother was described as a compulsively neat woman who would not adjust or rearrange her home for her increasingly mobile grandson. She refused to participate in John's care because he was crawling and required control. Mrs. A cut her visit short and returned home feeling dominated by John. The demands of his care seemed endless, and she felt that she had no time for herself. A few days later, her former employer called to see if she could return to work. She felt tempted but was ambivalent about leaving John in the care of a stranger. During the office visit in which the child psychiatry fellow obtained this information Mrs. A had to leave the room for about 5 minutes. The change in John was dramatic. He had been smiling joyously at every eye contact with his mother, the child psychiatry fellow, and the video cameraman to that point but quickly became somber and started looking around as if trying to locate his mother. By the time his mother returned he was fussing and, although he was comforted when she held him, he would not cooperate in the developmental testing.

The child psychiatry fellow used the above observation to underscore the intervention he now made. He told Mrs. A that he agreed with her about her continuing importance to John but empathized with her, noting how endless the process of child rearing can seem from time to time. He interpreted her desire to return to work, now that John could crawl, as being somehow an echo of her mother's attitude during the recent visit. Mrs. A recalled intense conflict with her mother about messiness, seemed to settle down, and left the office thinking in terms of a part-time job that would give her some time for herself. A month later, she had dropped the idea of returning to work and contented herself with taking some informal classes.

We believe that John's despair at discovering his mother's absence in the presence of strangers in a strange place shows that at 7 months his cognitive development had proceeded to the point at which he could clearly differentiate the strange from the familiar. He responded with anxiety when his mother left the room because of fear of the loss of the preobject, i.e., the person who reduces tensions through appropriate care. He had recognized his mother as a source of need satisfaction, and her absence evoked anxiety. Indeed, other observations indicated that John desired his mother for more than a source of tension relief, and thus his object relationships had started along the developmental line from need satisfaction to object constancy. To have been separated from her at this point in his development could have been particularly traumatic for him and might have resulted in developmental interference or more serious problems. The intervention helped to allow John's development to unfold without hindrance.

Case 2. Margaret B was conceived 6 months after the death, at age 3 weeks, of an infant born with multiple congenital anomalies. Throughout the prenatal interviews both parents made repeated references to the first baby and expressed concern about the health of the unborn infant. At delivery, Margaret was found to be fully intact, but the parents, understandably, continued to harbor latent concerns. By age 6 months, there were slight differences between Margaret's development and that of the other infants in the study of similar age. Mrs. B became overtly anxious. At this point the child psychiatry fellow made an extra home visit without a video cameraman. Parental concerns were explored and the death of the first baby was reviewed. Mrs. B tied her awareness of Margaret's slightly delayed motor milestones to her own lack of athletic abilities and implied that she wanted her daughter to be better endowed than she had been. The child psychiatry fellow reaffirmed his earlier statements about the baby's normalcy. Shortly after the home visit, Margaret achieved important milestones and the mother's anxiety waned considerably. Since that time, the parents have periodically used the child psychiatry fellow in "consultation" when questions or concerns have arisen.

In this case, the primary cause for the parental anxiety, which threatened to interfere with optimum parenting for Margaret, was the earlier birth and death of the multiply deformed child. The mother's reference to her own "unathletic" nature and some of her other comments suggest a self-concept of damage or deformity that would have been (understandably) exacerbated by the birth of a deformed baby. The supportive and expressive intervention gave the parents a chance to once again review their feelings about the first baby, to express their concerns about Margaret, and to review the objective evidence indicating Margaret's intactness.

DISCUSSION

A comprehensive discussion of the advantages to the child psychiatrist, and the adult psychiatrist for that matter, of a thorough understanding of maturation and development (see reference 12) during the first year of life by direct observation could be the subject of another paper, and a few of the most important aspects will be mentioned here (see reference 13). Near the top of any list of advantages must be the opportunity to observe that the human infant develops in a matrix of object relations and that during the first year the relationship to his mother is of overriding prominence. The importance of the infant-mother relationship is usually taught so didactically that the phrase may become meaningless. Its meaning is given immediate substance when one observes the interaction between the pair, beginning when the baby is given to the mother in the delivery room. The informed observer can see that the mother's role as the infant's first teacher begins then. The teaching may have varying qualities within the same mother-infant pair, as seen recently when a delighted young mother, who was very adept at holding, comforting, and nursing her 2-day-old baby, stopped the baby from touching his mouth, saying that it could develop into a bad habit.

The relationship is not only in terms of how the mother affects the infant. The infant's characteristics, such as sex, health, appearance, activity, and ease with which it can be comforted, can have a profound influence on the mother. The trainee is in a unique position to observe this phenomenon because of his previous knowledge of the mother's hopes, fantasies, and expectations concerning the baby. Knowledge of the mutual influences of the mother and child from birth is useful to the future child psychiatrist in assessing both

infants and children. The skill of unintrusive, nonjudgmental observation that the trainee learns in observing infants will enhance his work as a psychotherapist of children and adults.

Nearly everything that is learned in lectures, seminars, and from reading takes on new meaning when directly observed. Developmental milestones are a good example. Observing the 4-week-old infant smile in response to its mother, watching the 16- to 20-week-old's efforts to reach and grasp, noting the transfer patterns of the sitting 28-week-old, the wariness toward strangers of the 40-week-old, and the delight in locomotion of the 52-week-old, provides more powerful inducements to remembering milestones. Perhaps even more important is the opportunity to watch the precursors of mental functioning taking shape. Primitive reactions to displeasure may foretell what kinds of ego defenses the infant will use in the future (11). Earliest memory for inanimate objects can be seen when, as part of the infant test, a toy is hidden. Very early, one can see which infants are people-oriented, which are thing-oriented, and which show an even balance between the two. One can discern styles of cognitive development and the different ways by which early concept formation is achieved. The teaching and learning of all developmental concepts is enhanced by direct infant observation.

We are often asked why families volunteer to be in our program and what we give them in return. We do not offer service or advice. The answer, in general, is on multiple levels. On the surface are such motivations as the wish to cooperate in a program that helps to train doctors, the opportunity to have their baby seen (and admired) by experts, and reassurance that the baby's development will be monitored. Slightly below the surface is the desire for emotional support during the transition from being a separate entity into that ever demanding and potentially rewarding role as parent. Parents often wish, unconsciously, that participation in the program will protect their infant from disasters that have happened to family members in the past. This was illustrated in case 2.

A training program like ours is not designed to graduate infant mental health specialists; rather, it is designed to acquaint the trainee with the developmental vicissitudes of infancy. With that background, the graduate can make some interventions and knows when to refer more complicated cases.

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Prediction of Imipramine Antidepressant Response by a One-Day *d*-Amphetamine Trial

BY DANIEL P. VAN KAMMEN, M.D., PH.D., AND DENNIS L. MURPHY, M.D.

This study provides additional evidence that there is a moderate association between the acute activation, euphoria, and antidepressant responses to d-*amphetamine and the antidepressant response to imipramine during a four-week trial. Comparison of the responses of 13 patients to* d-*amphetamine on two different days during a double-blind trial indicated that differences in* d-*amphetamine response are consistent, replicable characteristics of individual depressed patients. The variables of sex, diagnosis, diurnal mood variation, platelet MAO activity, and MMPI scale scores were of minimal assistance in revealing factors that might be associated with activation or antidepressant responses to* d-*amphetamine in this small patient group. The authors suggest the need for larger-scale studies in this area.*

SINCE THE tricyclic antidepressants became available in 1954 (1, 2), attempts have been made to develop criteria for their most effective clinical use (3, 4). However, only a few studies of response prediction have appeared in the literature (5). In 1971 Fawcett and Siomopoulos (6), in a study with a small number of depressed patients, reported that the antidepressant response to 1- or 2-day administration of *d*-amphetamine provided an indication of the fourth-week treatment response to tricyclic antidepressants. If a 1- or 2-day administration of a drug such as *d*-amphetamine can predict responses to antidepressant drug trials, which may otherwise require a 3- to 6-week trial, a more rational psychopharmacological approach to the treatment of depression could result, rather than present-day trial and error (7).

Although amphetamine is known to be a CNS stimulant, there is a wide variation in individual response (8–11). This study is an attempt to replicate the Fawcett and Siomopoulos findings (6) and the subsequent more extended studies by Fawcett and associates (12, 13) and also to examine some of the individual characteristics that may play a role in the response to *d*-amphetamine. We report on the self-rated effects of oral *d*-amphetamine in a group of depressed patients and compare this response with the fourth-week antidepressant response to the tricyclic antidepressant imipramine.

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The authors wish to thank the patients and nursing staff of the 3-East clinical-research unit, NIH Clinical Center, for their cooperation in the study, Jean Colison for assistance with the data analysis, and Estelle Churgin and Janice Grice for editorial and secretarial assistance.

METHOD

Subjects

Thirteen inpatients (7 women and 6 men, with a diagnosis of primary affective disorder, including 9 unipolar patients, 1 bipolar I, and 3 bipolar II depressed patients, received trials with both *d*-amphetamine and imipramine. The patients' ages ranged from 18 to 60, with a mean of 43 years. Eight additional patients (4 men and 4 women, of whom 5 were unipolar and 3 bipolar I depressives) received *d*-amphetamine trials alone. These 8 patients had all previously received a full imipramine trial before they were admitted to the NIH Clinical Center and were all considered imipramine nonresponders because they were still sufficiently depressed to require hospitalization after meeting the criterion of 3 weeks or more of treatment with imipramine in doses of 150 mg or more per day as outpatients.

All patients were voluntarily hospitalized and had signed informed consent forms before participating in the studies. They were diagnosed according to the Research Diagnostic Criteria (14) for primary affective disorder. Patients with organic brain syndrome, alcoholism, or medical illness were excluded. Unipolar patients were depressed patients without a history of mania or hypomania, while bipolar I patients were depressed patients with a history of hospitalization for mania; bipolar II patients had a history of hypomania that was not severe enough to require hospitalization for mania.

Patients were rated daily for depression and mania by the research nursing staff using the Bunney-Hamburg Rating Scale (15). The means of the depression ratings of the week before each drug protocol were taken as baseline depression ratings for that protocol. All patients had been off psychoactive drugs for at

least 15 days before any of the drug protocols and had been maintained on a controlled monoamine diet. Before each drug protocol all patients had had experience with the self-rating scales. They received identical capsules four times daily during the entire study period.

d-*Amphetamine Protocol*

Twenty-one patients went through a 7-day double-blind, placebo-controlled protocol. This consisted of oral administration at 7:25 a.m. of 30 mg of *d*-amphetamine (as the base) on 2 separate days and placebo capsules on the other 5 days. Placebo always was given on the first and last days of the protocol. In every case the 2 *d*-amphetamine days were separated by at least 2 days on placebo. For the evaluation of the subjective effects of *d*-amphetamine, the patients completed a 34-item mood and behavior self-rating scale 11 times during the protocol day between 7:15 a.m. and 9:30 p.m. (10). Groups of items covered by the scale included activation, euphoria, depressed affect, and dysphoria (10). The average of the 9:00, 10:00, and 10:30 a.m. ratings (the times of peak drug effects) was used for the evaluation of the morning drug effect. Previously reported *d*-amphetamine levels peaked around this time and correlated with behavioral responses to amphetamine (16).

The ratings from the first placebo day were always excluded from the analysis to eliminate "first-day" effects of the study. The self-ratings from the morning (9:00, 10:00, and 10:30 a.m.) were compared with those from both the afternoon (2:00 and 4:15 p.m.) and evening (7:00 and 9:30 p.m.) on the placebo days for the evaluation of a diurnal variation in depression. The self-ratings from the 4 placebo days were averaged and compared with the self-ratings from the same times on the *d*-amphetamine days. The mean of the placebo days' ratings was subtracted from the mean of the *d*-amphetamine days' ratings to determine the *d*-amphetamine response. We have previously reported that the administration of *d*-amphetamine on 2 different days induced similar behavioral responses, which permitted the use of the mean of the 2 days' responses (10). The replicability of the effects of *d*-amphetamine on the 2 days was evaluated again in this group of depressed patients.

Imipramine Inpatient Trial

Thirteen patients received a 4-week trial of imipramine during their hospitalization at NIMH, with a mean daily dose of 230 mg per day during the fourth week (range of 150–300 mg per day). Imipramine administration was preceded by 2 weeks of placebo and followed by placebo. The response was defined by comparing the means of the daily behavioral depression ratings on the fourth week with the means of the daily ratings before the onset of the imipramine trial. A change larger than 1.5 on the Bunney-Hamburg scale represented a statistically significant change and reflected a clinically significant improvement (13). Responding patients were divided into 2 groups: moderate improvement (1+) if the mean changes in depression ratings were between 1.5 and 2.5, and marked improvement (2+) when the mean response was larger than 2.5 rating units. Responses to imipramine were defined as "no change" if a mean depression rating decrease of less than 1.5 occurred. This allowed us to rank order the patients over a 3-point range. Responders and nonresponders received similar mean daily doses of imipramine in the fourth week; responders received 235±40 mg per day and nonresponders 216±67 mg per day (t=0.638, p=n.s.).

TABLE 1
Correlation Between *d*-Amphetamine Responses and Imipramine Antidepressant Trial Response

Group	N	Correlation (τ)			
		Activation	Euphoria	Depression	Dysphoria
Women	7	.55	.55	−.65	−.22
Men	6	.64	.70	−.23	.08
Total unipolar	9	.48	.53	−.46	−.13
Unipolar women	6	.45	.45	−.60	−.15
Total	13	.61*	.61*	−.48**	.09

*p<.01.
**p<.05.

MMPI and Individual Differences in d-*Amphetamine Effects*

It has been suggested that personality factors may play a role in response to *d*-amphetamine (17–19). Twenty-one patients completed the Minnesota Multiphasic Personality Inventory (MMPI) (20). The clinical subscale scores were examined for possible correlations with the individual responses to *d*-amphetamine.

Monoamine Oxidase

Platelet monoamine oxidase (MAO) activities were evaluated against *d*-amphetamine responses. MAO platelet activity (in nmol/10^8 platelets per hour) was determined in 21 patients using blood samples obtained just before the first placebo day of the *d*-amphetamine trial, according to previously described methods (21).

Data Analysis

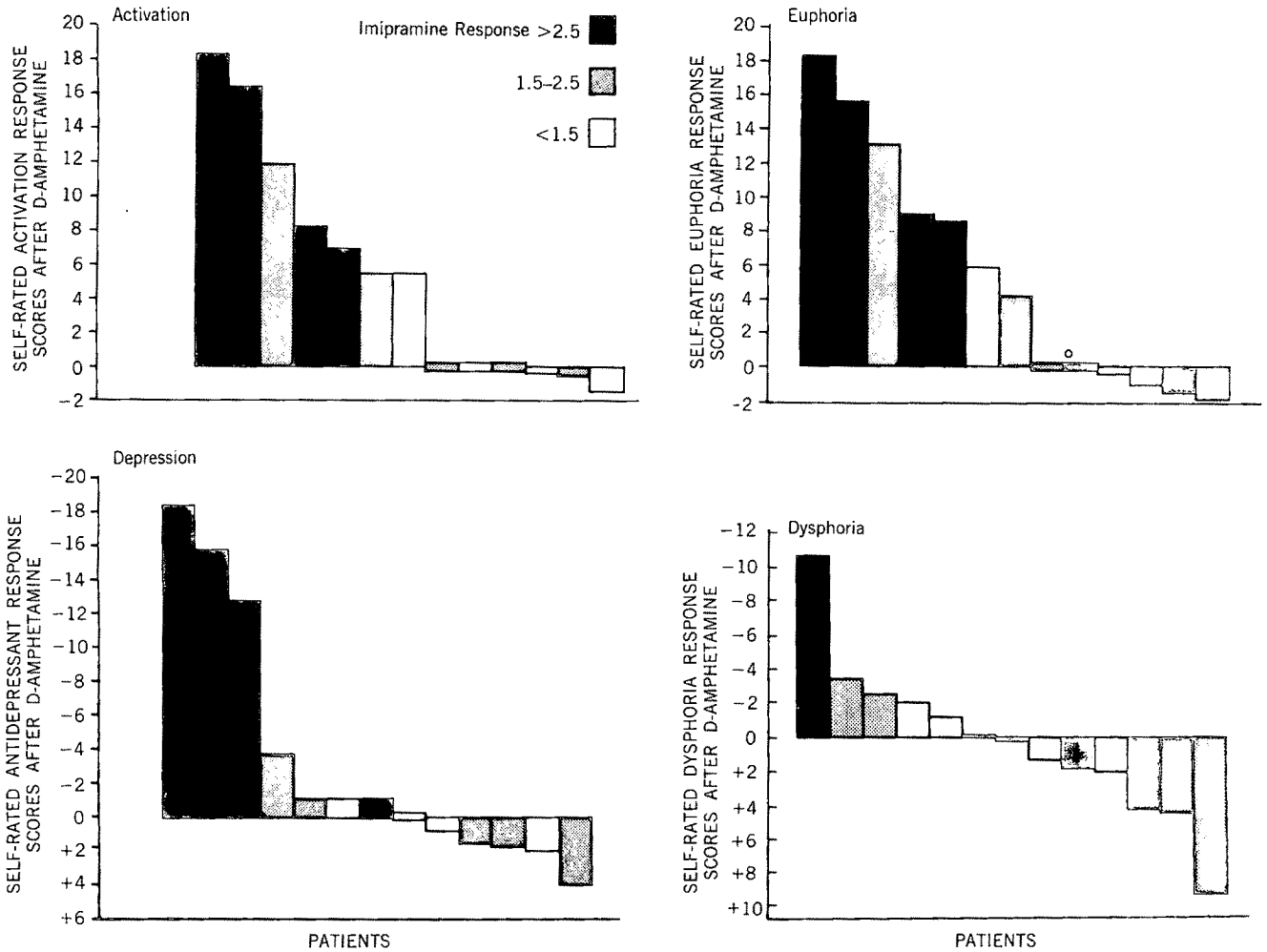
Spearman's (r) and Kendall's (τ) rank-order correlations were used for nonparametric data analyses. For continuous data Pearson's correlation coefficient was used. Two-tailed t tests were applied for paired and nonpaired data analyses.

RESULTS

Relationship Between d-*Amphetamine and Imipramine Responses in the Imipramine Inpatient Trial*

Significant correlations were found between the *d*-amphetamine-induced changes in activation, euphoria,

FIGURE 1
Self-Rated Change Scores of 13 Patients on Activation, Euphoria, Depression, and Dysphoria Items*



*The change scores were recorded 1½–3 hours after the oral administration of 30 mg of *d*-amphetamine in the morning. The imipramine responses were observed during the fourth week of imipramine treatment for each patient.

and depression self-ratings and the antidepressant effects of imipramine (table 1). Figure 1 shows the individual responses of these patients to *d*-amphetamine and imipramine. Patients are rank ordered according to their responses to *d*-amphetamine. In general, patients with the greatest activation, euphoria, and antidepressant responses to *d*-amphetamine also tended to improve with imipramine. There was no apparent relationship between the dysphoria responses to *d*-amphetamine and imipramine antidepressant responses.

Relationship Between d-*Amphetamine and Imipramine Responses in the Total Sample*

The *d*-amphetamine responses of the patient group that had only preadmission outpatient trials with imipramine closely resembled those of the inpatient imipramine responder group rather than those of the inpatient nonresponders (table 2). When these 8 imipramine nonresponders by history were included in the rank-order study, the previously significant correlations all became nonsignificant. Furthermore, when this entire group was divided into male, female, unipolar, and bipolar groups, almost no significant correlations were observed between the self-rated responses to *d*-amphetamine and the antidepressant responses to imipramine, although the small numbers of patients and lack of equal numbers of patients in the different sex-diagnosis categories render these comparisons unlikely to be informative. Only the euphoria self-rated response in the 7 bipolar patients correlated significantly with the antidepressant imipramine responses (Kendall's $\tau=0.65$, $p<.03$). It is of interest that the bipolar and unipolar patients exhibited other differences when intercorrelations between the subjective responses to *d*-amphetamine were examined (i.e., activation versus euphoria versus dysphoria versus antidepressant response). While the bipolar patients had no significantly intercorrelated subjective responses to *d*-amphetamine, the unipolar patients showed highly significant intercorrelated subjective re-

TABLE 2
Means and Ranges of Self-Rated Changes in Behavior After *d*-Amphetamine in Imipramine Inpatient Responders and Nonresponders and in Imipramine Nonresponders by History

Group	N	Response			
		Activation	Euphoria	Depression	Dysphoria
Inpatient imipramine responders	9				
Mean		7.4±2.4	7.5±2.8	5.0±2.8	0.3±1.9
Range		−6.0−18.3	−1.5−18.2	−4.4−18.3	−10.7−9.2
Inpatient imipramine nonresponders	4				
Mean		0.9±1.6	0.6±1.8	−0.4±0.7*	0±1.0
Range		−1.5−5.5	−1.9−8.9	−2.0−1.1	−2.1−2.0
Nonresponders by history	8				
Mean		8.3±3.0	7.9±2.9	6.0±1.4	−0.6±1.0
Range		−0.5−24.8	−3.1−19.1	1.4−12.8	−5.6−3.1

*p<.005 (comparison of imipramine inpatient responders with nonresponders).

sponses, with a strong positive correlation between activation and euphoria (r=92, p<.001) and strong negative correlations for activation and euphoria versus dysphoria (r=−.59, p<.01) and depression versus the other self-rated categories (r ranged from −.72 to −.77, p<.001).

Replicability of d-*Amphetamine Responses*

Self-rated antidepressant responses on the first and second days of *d*-amphetamine administration were significantly correlated at the time of peak drug effect (r=91, p<.001, N=19). This was also the case with the self-rated activation (r=.52, p<.05), euphoria (r=.63, p<.01), and dysphoria responses (r=.87, p<.001, two-tailed t test).

Relationship Between Diurnal Variation in Self-Rated Depression on Placebo and Response to d-*Amphetamine*

To evaluate whether circadian variations in mood would affect drug response, we examined the relationship between *d*-amphetamine responses and change of mood during the day. Differences between morning versus afternoon and morning versus evening self-rated depression on placebo were not significantly correlated with *d*-amphetamine responses in the 21 patients.

MMPI Clinical Subscales and Individual Differences in d-*Amphetamine Effects*

The depression (r=−.47), psychasthenia (r=−.48), and social introversion (r=−.46) subscales of the MMPI showed significant negative correlations with activation responses to *d*-amphetamine at the time of peak drug effect (p<.05). Dysphoria responses to *d*-amphetamine were negatively correlated with the hysteria subscale (r=−.52, p<.02). No other significant correlations were observed between the MMPI clinical scales and the subjective responses to *d*-amphetamine.

MAO and Individual Differences in d-*Amphetamine Effects*

Platelet MAO activity (mean ± SEM) for the men was 8.8±0.97 nmol/10^8 platelets per hour; the women 13.2±1.23; the unipolar patients 11.9±1.2; and the bipolar patients 9.4±1.20. MAO activity did not seem to be related to the euphorogenic (r=.06) or activation effects (r=−.03) of *d*-amphetamine in this sample, in contrast to the findings of a previous report (22).

DISCUSSION

We found significant correlations between the self-rated responses to *d*-amphetamine and the antidepressant effects of imipramine, results consistent with the one previous study reporting a significant association between individual differences in responsiveness to these two drugs (6). We caution that our data and the data of Fawcett and his colleagues (6, 12, 13) do not necessarily imply that *d*-amphetamine response will predict response to imipramine in all patients, because both of these studies used small groups of hospitalized patients who were carefully selected for depressive features. Larger-scale investigations, such as inter-hospital studies, are needed to establish the predictive value of *d*-amphetamine in a wider variety of depressed patient subgroups.

The relationship between the responses to the two compounds disappeared when 8 additional patients were included in the sample after their *d*-amphetamine responses were evaluated. These 8 patients presumably had not responded to a trial of imipramine before coming to NIMH and were currently in the same depressive episode. One possible explanation for the discrepancy between the inpatient imipramine sample and the imipramine nonresponders "by history" may come from the work of Johnson (23) and Kotin and associates (24). These authors reported that depressed patients frequently are inadequately treated with antidepressant drugs before hospitalization, often because of premature discontinuation, incomplete compliance, or inadequate dosage regimens. Since the 8 patients had *d*-amphetamine responses that were similar to those of the NIMH imipramine responders, some nonresponders may have responded to imipramine under strict inpatient supervision and/or with higher doses. Careful clinical documentation before and during

treatment may also have been lacking with these patients. It would seem that quantitative psychological ratings could be useful not only in a clinical-research setting but also as a part of standard clinical practice. Similarly, in our double-blind evaluation of the neuroleptic treatment of schizophrenia, we frequently have found that supposed nonresponders may respond to neuroleptics if the dosage and treatment course are carefully monitored (van Kammen and associates, unpublished data).

Our methodology is somewhat different from that of Fawcett and Siomopoulos (6) in that we used two administrations of 30 mg of *d*-amphetamine on two days that were separated by two days of placebo administration, while they gave two doses of 15 mg of *d*-amphetamine at 7:00 and 10:00 a.m. during the study day. Furthermore, we used only self-rated data (means of three observations) on each of two days. We have previously reported significant correlations between antidepressant responses to *d*-amphetamine found with this self-rating scale compared with observers' behavioral ratings (10).

It is known that *d*-amphetamine acts almost immediately, compared with the longer time required for tricyclic drug effects to appear. The activation, euphoria, and antidepressant responses to *d*-amphetamine are significantly correlated with *d*-amphetamine blood levels (16). It is intriguing that a correlation was found between the two drug responses (amphetamine and imipramine) because much of the variance in response to both compounds has been reported to be accounted for by differences in plasma drug levels among different individuals (16, 25). This raises the question of whether some common factors influencing the blood levels of both drugs (e.g., microsomal enzyme activity, absorption, and distribution) might be present. That a correlation was found between *d*-amphetamine plasma levels and the self-rated response to *d*-amphetamine (16) and between the latter and the observed response to imipramine (this report) may indicate that those patients with only a moderate response to imipramine did not develop sufficiently high blood levels, for example, on the basis of not receiving an adequate therapeutic dose. This possibility suggests the need for clinically available methods for tricyclic blood level determination and for a better understanding of the kinetics of imipramine metabolism.

The amphetamines have been used in the treatment of depression, although their efficacy has not been consistently demonstrated (24–26). However, the individual response is extremely variable (8, 10, 11). In studies with larger groups of patients these individual variations may have accounted for the differences in antidepressant effects of chronic *d*-amphetamine reported by some researchers (27), although not by others (28).

Amphetamines may also have unpleasant side effects—for example, irritability or loss of sleep—which may render more complex the assessment of an antidepressant effect. How sustained the antidepressant properties of *d*-amphetamine are has apparently never been comprehensively evaluated. Since Overall and associates (28) reported the similarity between the antidepressant effects of *d*-amphetamine and those of tranylcypromine (a MAO inhibitor) in depressed patients, apparently no reports of attempts at replication have appeared in the literature. Anecdotal reports indicate that some depressed patients may do well on these drugs, while some psychiatrists use *d*-amphetamine or its congener, methylphenidate, to overcome the lag time in response to tricyclic antidepressants (29).

Low urinary excretion of 3-methoxy-4-hydroxyphenylglycol (MHPG) has been associated with a positive response to imipramine (12, 13, 30–32). Fawcett and associates (12) and Maas and associates (13) have also reported that low treatment urinary MHPG tended to be associated with an antidepressant response to *d*-amphetamine. In a preliminary report on 8 depressed patients receiving *d*-amphetamine (32), low pretreatment MHPG also tended to correlate with an increase in activation response to *d*-amphetamine. MHPG is considered to reflect central norepinephrine metabolism (13). Low pretreatment MHPG excretion is consistent with the norepinephrine hypothesis of depression (33, 34). Schildkraut (30) and Beckmann and Goodwin (31) reported that patients with high pretreatment MHPG excretion values are more likely to respond to amitriptyline. A study similar to ours for amitriptyline may clarify this.

It has been suggested that personality factors contribute to individual differences in *d*-amphetamine response (17, 19). Other factors, such as sleep deprivation, may alter individual responses to *d*-amphetamine (18). In the present study patients who rated themselves as relatively more depressed, anxious, and withdrawn on the MMPI tended to experience less activation in response to *d*-amphetamine. However, in apparent contrast to the report of Kiloh and associates (17), depressed patients in the present study who had higher MMPI hysteria scale scores reported feeling relatively less dysphoric during the amphetamine trial. It may be assumed that the relationships between the amphetamine response and the personality factors are largely chance findings.

A worsening of depression in the morning has been considered a sign of endogenous ("vital," serious) depression in contrast to neurotic depression, which may be worse in the evening (35, 36). Endogenous depression responds better to tricyclic antidepressants than does neurotic depression. Our data do not indicate that a diurnal variation in depressive symptoms correlated with *d*-amphetamine responses.

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Third-Party Payers: To Pay or Not to Pay

BY STEVEN S. SHARFSTEIN, M.D.

Insurance companies have traditionally been wary of providing coverage for mental illness for two reasons: 1) they fear that people would bring a mental illness on themselves or would use treatment for self-actualization, and 2) they fear the risk of providing never-ending treatment for "incurable" illness. The author states that the insurers' fears are groundless but suggests that psychiatrists research the utilization and costs of their treatments in insurance plans collaboratively with the actuaries who determine policy and premiums. Retrospective and prospective criteria for outcome and effectiveness of psychiatric treatment must be developed and applied.

THE EXTENT to which health insurance meets the cost of psychiatric services is limited. In their landmark publication *Health Insurance and Psychiatric Care: Utilization and Cost*, Reed and associates (1) estimated that only 12.5% of expenditures for mental health care in 1968 was paid for by health insurance. Although there is some reason to believe that this particular figure is expanding, it is still estimated to be far below the coverage by insurance benefits for physical illness, which represents over 25% of total personal health care costs. The historical reasons for less coverage for mental health care under health insurance are complex, and major issues today remain cloudy. It is the purpose of this paper to review the difficulties insurance carriers have in "working through" insurance coverage for psychiatric care.

In the early development of health insurance in the United States in the late 1930s there was debate over which services should or should not be covered and the extent of such coverage. One overt concern then and perhaps a more covert concern today regarding mental health coverage was something called the "moral hazard" of the insured. This is defined as any characteristic of an insured person that tends to increase the likelihood and/or the severity of a situation for which the person has insurance. Insurance presupposes that the covered situation is likely to be a chance event uncontrollable by the insured person. If the insured person finds it desirable to develop the condition for which he or she has the insurance, it undermines the whole integrity of the insurance system. A dramatic example of this is setting fire to one's own house in order to collect homeowner's insurance.

Mental illness and substance abuse in our culture are not seen or understood in the same way physical illness is understood. The mentally ill individual is considered more responsible for bringing on himself or herself an anxiety state, depression, or an alcohol problem. Furthermore, there is the cultural stereotype that relatively healthy people seek psychoanalysis for self-actualization or self-fulfillment rather than because they have developed an incapacitating illness that requires treatment. In the 1950s the case for coverage of mental disorders under insurance was set back by the experience of the Actors' Equity: many of the enrolled population promptly went into psychoanalysis after insurance that covered this treatment was provided to them. Today, in the Federal Employees Health Benefits Program, which maintains excellent coverage for nervous and mental disorders, the discussion rages in relation to longer-term treatments as to whether the care is medically necessary and appropriate. More and more confidential information is being required by insurance carriers to ascertain the medical necessity of continued treatment; special concern is voiced about psychoanalysis.

THE ROLE OF THE PUBLIC SECTOR

Mental health services contrast with other health services not only in the fact that they represent a lower proportion of the cost paid by health insurance but also in the fact that a dramatically higher proportion of the costs are paid by the public sector—chiefly state and local governments. Two-thirds of all mental care costs come from this sector, compared with less than one-third for all other health care. The public institution and the traditional custodial care provided in public institutions have fixed an ugly image of mental health care and treatment in the public eye. Despite recent developments many people feel that serious mental illness is incurable and drains public and private resources of society. The issue of public support

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for the lion's share of mental treatment is also related to the fact that different types of mental illness are related to social class. Studies associating the most severe pathologies with the lower social classes maintain the poorhouse image for treatment of the mentally ill.

Thus at the one extreme private insurance companies are wary of mental coverage because of the "moral hazard" of people bringing a mental illness on themselves or seeking self-actualization or self-fulfillment through mental treatment, and at the other extreme they are wary of the thousands of "incurables" who languish in custodial institutions. The insurers fear that either extreme will bring financial ruin to an insurance program.

EXPLODING THE MYTHS

Since the 1950s dramatic developments have brought mental health care into the mainstream of general medical care. These changes include the development of psychiatric units in general hospitals, the effective use of psychotropic medications, and the shift in locus of care away from the remote long-term custodial institutions back to the community. Data from recent studies of health insurance plans and the community mental health centers program indicate that the fears of insurance companies about the utilization and costs of mental treatments are groundless. Data from the Blue Cross/Blue Shield federal employee plan, for example, with its very generous coverage for psychiatric conditions (with cost sharing), indicate that the proportion of mental and nervous to total health benefits has stabilized at around 7.5% for the past 5 years (2). Data from the provincial program of medical care insurance in Canada indicate that the proportion of psychiatric care to total medical payments is between 1.4% and 5.4% in various provinces, depending on the availability of psychiatrists (2).

Furthermore, data from community mental health centers indicate that long-term care is much more the exception than the rule. The 1973 data from community mental health centers in Kentucky show that 87% of outpatients were seen for 10 or fewer visits per episode of care (3). Even in state and county mental hospitals, most inpatient care is now for less than 30 days; these hospitals thus meet the definition of acute short-stay hospitals (3).

The compelling accumulation of utilization and cost information is forcing both private health insurance companies and national health insurance planners to reassess the role of mental health coverage. Major medical insurance coverage almost always includes a short-term psychiatric hospitalization benefit (30–45 days/year) and limited short-term outpatient visits (10–30 visits/year). An issue of concern, however, is the extent to which mental care can be reviewed in a similar fashion to physical care, especially in the outpatient sector. The insurance industry must provide more evidence of the "cost legitimacy" of the benefits it pays. The rapid escalation in health care costs endangers the accessibility to all health care. Therefore, third-party payers insist that the provider of care demonstrate that every dollar spent is for medically necessary and appropriate care.

ACCOUNTABILITY OF MENTAL HEALTH COVERAGE

Most inpatient psychiatric care can be reviewed on the basis of admission criteria and by length-of-stay norms. It is in the outpatient sector that the review of psychiatric care becomes more problematical. This is especially true in the case of intensive outpatient psychotherapy and psychoanalysis. The following issues are often raised:

1. What is the validity of the diagnosable condition? Is it a medical condition? The fact that most outpatients superficially seem to function well in work and with their family is inconsistent with the medical model, which assumes incapacitation from disease and assumption of the patient role.
2. Is the treatment offered the most cost effective? Would shorter term individual treatment or group treatments produce an equally good result?
3. What are the qualifications of the person who provides the treatment? Since there is no Board certification for intensive psychotherapy, who is qualified to conduct this technically demanding treatment? Why is a medical degree necessary for talking treatments? Which nonphysicians are qualified?
4. To what degree does the communication of information from the person who provides the treatment to the insurance carrier compromise the privacy of the therapist-patient relationship? Most mental health professionals insist on a need for strict confidentiality in order for psychotherapy to proceed. If there are many questions about the medical necessity of treatment, its length and intensity, and the qualifications of the provider, how much and how often must such information be provided to an insurance company or other third-party payer? How much peer review should be expected for this treatment? What about prior authorization? When does providing the information interfere with the treatment itself?
5. Finally, in any open-ended benefit structure, the question remains at what point you can say that treatment has gone far enough and should go no farther.

Quality assurance efforts and the review of the necessity of continued treatment presuppose a model that may be antithetical to psychotherapy. The law of diminishing marginal return applies to most medical conditions: this is to say that in any given medical treatment a (usually predictable) point is reached where an additional increment of the medical treatment will not provide an additional increment of health. With psychotherapy, however, it is unlikely that a predictable increment of health will be evident with each increment of treatment. The phenomena of

resistance, regression, and negative transference lead to a wave curve of treatment progress. An increment of treatment could lead to a decrement of mental health over the short term; only much later will the progress toward health resume. How, then, is one to measure progress in the course of a treatment that defies the law of diminishing marginal returns? Also, how can the insurance companies and other third parties be educated about the differences in psychiatric treatments without completely throwing out the medical model?

We have little experience in the review of psychiatric care. Only recently have we been able to demonstrate the cost stability and predictability of comprehensive mental coverage under health insurance, and this is not enough. Research on the cost-effectiveness issue that is related to health outcomes and is convincing is necessary. A model for such research was provided in West Germany 20 years ago.

WEST GERMAN INSURANCE STUDIES

Duehrssen (4, 5) and her colleagues at the Central Institute of Psychogenic Illness of the Berlin General Health Insurance Office did an extensive follow-up of patients who had had analytic psychotherapy or psychoanalysis. They developed a system for evaluating patients at the onset of treatment and at the end of treatment and then followed up on nearly 1,000 patients after 5 years. The criteria used for evaluating patients in this study included 1) a precise description of the symptoms of illness as well as its duration, 2) data on utilization of general health care, especially in-hospital care, 3) data on the work capacity of the individuals, and 4) self-evaluations of the treatment by the patients themselves.

The 1,004 study patients who underwent individual analytic psychotherapy or psychoanalysis had had an average number of 100 hours of treatment. Twelve percent ended treatment prematurely, 12% continued privately after their insured limit of 200 visits ran out, and 10% said they wanted further treatment after 200 visits but could not afford further treatment. The researchers could not find 101 of the study patients at 5-year follow-up, 13 had died, and 845 of the remaining 890 patients were evaluated, providing an extremely high response rate. Only 45 of the 890 patients refused to provide follow-up information. In 5 years 647 patients were followed up by direct interviews, 104 returned detailed questionnaires, and 94 had home visits by a social worker.

In terms of outcome, 13% of the patients were judged to have had at least one relapse during the 5-year follow-up period. The most interesting statistic was that of 845 patients, the hospital rate was .78 hospital days per year, which compared with a pre-treatment average of 5.3 days per year and a general average for the insured population of 2.5 hospital days per year. This included hospital days for any illness, not just mental illness. Prognostic measures built in at the onset of treatment also had a high degree of validity and reliability: patients wtih favorable prognoses had excellent outcome measures at the termination of treatment and this held true at 5-year follow-up. There was also a very high degree of patient satisfaction: 81% felt strongly at 5-year follow-up that they had been helped by the treatment.

Although there were some methodological problems with this study, it is clear that follow-up had been conducted on a large number of patients with a sufficiently long interval between treatment and follow-up. This kind of research helped both preserve and extend the national health insurance benefit for the mentally ill in West Germany and led to a more efficient and effective prior authorization and peer review system throughout that country.

WHAT CAN PSYCHIATRY DO?

Psychiatry is a part of medicine. Together with other physicians, psychiatrists must promote the value of their treatments to the public and must be accountable for the costs. Continued study of utilization and costs in both private and public insurance plans is necessary and should be done collaboratively with the actuaries who determine policy and premiums. Working with insurance carriers also means mutual education: psychiatrists could decide on the rationale for particular treatments, especially as they depart from the "medical model." Standards for claims reviewers must be developed so that there is a system of accountability that allows for increasing intensity of review based on flagging criteria and leading to timely peer review. These standards must include adequate safeguards for confidentiality.

Peer review must be more than a ratification of the "usual and customary." It must begin to develop a body of knowledge similar to case law and then be able to apply this knowledge around the country. Outcome and effectiveness criteria must be developed and applied—not only retrospectively but prospectively, as it was in the West German studies. Both the insurance industry and psychotherapists have an interest in demonstrating cost savings as a result of psychiatric treatments. It is astonishing that so little has been done to demonstrate such savings in the United States to date (6–8). What has been done is tantalizing but not so overwhelmingly persuasive as to overcome long-held beliefs on the uninsurability of mental illness. As a result, generous national health insurance coverage for psychiatric disorders in in doubt. As treatments depart from the perspective of the medical model it is essential to make extra efforts to justify their necessity and cost effectiveness. If the evidence on cost effectiveness is unconvincing, national health insurance in the United States will not pay for treatment for mental illness. This kind of care is costly. Who will pay?

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Foundations' Fund Prize for Research in Psychiatry

The American Psychiatric Association is inviting submissions for the second annual Foundations' Fund Prize for Research in Psychiatry.

Candidates for the prize should be citizens of the United States or Canada and should be nominated by a sponsor. Sponsors should be members of the American Psychiatric Association. Members of the prize board are excluded from submitting nominations.

The *sponsor* should submit a supporting letter justifying the nomination in detail and summarizing the nominee's research accomplishments.

The *nominee* should submit the following:

1) An up-to-date curriculum vitae,
2) An up-to-date bibliography,
3) A group of representative and thematically linked papers or books published (or accepted for publication) in English and dated within 10 years of the deadline for submission, and
4) A summary statement written by the nominee that emphasizes the principal theme or themes running through the work and its internal cohesiveness and consistency.

Six copies of each entry should be submitted to Joel J. Elkes, M.D., Chairperson, Foundations' Fund Prize Board for Research in Psychiatry, American Psychiatric Association, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but cannot be returned. The award will be presented at the Convocation of Fellows at the APA annual meeting in May 1979.

The deadline for submission is December 1, 1978. Any entry received after that date will automatically be entered for consideration for the 1980 award unless withdrawn by the nominator or candidate.

Am J Psychiatry 135:10, October 1978

Combat Neurosis in Inner-City Schools

BY ALFRED M. BLOCH, M.D.

The author studied 253 classroom teachers who were referred for psychiatric evaluation because of varying degrees of psychological stress and physical trauma. These teachers had experienced actual assault or the threat of assault in inner-city schools. The author evaluated these patients' ego functioning, attitudes, current life situations, and the presence of any predisposing factors. On the basis of these evaluations and using guidelines established in studies of combat neurosis, he recommends that individuals who experience severe occupational stress be provided psychological training to prepare them to handle stress, constant support of and access to people in administrative positions, and a mechanism for crisis intervention.

THE CONCEPTUAL FRAMEWORK provided by definitive studies of military casualties more than 30 years ago by Grinker and Spiegel (1) and more recently by Glass (2) and others (3–6) established clearly the causal relationship between continued environmental stress and strain and symptoms of psychological and psychophysiological manifestations of traumatic war neurosis, or "combat neurosis." I have observed a more recent form of combat neurosis. Classroom teachers in inner-city schools are seeking treatment for symptoms of continual psychological stress and physical assaults that result in temporary and sometimes total decompensation. A group of these "battered" teachers form the data base of this study. These people face environmental stresses that have been and still are escalating.

Since 1972, classroom murders have increased 18%, rapes 40%, robberies 37%, and physical assaults on teachers 77% (7). According to *Newsweek* (8), the Senate Subcommittee on Juvenile Delinquency reported that during the academic year 1975 vandalism and violence in schools continued to increase: annual destruction of school properties exceeded $600 million, and 70,000 classroom teachers reported serious injuries from physical assaults by students. Many more injuries are probably never reported. For a variety of political reasons, the quantitative and qualitative extent of such physical assaults is not widely acknowledged.

Unless powerful defense mechanisms are used, the response to danger is fear, the most potent source of arousal of the autonomic nervous system. Changes are effected in respiration, blood pressure, and catecholamine secretions (increased plasma corticosteroid concentrations and urinary steroid excretions). Sustained arousal of hypothalamic action systems channeled to differenct target organs has been shown to culminate in such diseases as hypertension, peptic ulcer, and diabetes mellitus (9–11). Many of the psychological symptoms evident in the teachers who seek treatment are also analogous to those observed in combat neurosis: emotional tension (anxiety, insecurity, nightmares, excessive startle response, phobias), cognitive impairment, and conversion symptoms.

Although there are many studies of various causative aspects of juvenile aggression leading to violence in schools, these only illustrate Holt's concept that "the school is the battleground of society" (12). I am aware of no comprehensive study of the effects of violence and its psychological and psychophysiological sequelae in classroom teachers who have been its target. These people are on one of the front lines of society. Others on the front lines include the police, but they have chosen to be there, they are better equipped physically and psychologically for such duty, and they possess devices for self-protection and implementation of their will.

PATIENTS

The patients selected for this study were 253 classroom teachers (158 women and 95 men) from Los Angeles inner-city schools. All of these teachers were victims of varying degrees of psychological stress and physical trauma. Evaluated during the period of October 1971 through June 1976, these patients were white, black, Mexican-American, and Oriental and had taught in inner-city schools for periods ranging from 3 months to 14 years. Their average age was 42, with a range of 23–61. They were referred to me for psychiatric evaluation by a variety of sources, including other physicians, coworkers, union representatives, and attorneys representing them in Workers' Compensation applications.

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The evaluations of these patients revealed an evolving pattern dramatically similar to that seen in early studies of combat neurosis, with implications for diagnosis, treatment, and, more importantly, prevention. The teachers themselves referred to the schools they worked in as the combat zone. Therefore, I used studies of combat neurosis (1–6) as guidelines in assessing the results of the psychiatric evaluations of these patients and in testing the patients.

Before each psychiatric evaluation I reviewed each patient's medical record. I then evaluated the patient's ego functioning, attitudes, current life situation, and the presence of any predisposing factors. Each patient routinely completed a Minnesota Multiphasic Personality Inventory (MMPI), the Bender Gestalt Perceptual Motor Test, and the Beck Inventory of Depression. If there was an indication of organicity or psychosis, projective testing was also performed. Testing was also used as an indicator of progress in treatment.

SIGNS AND SYMPTOMS

Review of Medical Records

Because these teachers' employment contract provided medical coverage (usually with the Kaiser Permanente Medical Group), a clear, well-documented chronological medical record was sent to me before my evaluation of the patient. Therefore, unlike in the earlier studies of combat neurosis, I had documentation of the fact that 134 of the 253 teachers had extensive medical histories representing 2–10 years of psychophysiological response to continued stress. These data also allowed a more comprehensive study of symptoms developed during their teaching career due to the stress response of repeated and sustained arousal of hypothalamic action systems channeled to different organs, with resulting cardiovascular, gastrointestinal, and respiratory disorders. Important life stress events and the development of symptoms for which medical consultation was sought could usually be correlated.

Clinical Evaluation

Twenty-eight percent of the patients had sustained actual physical assault on campus. They had suffered lacerations, bruises, head injuries, seizures, and deafness. Most of the physical injuries were minor, however. Those who had been attacked without provocation or did not know their assailants seemed to experience the greatest difficulties. The majority of assaults were of this type.

The symptoms described by Grinker and Spiegel (1) of hypochondriasis and conversion phenomena were evident in these patients as complaints of fatigue, weakness, blurred vision, tinnitus, irritability, sensitivity to weather, dizziness, malaise, and a variety of depressive equivalents. Almost all of the patients had some psychophysiological manifestations of long-term stress. I frequently observed during the interview that mental reliving of stressful events reproduced the psychophysiological responses. Other similarities to patients with combat neurosis included predisposing personality factors, the patients' tendency to focus on somatic symptoms, and their impaired morale and sense of futility. A relatively minor physical injury or somatic complaint often became the unconscious focus of psychological decompensation with ego-syntonic secondary gain. Depending on the particular target organ, the symptoms of these patients represented the entire spectrum of stress-related illness (see table 1).

Psychological testing generally indicated obsessional, passive, idealistic, dedicated individuals who were unable to cope with or understand the violence directed toward them. They were unable to defend themselves or strike back. Unconsciously, these patients identified authority figures as parental figures with idealized attributes of wisdom, justice, love, and protection. The patients' levels of anxiety and depression were consistently high, and they had a tendency to focus on various somatic expressions of anxiety.

Predisposing Factors

Factors predisposing to neurosis in military personnel were applicable to these teachers (13–15). Primarily, these centered around an impaired ability to deal effectively with fear or anger. Almost 80% of the teachers who succumbed to sustained stress were categorized as passive, rigid, and moderately obsessional. They were moralistic and had relatively harsh super-egos. Unable to strike back when they were the target of violence or hostility, they internalized their fear and rage.

It is important to emphasize that none of the patients in this study had received any psychological or physical training to prepare them to face the threat of violence in the schools. Without this basic training they were unable to facilitate optimal autonomic function during crisis or attack (16–18).

Environmental Stress

The continuum of violence directed toward these teachers included repeated threats of murder and/or rape. Actual physical assault and injury occurred, as did theft, arson, and vandalism of their property. Threats of a brutal attack were often more psychologically disabling than the actual event. Campus violence not specifically directed at these teachers included bombing of school buildings, theft and destruction of campus equipment, fights between students and gang members, murder, rape, and the presence of weapons on campus. Student locker searches revealed drugs, dynamite, knives, ammunition, and firearms. The presence of anonymous gang members and campus vagrants in inner-city schools is a major source of violence and has caused continued anxiety to the students and faculty.

TABLE 1
Primary Physical Complaints of 253 Teachers Referred for Psychiatric Evaluation*

Complaint	Number of Patients
Gastrointestinal disorders: burning, cramping, nausea, diarrhea, ulcer, colitis, other	114
Musculoskeletal disorders: functional overlay to injury, backaches, other	34
Respiratory disorders: asthma, frequent bronchial infections, other	31
Headaches: migraine, tension, other	29
Cardiovascular disorders: palpitations, hypertension, coronary artery disease, atherosclerosis, other	19
Skin disorders: neurodermatitis, allergies, other	14
Miscellaneous: hypochondriasis, conversion symptoms, other	12

*Almost all of the patients had symptoms representing more than one system involved in the stress response. Headaches and gastrointestinal symptoms were extremely common.

Many of the patients reported that their classrooms were overcrowded by as much as 75%. These classes often contained a large proportion of violence-prone continuation students, many of whom had police records of juvenile delinquency and detention. The teachers reported that when violence-prone or acting-out students became disruptive in class they were sent to the administrator's office. They usually returned promptly without having been reprimanded. If they assaulted a member of the faculty, they were suspended from school for a few days and then returned, often as heroes to their classmates.

Sixty-eight percent of the patients reported that they had been discouraged from discussing the incident with other members of the faculty. Thus, they were denied an important opportunity for obtaining support and reality testing. The majority of the petitions submitted annually by teachers requesting transfer to less stressful schools were denied. The added stress of no exit from what they viewed as an intolerable situation contributed to the development of symptoms. Psychophysiological complaints increased until many became disabled.

Morale and Leadership

The teachers felt especially demoralized when, in attempting to report an attack, they found the principal to be indifferent or, worse, fault-finding. Many, like rape victims, were made to feel responsible for being assaulted.

After sustaining a prolonged battering from an angry black student wielding a chair as a weapon, a white teacher reported the assault to his principal and was informed, "This [incident] is your fault and reflects your inability to communicate with the minorities." The patient has not returned to teaching. The same teacher had survived an even greater physical assault a year earlier. In that instance, however, he had felt the immediate and complete support of a different administrator. He had returned to his classroom the next day. When asked to subjectively correlate the effect of the lack of administrative support in the more recent incident to his inability to resume his teaching career, this patient unequivocally acknowledged that he would still be in the classroom today if the situation had been handled better. This experience of crossfire is not unique and contributes substantially to disability and the need for psychiatric treatment among battered teachers.

The teachers said that they were usually discouraged from reporting incidents of violence. Although legally required to do so, they received the implicit (and sometimes explicit) message that it was "not in their best interest" to pursue the matter, i.e., that the administrators might consider them unsuccessful. They thus functioned under a two-edged Damoclean sword: the threat to their job security and the ordeal of facing continued classroom and campus violence.

CONCLUSIONS

These teachers had symptoms of posttraumatic or combat neurosis as clinically defined. The relationship of stress to physiological response and physical illness has been well documented in certain diseases. The relationship between stress and psychological depletion has also been studied extensively. Although the psychiatric needs of teachers who succumb to stress still require further definition, I reviewed the effects of trauma and stress on these patients by applying the guidelines used in studying the survivors of war. The consensus of military studies of morbidity following periods of stress was based primarily on the following factors: 1) severity of the event, 2) chronicity of the stimulus, 3) unexpectedness of the event, 4) lack of counterbalance to unpleasant stimuli, and 5) impaired morale. The patients reported on here have shown the same correlation between incidence of symptomatology and factors of stress as was shown in the military studies.

RECOMMENDATIONS

Careful investigation of etiological factors in the problems of these patients indicates that certain prophylactic measures can be taken to diminish or avoid the psychological and psychophysiological breakdowns observed. With appropriate modification, these measures may be applicable to other individuals who experience severe occupational stress (e.g., firemen, policemen, and social workers).

Preparedness

Psychological training to prepare people for stressful situations must take place to minimize the impact of violence. This training could include rehearsing and familiarizing the teacher with potential violent events.

This would develop a qualified rather than a total belief in personal vulnerability and would teach methods to cope with and diminish the anger of violence-prone, acting-out students. The teacher could be taught to understand the language of the streets as well as the games students play. Skillfully handled, one of these provocative games can result in a shared joke that improves morale. Poorly handled, the game can have catastrophic results.

Morale

As in war, morale is a critical factor for teachers who deal with violence. The opporuntity to share his or her experience with another person or with a group and to work through some of the problems should always be available. This would represent an acceptance of the events and consequences of campus situations, reduce the psychic load on the battered teacher, and facilitate individual development, improving morale through constructive attitudes and efforts. "Rap groups" led by teachers with mental health experience could be useful in this situation. If assault or breakdowns occur, leaders with greater expertise must be used.

The teaching staff, especially in schools with high profiles of violence, must be able to believe in the absolute and constant integrity and support of the school and district administrators. The teacher must feel the support of the school administration. Teachers assigned to schools with high profiles of violence should be rotated to less stressful schools after a specified period (perhaps 2 or 3 years).

Students who have attacked a teacher or staff member should not be allowed to return to the same school. Too often, these students return as heroes to the other students after 1 or 2 days of suspension. They siphon the energy of the teachers and obstruct the learning opportunity for the majority of students.

Classroom teachers should have an opportunity to report directly to the school board concerning such issues as adverse school conditions, unfair administrators, overcrowding, and violence. This is most important; it enables the individual to sustain the hope that someone will be made aware, that some change for the better may occur, that there has been some meaning or value to their suffering. They should be encouraged to report incidents of acute or chronic stress rather than discouraged.

Crisis Intervention

A crisis intervention team should be assigned to each school district. This team would consist of two teachers with mental health training who would be supervised by a psychiatrist or a clinical psychologist. Their function would be to defuse crises by implementing open-forum discussions at schools where teachers and students are involved in conflict. The crisis team would offer the immediate and subsequent psychiatric care needed to decathect trauma if a teacher's stress response syndrome is not worked through and mastered. A teacher-oriented walk-in clinic should be available. To be effective, there must be no conflict of interests in the orientation of such a setting.

The problems described in this paper are increasing rather than decreasing. Every effort must be made to minimize the violence or stress itself as well as the impact of that stress on students and teachers. I hope this study and its recommendations represent a step in that direction.

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Patient-Therapist Matching: A Research Evaluation

BY JOHN G. GUNDERSON, M.D.

The author tested the predictive power of 10 hypotheses about the personality qualities of therapists that match with those of schizophrenic inpatients to affect therapeutic course. The personality matching traits predicted with significant accuracy the quality of the therapeutic relationship and even the type of overall clinical outcome. Examination of the data revealed that most of this surprising predictive strength derived from a group of highly intercorrelated personality qualities. Taken together, these qualities reflected a capacity of therapists to be comfortable while being involved with a variety of strong affects in their schizophrenic inpatients. The limitations of this study and its advantages over the A-B typology are discussed.

THIS PAPER describes a research effort to evaluate the validity of 10 hypotheses about personality dimensions of psychotherapists that match well with particular personality characteristics of hospitalized patients, primarily schizophrenics. The hypotheses about patient-therapist matching arose from discussions in a Washington, D.C., psychotherapy of schizophrenia study group.[1] This study derives from the clinical observation that the qualities of psychotherapists who work well with schizophrenic patients are impossible to define without relating such qualities to corresponding characteristics of the patient with whom the therapist is to work. The clinical and theoretical background of this study has been described elsewhere (1, 2).

The best-known effort to distinguish characteristics of good (type A) and bad (type B) therapists with schizophrenics is the Whitehorn and Betz study (3). That study and the many which followed it (4) have attempted to correlate the effectiveness of treatment with the therapist's score on the Strong Vocational Preference Blank. Despite the great amount of work that has been done, the significance and meaning of the A-B typology have remained elusive. The hypotheses tested in the present study differ by focusing directly on clinically meaningful personality dimensions of patients and therapists. The focus on personality dimensions arises from the belief that it is especially valuable to match the therapist's personality with schizophrenic and other very disturbed patients because of the relative importance of the "real" relationship during the critical initial phases of therapy (5). This impression has been confirmed by the Menninger psychotherapy study (6) and, more specifically with schizophrenics, by Rogers and associates (7). The personality dimensions are intended to be qualities that might exist relatively independent of the therapeutic situation.

In clinical practice the subcategorization of schizophrenia is more likely to be done according to personality characteristics than symptoms; that is, patients are more frequently described as suspicious, hostile, or seductive rather than catatonic, hebephrenic, or even as having a chronic versus an acute breakdown. Just as with therapists, the personality characteristics of the patient that are enduring and somewhat independent of the specific symptoms manifest during acute regressions are felt to be especially important in attempting to match the patient with the therapist. Table 1 indicates 10 qualities of patients and the corresponding qualities of therapists that were hypothesized as making for either a good or bad match.

RESEARCH DESIGN

The 10 hypotheses about matching listed in table 1 were translated onto separate rating forms for patients and for therapists (1). On these forms each of the 10 personality dimensions for a sample of patients and their therapists was rated on a 1-to-5 anchored scale. From these ratings a prediction about the quality of the match could be made when the profiles of the patients and therapists revealed evidence of either a mismatch or a particularly good match. In about one-third of the matches no suggestion of either good or poor matching was apparent and no prediction could be made. For matches where predictions were made, these were

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The author gratefully acknowledges the contributions of Arthur Cain, M.D., Stanley Eldred, M.D., David Feinsilver, M.D., Clarence Schulz, M.D., and Kris Mooney.

[1]The participants in the Washington, D.C., study group included Marvin Adland, M.D., David Feinsilver, M.D., John Fort, M.D., Loren Mosher, M.D., Clarence Schulz, M.D., Helm Stierlin, M.D., Lyman Wynne, M.D., and the author.

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TABLE 1
Ten Hypotheses About Matching Personality Qualities of Patients and Therapists

Patient Quality	Type of Match	Therapist Quality
Passivity: submissive, passive, compliant	Good	Activity: energetic, playful, intrusive
	Bad	Passivity: "analytic," unstructuring, impassive
Hostility: irritable, belligerent, assaultive	Good	Comfort with aggression: either as a "Grandma Moses" or a "Napoleon"
	Bad	Lack of this quality: anxious, "chicken"
Paranoia	Good	Externalization: blames, antiauthoritarian, sympathizes with victims
	Bad	Naiveté: ready trust
Sociopathy: addictive, impulsive, manipulative	Good	Political: adventurous, flexible, power conscious
	Bad	Prudishness: conventional, rigid, principled
Fragility (of defenses): sensitive, regression prone, excitable	Good	Gentleness: easygoing, soft-spoken
	Bad	Intense: assertive, loud, boisterous
Seductiveness (involvement with sexual id, body matters)	Good	Grandfatherliness: accepts id material without excitement or judgment
	Bad	Lack of this quality: puritanical or seductive
Depression: sad, helpless, suicidal	Good	Comfort with depression: patient knows grief "inside out"
	Bad	Lack of this quality: responds with sympathy, anger
Hopelessness (prognostic): chronic, incurable	Good	Optimism: charismatic, tenacious, grandiose
	Bad	Pessimism: scientific, selective, realistic
Anxiety	Good	Composure: contained, reasonable, stable
	Bad	Frenzied: frenetic, disorganized
Dependency	Good	Comfort with dependency: independent, has mastered separation
	Bad	Lack of this quality: lonely, needy, guilty

correlated with the patients' subsequent clinical course.

To help isolate the matching variable from the many factors that influence clinical course, this study used a sample of patients who changed therapists during their treatment. This design became feasible because of a data base provided by a survey of the psychotherapy experience at McLean Hospital in the 1960s.[2] From the over 500 psychotherapy cases that were surveyed, there was a subsample of 50 patients who had 2 or occasionally 3 therapists during the course of their hospitalization. About 85% of the patients in this sample had been diagnosed as schizophrenic. The usual reason for a change of therapist was because problems had arisen in the psychotherapy work with the first therapist. Occasionally there were changes because the first therapist had become unavailable. As part of the hospital survey, when therapists finished working with a patient they were asked to evaluate two factors: the clinical outcome and the quality of the therapeutic relationship.

Ratings on the therapists' dimensions were done independently by two experienced clinicians (Drs. Stanley Eldred and Arthur Cain) who knew all of the therapists involved. Ratings on each patient's personality dimensions were made retrospectively by the therapist who had worked with the patient. The numerical scores provided by the ratings of patients and therapists formed the basis for research predictions as to the quality of the patient-therapist match. These predictions were made without any knowledge of the identity of the patients or the therapists, the sequence in which the two or three therapists worked with a given patient, or the frequency of outcome type (in terms of success or failure).

[2]This work, titled "Psychotherapy at McLean," was prepared by Dr. A. Cain in 1973 and is available from him at McLean Hospital.

RESULTS

Interrater Reliability

Previous reports on the patient and therapist scales have indicated that clinically experienced raters can achieve reasonable reliabilities (1). In the present study the two trained raters evaluated all therapists independently, and their mean score was used for the actual study. The interrater reliability of ratings in this study was .6 (intraclass correlation coefficient).

Predicting Matching Success

The first hypothesis to be tested was that the scores would accurately predict that a particular patient-therapist match would correlate with the clinical outcome. Data were available on 49 matches; the predictions were correct for 31 of them ($p=.044$, binomial test). Because other treatment and situational factors certainly contributed to the overall outcome, the predictions were then compared with the therapists' judg-

TABLE 2
Accuracy of Predictions and Therapist Ratings of Clinical Outcome and Therapeutic Relationships

Therapist Rating	Predictions		p*
	Accurate	Inaccurate	
Clinical Outcome			
Good	25	4	.00007
Poor	6	14	.979
Total	31	18	.044
Therapeutic Relationships			
Excellent	8	2	.055
Good	13	2	.004
Fair	4	0	.0668
Poor	1	4	.969
Total	26	8	.0018

*Binomial test.

ment about the overall quality of the therapeutic relationship. This correlation was felt to test a narrower goal that is more specific to patient-therapist matching. Judgments about the quality of the relationship were available for 34 matches; the predictions agreed with the therapists' judgment of the quality of the relationship in 26 cases (p=.002, binomial test). This shows that the scales were better able to predict the quality of the therapeutic relationship than the overall clinical outcome. The predictions tended to be more accurate when the clinical outcome or the relationship went well than when it went poorly (see table 2). Thus the scales seemed better designed to indicate good matches than to detect poor ones.

Predicting the Effect of Changing Therapists

The second hypothesis to be tested was whether an accurate research prediction could be made on the effect of a switch from one therapist to another. The 2–3 therapists were rank ordered in terms of their likelihood of success with a particular patient without foreknowledge of the actual sequence of the therapists. This allowed an unbiased prediction to be made on the effect of the switch after the sequence of the therapists was made known. Of the 14 cases for which information was available, 11 predictions were correct (p=.031, binomial test).

The Predictive Power of Each Hypothesis

Two methods were used to determine what predictive weight should be given to each of the 10 matching dimensions. In the first method records were made of whether and with what frequency each dimension correlated positively or negatively with the clinical outcome (see table 3). The most accurate predictor was the dimension of the therapist's composure in the presence of the patient's anxiety. This was also the most frequently used predictor. Other dimensions that emerged as having strong predictive power—when there was either good or poor matching on them—were the therapist's grandfatherliness with seductive patients and the therapist's comfort with aggression for hostile, physically threatening patients. That the comfort with depression and the grandfatherliness dimensions both emerged among the better predictive items confirmed the earlier clinical judgments and research impressions obtained from listening to case conferences (1). The dimensions of sociopathy and, especially, of optimism seemed to be poor predictive items. The dimensions of externalizing, gentleness, and comfort with dependency showed very little predictive strength.

The second method of determining predictive weight was by a multiple regression analysis. This analysis confirmed that the variable which accounted for most variance in outcome was the same one, "composure," that predicted outcome best using the first method. The second, third, and fourth variables (optimism, gentleness, and externalization) were all negative and were among the weakest predictors above. This means that reversing the direction of these hypotheses would allow them to add to the predictive power of composure. The fifth component of the regression analysis (comfort with depression) was positive but added very little.

Intercorrelations of the Therapist's Personality Dimensions

There was considerable interdependence of ratings of some dimensions upon each other. Most striking was the high intercorrelation between the dimensions that were strong predictors; for example, composure correlated highly with comfort with aggression (r=.82), comfort with depression (r=.70), and with grandfatherliness (r=.52). The fact that the strong predictors had generally higher overall mean intercorrelations with all other dimensions suggests that they gain their power by being more global or encompassing (see table 2). This was particularly evident with the "comfort with" dimensions (composure, grandfatherliness, comfort with aggression, depression, and dependency). This analysis then supports the conclusion that the "comfort with" dimensions all reflect a global factor in the therapist, and its predictive strength is largely captured in the one therapist dimension of composure.

Despite the generally high intercorrelations, there were some dimensions that maintained considerable independence, e.g., optimism, sociopathy, activity, and gentleness. None of these was a good positive predictor. It is likely that the predictive strength of these dimensions was weakened by their specificity.

Influence of Overall Similarities

It is possible that individual hypotheses are not so important as the overall similarities between the profiles of the therapist and of the patient on the 10 dimensions. To test this, the scores of the differences between the patient and therapist were totaled and then correlated with the clinical outcome. Those 19 matches with the largest differences were no more likely to have a poor outcome (9 matches) than a good outcome (10 matches). Similarly, the 31 matches with the great-

TABLE 3
Predictive Weight of Each Dimension

Dimension	Number of Times Used	Ratio Right:Wrong	p*	Rank Order	Mean Correlation with All Other Dimensions
Activity	13	8:5	.2905	5	.143
Comfort with aggression	13	9:4	.1338	2	.286
Externalization	13	6:7	.7095	8	.176
Sociopathy	5	2:3	.8125	9	.030
Gentleness	28	14:14	.4247	7	.097
Grandfatherliness	10	7:3	.1719	3	.261
Comfort with depression	18	11:7	.2403	4	.273
Optimism	10	3:7	.9453	10	.094
Composure	37	26:11	.0107	1	.250
Comfort with dependency	29	16:13	.3717	6	.234

*Binomial test.

er similarity failed to predict good outcome (17 matches) more frequently than poor outcome (14 matches). If overall similarities or differences in the profiles were important predictors, then a patient having 2 therapists could be expected to work better with that therapist whose profile was more similar to his or her own. This hypothesis was also tested and was not found to be valid. These results suggest that the predictive power of the research instruments derived from the specific clinical hypotheses and not from overall profile similarities or differences as recorded by the scales.

DISCUSSION

The results reported here support the idea that certain personality dimensions in the therapist are important predictors of how well he or she can work with certain schizophrenic patients. The matching dimensions that proved to be the best predictors were as follows:

1. Composed therapists work especially well when matched with schizophrenic patients who are anxious.
2. Therapists judged to be comfortable with aggression work well with hostile patients.
3. Grandfatherly therapists do well with seductive schizophrenic patients.
4. Therapists considered to be comfortable with depression do well with depressed schizophrenic patients.

In retrospect it also seems sensible that the optimism hypothesis proved to be predictive in the opposite direction, i.e., to the chronic patient who has a sense of failure, a charismatic or optimistic therapist may seem intimidating and dysjunctive. In future research we would retain this matching dimension but reverse the predictive direction. The results suggest that paying attention to these personality dimensions might help avoid particularly bad matches, increase the likelihood of good matches, and might provide guidelines to changing therapists when a case is not going well.

The results of both a multiple regression analysis and an intercorrelational matrix suggest that an overall dimension having to do with being comfortable, knowledgeable, and interested in the variety of intense affects of schizophrenic patients (anxiety, anger, sexual feelings, and depression) may be the central quality of successful therapists. This interpretation is supported in a recent study by Geller and Berzins (8), who concluded that A-type therapists may derive their effectiveness with schizophrenics from their greater tolerance for disturbed communications and behaviors. As a general statement, the present study suggests that in the absence of other more specific indications or contraindications, the presence or absence of this quality in a therapist can significantly influence the evolution of a therapeutic relationship and can even affect outcome. It should be emphasized that "comfort with" as used here does not imply distance but applies only in a context of involvement. This quality is one that can be expected to evolve from training, experience, and personal treatment, and this gives credence and direction to programs that intend to develop psychotherapists for work with seriously disturbed patients.

A conservative interpretation of the results is necessary because of the unique patient sample used (i.e., patients who during a long-term hospitalization required a change in therapist), because incomplete information meant that no predictions could be made on 33%–50% of the possible matches, because the validity of therapists as judges of the outcome variables is suspect, and because some therapist ratings were difficult to interpret.

It may be that successful psychotherapy for schizophrenic patients is contingent upon finding a good match between the therapist and the particular patient. The results invite further evaluation of the potential impact of the matching variable in evaluating psychotherapy of schizophrenia. If the positive findings of this study are borne out, the simplicity of ratings and the obvious clinical sense of the concepts used offer definite advantages over the A-B typology for therapists. More importantly, it offers a more rational approach to matching than leaving it to chance alone.

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The Manfred S. Guttmacher Award

The American Psychiatric Association invites applications for its annual Manfred S. Guttmacher Award for 1979. The award is given for any outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or other work, including audiovisual presentations (films, etc.), submitted to or presented at any professional meeting or published during the period from November 31, 1977, to December 1, 1978. A plaque and an honorarium of $500 will be presented to the award recipient at the Convocation of Fellows at the 1979 APA annual meeting in Chicago, Ill. Please note that the award recipient is now required to give a lecture at the annual meeting.

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"No-Commitment Week": A Feasibility Study

BY ISRAEL M. ZWERLING, M.D., PH.D., HOPE R. CONTE, PH.D., ROBERT PLUTCHIK, PH.D., AND TOKSOZ B. KARASU, M.D.

To determine the extent to which involuntary hospitalization is overused, a "No-Commitment Week" was set aside, during which emergency room psychiatrists committed only patients in absolute need of hospitalization. Compared with the week before and the week after, there was no significant difference in the number of patients committed during No-Commitment Week. The authors propose replication of the study on a larger scale but suggest that decisions about involuntary hospitalization in public mental hospitals are the result of societal attitudes, which will be subject to change as long as society itself continues to change.

THIS REPORT addresses the controversy concerning involuntary commitment of patients to psychiatric hospitals, which continues to divide psychiatrists from each other and the mental health system from the judicial system. At one pole are those who argue that psychosis is an illness that permits a reliable diagnosis by appropriately trained physicians; that psychotic patients present a high risk of behavior that is dangerous to themselves or to others; that psychotic patients, precisely because of their psychoses, are frequently incapable of recognizing that they are ill and need treatment; and that the judgment that a person is psychotic and dangerous, and therefore in need of treatment, with or without consent, is one which a psychiatrist must be permitted to make without interference by any societal regulation or control beyond concurrence by a responsible member of the family of the patient.

At the other pole are those who argue that involuntary hospitalization represents a deprivation of liberty; that whatever the rationale for its justification it must remain, along with any other deprivation of liberty, an issue to be decided by the societal agency established for this purpose, i.e., the judiciary; and that, in any event, psychiatric diagnoses and the prediction of dangerousness by psychiatrists are highly uncertain and cannot be the sole basis for so harsh an action as involuntary hospitalization. That this controversy continues to be unresolved is eloquently attested to by the fact that 18 states[1] are currently revising or have in the past four years revised their statutes governing commitment.

The focus of this study is on involuntary commitment resulting from the certification by two psychiatrists that a person is mentally ill, dangerous, or both and in need of treatment. In reviewing the literature, it becomes apparent that many factors unrelated to the severity of a patient's illness come into play in the decision to hospitalize a patient on an involuntary basis. For example, there is a greater likelihood of involuntary hospitalization of individuals who have a history of psychiatric hospitalization and whose socioeconomic status is considerably lower than that of the interviewing physician (1). The likelihood is also greater for those individuals who appear at the hospital during weekends and evenings rather than during weekdays, times when the decision makers are usually residents rather than staff psychiatrists and when many of the family and community resources cannot be mobilized to aid the patient (2).

Perhaps the single most important determinant in the decision to involuntarily hospitalize a patient is the psychiatrist's perception of his or her dangerousness to others or to himself or herself. Rubin (3) reported that,

> Approximately 50,000 mentally ill persons per year are predicted to be dangerous and preventively detained. . . . In addition, about 5% . . . of the total mental . . . hospital population of the United States . . . are kept in maximum security sections on assessment of their potential dangerousness. (p. 397)

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[1]In the past four years, the following states have substantially revised their civil commitment laws: Arizona, Colorado, Florida, Hawaii, Michigan, Mississippi, North Carolina, Ohio, Pennsylvania, South Dakota, Virginia, Washington, West Virginia, and Wisconsin. In addition, Alaska, Illinois, Louisiana, New Jersey, and Oklahoma are considering substantial revisions; and in the following states, federal (f) or state (s) courts have invalidated all or major portions of the state civil commitment law in the past four years: Alabama (f), Hawaii (f), Iowa (f), Kentucky (f), Michigan (f), Nebraska (f), North Carolina (s), Pennsylvania (f), South Dakota (s), West Virginia (s), and Wisconsin (f).

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Ennis and Litwack (4), however, present ample evidence to support the notion that violence toward either self or others is vastly overpredicted and that judgments made by psychiatrists are not sufficiently reliable or valid to justify nonjudicial hospitalization.

There can be little doubt that commitment laws and the machinery for involuntary hospitalization are open to overuse. Perr (5), to cite one recent example, reviewed the cases of 9 hospitalized psychiatric patients. In each case, an independent psychiatric review was requested by a private attorney, and results indicated that in 3 of the 9 cases mandatory hospitalization was not justified.

However, with the exception of extremists such as Szasz (6), who deny the concept of mental illness in its entirety, most concerned individuals would agree that there are situations when commitment of patients is necessary. There is also general agreement (7–10) that voluntary treatment is preferable to involuntary commitment under most circumstances. There is at the same time, however, the acknowledgment even by the most zealous civil libertarians that there are situations in which medical certification may be justified, for example, when a patient who is considered *imminently* dangerous to himself or to others refuses voluntary hospitalization or when a patient's very illness renders him incapable of making the judgment that he requires treatment. Nevertheless, as Ennis and Litwack (4) stressed, medical certification should authorize involuntary hospitalization only for the limited time needed to institute judicial proceedings.

The controversy over the issue of involuntary commitment centers, therefore, mainly on ways to ensure that the civil rights of psychiatric patients are not curtailed while at the same time satisfying the psychiatric profession's responsibility to provide care and treatment for those individuals who may be a danger to themselves or others or whose mental state is such that it is impossible for them to make a judgment concerning the need for hospitalization. It seems obvious that every effort should be made to obtain the patient's consent if hospitalization is deemed necessary. However, the limited amount of time usually available for a psychiatric evaluation in a busy psychiatric emergency room often precludes an adequate discussion with the patient of his or her problems and the reasons for hospitalization. Although administratively cumbersome, it is generally quicker to commit.

With these issues in mind, we undertook a pilot experiment at the Albert Einstein College of Medicine. The week of September 17–23, 1973, was designated "No-Commitment Week." The project was a study to determine whether it was possible to decrease substantially the number of involuntary admissions to Jacobi Hospital and Bronx State Hospital while at the same time providing appropriate patient care. This paper reports the procedures followed, the difficulties encountered in establishing such a week, the problems of implementation, the results obtained, and the questions that emerged.

METHOD

Jacobi Hospital is part of Bronx Municipal Hospital Center, which is affiliated with the Albert Einstein College of Medicine and contains a psychiatric emergency room and three inpatient wards, serving approximately 100 patients. Bronx State Hospital (now the Bronx Psychiatric Center) is a state facility serving approximately 700 inpatients and is also affiliated with the Albert Einstein College of Medicine. It also has extensive outpatient and satellite facilities. Each of these hospitals has its own admission office from which patients may be directly admitted. In addition, a number of patients are admitted to Bronx Psychiatric Center after having been evaluated at Jacobi Hospital and certified as mentally ill by two psychiatrists.

Before the project began, a detailed protocol was set up and efforts were made to notify all relevant staff members that for the stated one-week period commitment procedures would be altered. The basic plan was for the examining psychiatrist to try to persuade all patients believed to require inpatient treatment to accept voluntary hospitalization. Should the patient refuse voluntary hospitalization, the psychiatrist would then offer one or more of several alternatives to hospitalization: crisis intervention, day hospital, outpatient clinic, and home care service. Should the patient refuse these also, the psychiatrist was instructed to discharge the patient against medical advice, unless he believed that the patient was in absolute need of hospitalization. The guidelines for such an absolute need were as follows: "1) the patient has made a serious suicide attempt, is depressed, and still expresses a desire to kill himself; or 2) a patient has committed a serious homicidal or assaultive act and is concurrently diagnosed as psychotic; or 3) a patient has a serious disorganization of thought and behavior, has no place to go, and is unable to take care of himself." If the examining resident psychiatrist believed any one of these three criteria was applicable for a given patient, he or she was obliged to call one of four senior psychiatrists who were alone empowered to commit the patient. The "on-call" schedule for the four was posted in the admitting rooms of both hospitals, and the unit chiefs in each hospital were instructed that they were not to accept any patient during that week unless one of the four senior psychiatrists was a cosigner of the two-physician certificate. It was hypothesized that by making requirements for involuntary hospitalization more stringent the number of patients who would have been committed under usual circumstances would decline.

RESULTS AND DISCUSSION

Table 1 summarizes the data concerning the disposition of all patients who came to the admitting rooms of the two hospitals in the week before, the week of, and the week after the experimental period.

TABLE 1
Disposition of Psychiatric Patients

Disposition	Week		
	Sept. 10–16 (N=146)	Sept. 17–23 (N=153)	Sept. 24–30 (N=133)
Home	86	95	76
Bronx Municipal Hospital outpatient department	4	4	2
Other outpatient clinics	9	15	5
Jacobi Hospital wards	20	15	10
Bronx State Hospital wards	6	8	10
Other psychiatric hospitals*	6	10	10
Returned to emergency room	2	0	0
Walked out	5	5	8
Held overnight in emergency room	4	1	0
Private physician	0	1	0
Alcohol or drug programs	2	1	1
Crisis intervention	1	1	4
Medical emergency room	0	2	5
Other**	1	5	2

*Patient was out of district.
**Includes men's shelter, police, and disposition not recorded.

TABLE 2
Inpatient Admission Category

Week	Type of Admission		
	Voluntary or Informal	Involuntary	Court Remand or Emergency
Sept. 10–16			
Bronx Municipal Hospital	14	2	4
Bronx State Hospital	2	4	0
Total	16	6	4
Sept. 17–23			
Bronx Municipal Hospial	9	3	3
Bronx State Hospital	5	3	0
Total	14	6	3
Sept. 24–30			
Bronx Municipal Hospital	8	1	1
Bronx State Hospital	4	6	0
Total	12	7	1

The distribution of patients admitted to the wards of the two hospitals in the three time periods is summarized in table 2. There were no significant differences in the number of patients committed to the Bronx State or Jacobi Hospital wards during No-Commitment Week; these numbers are well within the normal range of week-to-week fluctuation at both hospitals.

This result was somewhat contrary to what we expected. The short period of time involved, one week, is not sufficient either to generate adequate numbers of patients for definitive results or to provide the many staff persons involved sufficient experience to insure complete familiarity with the protocol and to guarantee smooth implementation of the alternative dispositions when these are accepted by patients. For example, the senior psychiatrists said that in a small number of cases they would have made a greater effort to have patients who were committed accept crisis intervention services if they had had more confidence that the two crisis intervention teams could be relied on to immediately assume responsibility for the patients.

It may, of course, be hypothesized that the senior psychiatrists were themselves too locked into traditional criteria for commitment to permit any change, but given the focus of this study and the diligence with which each individual instance of an involuntary hospitalization was handled, we believe this simply was not the case.

In our opinion, the factor by far most responsible for these results is the resistance of public mental hospitals, perhaps in all cases but very markedly in the case of teaching hospitals, to admitting patients. Wilder and Karasu (11) recently described what they refer to as "the games institutions play," i.e., the devices employed to legitimize not admitting patients. Conversely, it has also been our observation that as soon as the census of a public mental hospital falls to levels that begin to provoke discussion of its not being needed—of its being phased out and closed—admissions increase and the census stabilizes or even rises. It would therefore be essential, in the planning of a future large-scale study, to take into full account the many nonmedical factors that bear upon hospital admission criteria.

Still another factor that surfaced during the present study as a potential danger in future studies is the geographic distribution of mental health resources in an area. Wilder and Karasu (11) were particularly incisive in their description of the "where-do-you-live" ploy, pointing out that a favorite device in dodging the issue of involuntary hospitalization of a floridly psychotic patient who appears to require treatment but refuses admission is to establish that the patient lives outside the catchment area or district for which the hospital is responsible. A total of 26 patients in our study were referred to other hospitals during the 3-week period under review—more than were involuntarily admitted to the Bronx Municipal and Bronx State Hospitals. It is readily conceivable that the data we have presented may have been significantly altered if the areas of residence of these 26 patients had fallen inside the boundaries of the two hospitals concerned.

Finally, a number of forensic issues were raised in the course of this study for which we can provide no answers and which would require serious attention in a more comprehensive study. To begin with, the legality of the very concept of "no commitment" was called into question by the residents covering the emergency room. What would be their position, they asked, if in the face of their professional judgment that a patient required involuntary hospitalization and in the face of a state law empowering them to commit such patients, they nevertheless refused and an unfortunate outcome resulted? Second, the issue of the validity of a voluntary consent for hospitalization was sharply raised. As

pointed out by Schwitzgebel (12), administrators and therapists often assume, quite incorrectly, that patients adequately understand consent forms. He cites, for example, a study by Palmer and Wohl (13) of 40 patients who signed voluntary admission forms for entry into a public mental hospital. Ten days after admission, 24 of the patients were unable to recall ever signing the forms, 14 did not recall the content of the form, and 2 patients denied having signed it. Schwitzgebel proposed a contractual rather than a consensual model for the protection of the rights of patients. It would appear, however, that a signed contract with an institution would be no more or less valid in terms of a patient's ability to understand what he or she has signed than would a signed voluntary consent form.

CONCLUSIONS

We were left with the impression that, at least in a university affiliated municipal or state mental hospital unit, the rigorous application of the most restrictive standards for involuntary hospitalization is not likely to produce a significant decrease in the commitment of patients. To the extent that there may remain the fantasy among civil libertarians that psychiatrists lurk in dark corners waiting to grab unsuspecting victims and commit them to extended stays in mental hospitals, this point should be emphasized. This study patently merits replication on a larger scale, perhaps statewide, for a longer period of time (perhaps a minimum of a month) and with adequate preparation of all staff members with regard to the issues concerned. It is possible that more informative data may derive from such a study. Our impression, however, is that decisions concerning the treatment of deviance in a society result from society's broadest attitudes regarding man's obligation to man and that a resolution of the question of involuntary hospitalization of mentally ill persons will be subject to change as long as society itself, and the structure and organization of its social roles, continues to change.

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OPINION AND COMMENT

The Internship Year: A Negative View

BY SEYMOUR L. HALLECK, M.D.

The decision to add a fourth year of training with emphasis on a primary care internship experience has created serious problems for most psychiatric residency training programs. The logistic problems involved in having a department of psychiatry develop an internship program are formidable. The experiences being provided are less than optimum. Conceivably, the logistic and quality uncertainties that now characterize some PGY-I programs may be a factor in discouraging medical graduates from entering psychiatry. The reasons for moving to an internship experience are also questionable. There are no data which suggest that the new training will produce better psychiatrists. Nor can the case for the internship be supported by past experience or an analysis of the educational needs of today's psychiatrist. Dr. John Romano comments on the author's presentation.

I AM TROUBLED by the decision to require a fourth year or internship year of training in psychiatry for the following reasons. First, the decision was not made on the basis of data that supported a need for an internship requirement. Second, it has created formidable and as yet unsolved logistic problems for training programs. Third, some of the arguments used to defend the decision have been based on emotion and ideology rather than on a rational approach to meeting training needs.

THE LACK OF DATA

The failure of our professional organizations to generate or to examine data on the need for an internship year before making a decision to add that year was especially unfortunate. We do not know if those residents who were trained during a period when they were allowed to omit the internship experience and who elected to do so eventually became less, equally, or more competent psychiatrists than those who took an internship. Some anecdotal material did appear in the literature which suggested that residents who had not completed internships were having difficulties in caring for their patients (1). During the early seventies, however, accelerated programs that deleted even more years of training than simply the internship year reported that their rapidly trained residents were doing as well as or better than others (2, 3). My own informal effort to evaluate performances of residents with and without internships at the University of North Carolina indicated that those without internships were doing somewhat better. Of course, neither studies of a relatively elite group of trainees in a specialized program nor the observation of a single psychiatric training director can be viewed as significant data. My point here is simply that the limited information we did have at the time did not support the need to create a fourth year. It would not have been too difficult to generate more useful data. By 1973 there were large cohorts of doctors with and without internships who had received identical training in psychiatry, and the performance of these two groups could have been compared.

LOGISTIC PROBLEMS

The logistic problems created by the decision to put the internship requirement into effect on July 1, 1977, have been formidable. The new requirement comes at a time when federal funding for psychiatric training is being drastically curtailed. For many programs, alternative sources of funding are not available. Some programs have had to deal with this issue by reducing the number of residency applicants they accept annually. Further, implementation of the internship experience is being required at a time when the percentage of medical students applying for psychiatric training is

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declining to a figure that may be as low as 4%. It is possible that the prospect of an additional year of training, often of dubious quality, may have been a critical factor in discouraging some graduates from applying for psychiatric residencies in 1977.

Major logistic problems developed when psychiatric training programs were required to discover positions for psychiatric trainees in old internship programs or to create positions in new programs. Psychiatrists do not have sufficient power to persuade university and community hospitals to spend time training psychiatric residents in primary care medicine. Departments of medicine, pediatrics, and family practice in medical schools have limited training facilities that are in great demand by residents who intend to stay in primary care specialties. In many instances, these departments have been unwilling or unable to cooperate in providing training experiences for psychiatrists. Only an occasional and highly fortunate psychiatric training program has been able to develop a quid pro quo arrangement in which the psychiatry department trains primary care physicians in psychiatry in exchange for the primary care department's training psychiatrists in medicine, pediatrics, and family practice. The logistic problem is less formidable in some large cities, where understaffed county hospitals welcome the addition of interns, whether or not they are going into psychiatry. However, there is some question as to the quality of the training experience these hospitals will provide.

Most departments have had some success in persuading community hospitals to provide primary care training for psychiatric residents. At this point, we have no way of knowing what the quality of teaching at these hospitals will be. Even if the experience proves to be good, community hospital training may impose additional types of hardships on the trainee. Many of the community hospitals that accept psychiatric trainees as interns are located as far as 30–100 miles away from the psychiatric training site. This is not a major inconvenience for an unmarried resident, but it can be a serious problem for a resident who has a family or whose spouse has a career.

As things stand now, there is little uniformity in the duration or quality of internship experiences in psychiatry. This is a source of great confusion to the applicant. It is also a problem for educators who are seeking to preserve standards of excellence in training and for accrediting agencies that will have to use a wide variety of criteria in evaluating the new programs.

There are also serious logistic problems in some programs insofar as the need to provide medical training for residents in postgraduate year one (PGY-I) will create at least temporary shortages of residents on traditional psychiatric services. Even the best psychiatric training programs require some service functions from their housefellows; but service positions cannot be manned when one-third to one-half of the fellows are on nonpsychiatric services. This is much less of a problem in relatively affluent programs that can recruit larger than usual classes, and some of the more creative of these programs began to provide an internship in July 1976. Other equally fortunate programs took two classes (PGY-I and PGY-II) in July 1977. However, in less affluent programs service responsibilities, particularly in the inpatient sector, may have to be taken over for at least a year by medical students, faculty, or second- and third-year residents. None of these alternatives is desirable, particularly the latter.

Disruptions in formal lecture and seminar schedules created by the internship year are minor but irritating problems. In programs that have created only four to six months of primary care internship training, a large proportion of the first-year residents—as many as one-third to one-half—will be unavailable for formal didactic instruction in psychiatry at any given time. This requires either twice as much teaching on the part of the faculty or a suspension or serious erosion of formal teaching activities in psychiatry during PGY-I.

CONCEPTUAL ERRORS

Some of the reasons for our professional associations initiating or endorsing the internship requirement have been rational. At a time when psychopharmacology is an integral part of psychiatric practice, it is essential that every psychiatrist be knowledgeable as to contraindications and side effects of psychotropic drugs. The more one knows about nonpsychiatric medicine, the more safely and efficiently he will use psychotropic medication. It is also true that some students in the many medical schools which shifted to an elective fourth year chose curricula that did not give them sufficiently broad medical knowledge. Many psychiatric educators worried that our medical school graduates were poorly prepared to assume patient care responsibilities. Adding a required year that includes some internship experience is certainly one way of trying to expand the medical knowledge of psychiatrists.

The internship requirement may also be self-serving; psychiatry is currently under attack from many sources, and most psychiatrists wish to sharpen their identity as physicians. A person or an institution under heavy attack relies on what is most familiar and dear when crucial decisions about third-party payment are about to be made and when other professions are competing with psychiatry for access to third-party remuneration, there is an additional advantage to allying ourselves as firmly as possible with our parent profession. Increased medical training requirements serve to identify psychiatry to the public, to politicians, and to other doctors as a medical specialty.

There have been other arguments put forward to defend the internship requirement that I believe are less than rational. In several years of discussing the internship issue with colleagues, residents, and medical students, I have repeatedly heard efforts to justify the need for the internship on the basis of two arguments. The first is that psychiatrists need to be more like other

physicians and, in particular, to develop modes of practice that are more similar to those of other physicians. The second argument is that much of the future role of psychiatry will involve consultation and liaison services to primary care specialists, and an expanded knowledge of nonpsychiatric medicine is absolutely essential if the psychiatrist is to adequately fulfill these roles. I believe the cases for both of these arguments are weak.

The first argument is extremely difficult to pin down. Its proponents often present it in almost mystical terms. The following type of statement is typical: "There is something about the endurance, the assumption of responsibility, and the performance of manual tasks required by an internship that makes one a better psychiatrist. There is something about the development of a sense of physicianship during the internship year that makes one a more mature psychiatrist." The "something" in these statements is never clearly defined.

A more understandable form of this argument is sometimes presented in terms of the psychiatrist's self-image. Psychiatrists want to be accepted by other physicians as esteemed colleagues. Often they are not. Many physicians do not consider psychiatrists to be "real doctors," and too many psychiatrists agonize over the possibility that their colleagues are correct. This situation has changed little in the 25 years I have practiced. There have always been a large number of physicians who are skeptical of the value of psychiatry and a large number of psychiatrists who are disturbed by that skepticism. One common approach psychiatrists have used to deal with the uncertainty of their role is to demonstrate that they can handle medical and surgical problems as well as other physicians can. An internship experience often assures the young psychiatrist that he is as capable of mastering medical and surgical skills as any other doctor.

I have little doubt that the internship experience may temporarily resolve the image problem for some psychiatrists. The "something" about an internship that is supposedly good for a psychiatrist may be an affirmation of his medical "machismo." If the psychiatrist is too insecure about his identity as a physician, he will either avoid certain medically ill patients or will be tempted to overcompensate for his perceived inadequacies by taking on medical tasks that exceed his skills. However, I doubt that this problem will ever be resolved simply by requiring an internship. Most of our psychiatric educators have had internships, but many are still preoccupied with their image as doctors. Psychiatric educators who are insecure about their own identity as physicians communicate this uncertainty to their students and keep them preoccupied with a medical machismo image. This unfortunate situation is also unnecessary. There is no reason for psychiatrists to feel that their style of practice either isolates them from other doctors or makes them less "medical" than other doctors.

Sometimes the argument that psychiatrists should practice more like other doctors is put in terms of the need of psychiatrists to return to the "medical model." Unfortunately, few who make such statements carefully define what they mean by the medical model. In its most basic form, the medical model simply means practice based on efforts to study pathological activities of the organism, to discover the causes of these activities when possible, and to intervene on an empirical basis when etiology is unknown or to treat the causes of pathological activity when etiology is more apparent. Within this broad definition of a medical model, there are many subtypes (4), which can be defined in terms of the manner in which etiologic variables are considered. In the most reductionistic and simplistic aspect of the medical model, attention is focused almost entirely on etiology related to biological dysfunction. In more sophisticated medical models, the practitioner focuses on multidetermination and the interaction of a variety of biological, psychological, and social factors in eliciting a given malfunction.

There is a certain efficiency and ease involved in adhering to a reductionistic medical model. Much of medical practice today is focused almost entirely on biological variables, and the nonpsychiatric physician rarely takes the time to investigate interpersonal or environmental variables. The physician is especially prone to ignore such variables during his early years of training as a house officer. An internship-type experience in most of the medical settings with which I am familiar will continue to emphasize a reductionistic medical model. This is not appropriate for psychiatrists. Psychiatrists should learn as much as they can about biological causes and treatments of emotional disorders, but training dominated by a model in which the physician is tempted to pay little attention to the complexities of interacting variables in producing emotional disorders is hardly desirable.

Psychiatric practice, or any effort to deal with the emotional problems of patients, requires an emphasis quite different from that currently being taught in most other branches of medicine. Even the capacity to make a good descriptive diagnosis and formulate a rational plan for treatment requires skills that are unlikely to be taught in most medical settings. The psychiatrist must have sufficient sociological perspectives to avoid overdiagnosing certain behaviors as pathological when they are actually manifestations of sociocultural phenomena. He must develop skills in dealing with patients who are uncooperative, resistant to treatment, and often ungrateful. He must develop a model of ascribing responsibility to patients for some of their actions and become skillful in using such a model to elicit behavioral change. He must learn to deal with patients who mimic, either consciously or unconsciously, symptoms of mental or physical illness and to view that mimicry itself as a symptom. He must take the time to develop enough rapport with the patient so that the patient will have sufficient trust to provide details on the phenomenology of his mental illness and the history of its development. Finally, the psychiatrist

must become comfortable in dealing with multiple variables in systems rather than a linear model of causality. These are not skills that are easily learned in an internship.

There is a great deal wrong with the profession of psychiatry, and some psychiatric practice can be careless and sloppy. However, when the psychiatrist is functioning efficiently and systematically attempts to integrate his observations and knowledge of multiple etiologic variables in understanding the patient, he is practicing the best medicine possible. To the extent that psychiatrists practice efficiently, we should not be trying to be more like other doctors. We should be urging other doctors to be more like us.

The argument that an expanded knowledge of nonpsychiatric medicine will make the psychiatrist a better consultant to his nonpsychiatric colleagues is often accepted at face value. Too many psychiatrists believe that our colleagues will more readily communicate with us and accept our recommendations if we can demonstrate that we have considerable knowledge in their own areas of expertise. Perhaps the best way of illustrating the weakness of this argument is to ask ourselves what we as psychiatrists are looking for when we call in an internist or a surgeon as a consultant. This is a role with which I have a great deal of personal familiarity because I run an inpatient unit and deal with many patients who have serious nonpsychiatric medical problems. When I seek consultation, I am looking for help from medical colleagues in areas in which I lack expertise. I want the internist to tell me about the patient's degree of cardiac pathology so I can assess the advisability of using tricyclics. I want the surgeon to tell me about the degree of stress and incapacitation that would result from a particular surgical procedure so that I can assess the manner in which the patient will be able to cope with it. The last thing I ever look for from a nonpsychiatric medical consultant is information about the patient's psychiatric status. I expect my consultants to treat the patient in a humane and compassionate manner, but I am not in the least concerned with their psychiatric skills. Occasionally, eager young consultants will comment on a patient's psychiatric status. Most of the time their assessments are grossly inaccurate.

I suspect that my medical colleagues are looking for the same thing in a psychiatric consultant that I look for in a medical consultant. They want someone who will help them deal with a situation in which they do not have expertise. They want a consultant who will assess the patient's degree of psychopathology and provide practical recommendations as to how to deal with it. Rarely will they welcome a consultant who spends a great deal of time discussing medical issues about which they already possess superior knowledge.

In recent years, an increasing number of internists have complained to me about younger psychiatric consultants who spend more time attempting to demonstrate their medical knowledge than providing psychiatric assessment and recommendations for psychiatric treatment. It is also interesting to note that teachers in some training programs in family medicine seem to develop an easier rapport with behavioral scientists, particularly psychologists, than with psychiatrists. Many family physicians prefer to consult with a psychologist rather than a psychiatrist. There may be several reasons for this phenomenon, but one explanation is that the psychiatrist is too preoccupied with his medical image and is less skilled than many psychologists in providing the family physician with psychiatric assessments and recommendations.

It is my hope that in the future, arguments for creating changes in psychiatric education will be scrupulously examined in terms of their logic, rationality, and logistic and political implications. Such an approach to change might even yield dividends in the near future. The current reality of a requirement of four years of postgraduate training in psychiatry is not entirely a burden. The extra training time can be put to good use if we are able to define what kind of nonpsychiatric medical and psychiatric medical experience are most essential in shaping a good psychiatrist. To do this, we must get back to the troubling question of defining a psychiatrist's task. We must develop a functional analysis of what services psychiatrists should provide. Once we can reach some consensus as to the psychiatrist's functions, training experiences in both psychiatric and nonpsychiatric medicine can be explicitly shaped to meet the trainee's functional needs. My expectations are that if we use such a rational approach, we will end up with a four-year program that puts minimum emphasis on traditional internship experiences.

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Comment

BY JOHN ROMANO, M.D.

DR. HALLECK is troubled by the recent decision of the American Board of Psychiatry and Neurology to return to requiring a fourth year, or internship year, of preparation for psychiatry. I, too, have been troubled. However, my concern dates back to 1970, when the traditional internship requirement (the fourth year) was summarily eliminated (1). Since then, I have also been concerned with the sometimes valiant but quite unsuccessful attempts on the part of departments of psychiatry to design, execute, and supervise substitute programs to take the place of the freestanding rotating internship or the more extended residency preparation in medicine or pediatrics of the past.

Before responding to Dr. Halleck's ahistorical objections, I wish to point out that the psychiatric training program was a four-year program from the inception of the American Board of Psychiatry and Neurology in 1934 until 1970. Within that period the major eligibility requirements for candidates for certification were graduation from an approved medical school with the Doctor of Medicine degree; possession of an unlimited license to practice medicine in a state, commonwealth, or territory of the United States or a province of Canada; and one year of clinical internship. These requirements applied to all medical school graduates of the class of 1934 and subsequent classes.

The formal psychiatric training program after the internship was for a period of three years, followed by two years of practice before a person was eligible for examination. This schedule remained unchanged with the exception of several years after World War II, when the wartime internship of nine months was accepted as equivalent to the one-year internship.

In 1970 the one-year internship requirement was removed for M.D. candidates but not for D.O. candidates, and in the following year it was eliminated for both. In the intervening years (1970–1977) departments of psychiatry accepted candidates for their residency programs who 1) had traditional freestanding internship or residency preparation in medicine or pediatrics, 2) had no internship, and 3) were to be engaged in a four-year program variously constituted of assignments to medicine, pediatrics, neurology, and family medicine, often in hospitals distant from the parent department and usually with four months or more of psychiatric assignment to the parent department. In the 1977–1978 *Directory of Medical Specialists*, mandatory training requirements for physicians entering training programs on or after July 1, 1977, included two patterns (2). The first pattern includes the categorical and flexible first-year programs, which include experience principally in medicine but also in family practice or pediatrics. The second pattern is a four-year training program in psychiatry in which at least one year is to be spent in an approved program that provides direct responsibility for the general medical care of children and/or adults.

Dr. Halleck questions the wisdom of deciding to return to the internship requirement without sufficient evidence to make the change. This is the very point I made at some length eight years ago, when the decision was made to eliminate the internship requirement (1). He suggests that a survey could have been undertaken to evaluate and compare the performance of residents who had internships with those who did not. I believe such a survey would have little validity if it were based on self-report of the residents or on data obtained exclusively from those who conducted the residency programs. Bias would be almost insuperable because of self-selection and the very human trait of blessing what one has done, particularly in preparation for a professional career.

Objective tests for the acquisition and retention of information, knowledge, and certain skills, similar to those used by graduate boards, could be designed. I learned from Dr. Lester H. Rudy, Executive Director of the American Board of Psychiatry and Neurology, that no attempt has yet been made to compare the examination performances, in whole or in part, of those candidates for certification who had an internship and those who did not (personal communication, March 20, 1978). He indicated that this may be done in the future. Furthermore, at this moment (March 1978), Dr. Rudy does not have data on the number of applicants for certification who did not have an internship.

Obviously, to obtain both reliable and valid appraisals of the operational competence of the psychiatrist would require an intimate surveillance of the day-to-day judgments he makes in the study, care, and treatment of his patient and the patient's family—hardly an easy task. Furthermore, prodigious difficulties would be posed by the controls necessary for the systematic study of sufficient numbers of psychiatrists, with attention to their medical school preparation, the nature of the internship, the pattern of the residency, and current modes of diagnosis and treatment.

Dr. Halleck does not exaggerate when he recounts

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the formidable logistic problems encountered by departments of psychiatry in assuming responsibility for the internship assignment. We cannot know with certainty that the problems posed by the internship have had a major effect on the recruitment of medical students to careers in psychiatry. This is a highly complex issue undoubtedly influenced by many determinants, but it seems possible that the considerable confusion and obviously second-rate quality of many of the internships have contributed to the current recruitment problems.

Over the past several years our interns have shared with me their experiences, pointing out the patchwork quilt design of the internship, the considerable unevenness in the quality of the teaching and supervisory experience and in the nature and breadth of clinical experience, the feeling that they do not belong or are accepted with sufferance, and, most particularly, the distance, both geographic and intellectual, from the parent department of psychiatry. Furthermore, a number have been dissatisfied with the four-month psychiatric assignment during the internship year because they are asked to assume responsibilities, often in midyear, that are equivalent to those of the more informed resident and for which they have not been adequately prepared.

The assumption that the department of psychiatry would, or could, be in a position to supervise or be responsible for the internship assignment in medicine, pediatrics, neurology, or family medicine in hospital and clinic services at some distance is not only mistaken but patently absurd. When one considers how such an internship could be inspected, examined, or evaluated by review committees, one realizes how impossible is the task. One has only to remember how perfunctory and inadequate is the evaluation of psychiatric residency by the residency review committee.

Although Dr. Halleck is aware of the reduced clinical experience of our medical students as a result of the fourth-year elective opportunities and of the obvious need for today's psychiatrists to be more informed of psychopharmacology and of biological matters in general than were their predecessors, he takes issue with what he considers the most common justifications for the internship. In his view, these are 1) that psychiatrists need to be more like other physicians, and 2) that the future role of psychiatry will increasingly involve consultation and liaison services to primary care specialists. He challenges the general notion of the psychiatrist's learning to be a physician, believing that the idea of physicianship is often couched in mystical terms. He believes that psychiatrists are unduly concerned about their image as doctors and that psychiatrists would err should they adopt the medical model of the practicing physician. Dr. Halleck describes the extreme reductionistic aspect of the medical model, in which he believes attention is focused entirely on etiology related to biological dysfunction. Although he concedes that there are more sophisticated medical models dealing with multiple determinants and with the interaction of biological, psychological, and social factors, he believes that most medical practice is focused almost entirely on the simpler reductionistic model and, further, that the traditional internship may reinforce the reductionistic medical model, which he believes is inappropriate for the education of today's psychiatrist.

Evidently Dr. Halleck and I have had quite different experiences. It has never occurred to me that the lessons learned during the internship could lead to a mystical notion of physicianship. From personal experiences, from direct observations of several generations of interns and residents, and from their direct reports to me, I believe the contributions of the internship year to their professional education can be stated simply and clearly, with no vestige of mysticism. Eight years ago I asked each of our residents to write the five most important contributions of their internship experience, aside from the obvious increases in knowledge and skills about medical and surgical conditions. The following clusters emerged:

> a) significant change in his perception of himself, and his awareness of the perceptions of others that he is a physician; this was a different perception from that he had encountered as a clinical student; he became aware of assuming a new role in terms of responsibility, accountability, and dependability in the direct care of patients, engagement with families of patients, and learning how to work with others in the multiple set of the several health professions; b) repetitive application of theoretical knowledge to practical ends, responding to emergency needs, dealing with the dead and dying and with the reactions of their families; remaining responsible to a patient's charge in spite of fatigue, becoming more efficient and confident in problem-solving, decision-making, and planning daily activities; c) exposure to senior colleagues in the several medical disciplines and learning from them different styles, methods of approach, and the application of useful knowledge and technique; learning something of the problems of other medical persons in dealing with their patients; d) additions to one's store of knowledge about the significant psychosocial aspects of patients and their families, both the acutely and chronically ill, the convalescent and the disabled, learning something of the special problems of the battered or burned child and his parents, of the pregnant teenager, the anxiety of the middle-aged man with myocardial infarction, and the legion of other patients for whom the intern becomes directly responsible for study, care, treatment, and disposition; skill and confidence in interviewing and examining patients with somatic and psychological signs and symptoms with particular attention to the differential diagnosis of pain and disability; e) opportunity for full measure of determining choice of career, having kept options open, and learning of one's interests and aptitudes in the various fields of medicine; opportunity to engage in the teaching of medical students, nursing students, and others. (p. 1574)

More recently, in meeting with our current group of interns, much of the above was restated. The following statement of one of our interns (R.M. Fuchs, personal communication) is quite representative.

> Becoming an intern involves more than obtaining an M.D. after one's name. The task of the medical student is to make the transition from layman to physician by acquiring knowledge and becoming comfortable with the label 'doctor.' An intern, however, *is* a doctor. He is forced to make critical decisions, to be ultimately responsible for patient care, and to deal first-hand with death and grief—all with scarcely more wisdom than he had a year previously. The intern is the pivot around which revolve the patient, the attending physician, the senior house officers, the family, and the hospital. He must objectively integrate concerns that are often contradictory. The responsibility of coping with human suffering is a burden new to the intern. He must learn to get beyond his own emotional responses to sickness and death in order to deal effectively with his patients.

There can be no argument with Dr. Halleck when he indicates the inadequacies of the reductionistic biological model as an appropriate preparation for tomorrow's psychiatrist and, equally important, for tomorrow's physician. Perhaps Dr. Halleck would agree that the reductionistic psychological model espoused by many psychiatrists is also an inadequate preparation for tomorrow's psychiatrist and physician. There are those who defend the notion that all human distress is explicable in the paradigm of unconscious conflict and attempts at its resolution.

Obviously, modern biology, as well as modern medicine and psychiatry, can no longer be satisfied with simplistic reductionistic notions of health and disease, whether they emerge from infectious disease or from neurotic conflict. In fact, I have been impressed by the increasing sophistication of our medical colleagues in dealing intelligently and perceptively with multiple determinants of behavior and with the complex interrelationships of biological, psychological, and social phenomena, which play a part in the maintenance of health and in the emergence of disease. This is apparent not only among the clinical investigators in the fields of endocrinology, toxicology, metabolic diseases, and neoplasia but also with practitioners in their dealings with patients and their families. Like Gill (3), I have difficulty understanding what is meant by the medical model; I have even more trouble understanding what is meant by the "nonmedical model," whatever that may be. In operational terms, the medical model is often, if not usually, depicted as one in which the physician is active, authoritarian, and directive and the patient is compliant, passive, and in general nonparticipating. An example would be the administration of an antibiotic drug to a patient with an infectious disease, in which the body takes over and the patient, as a person, has no part. However, as we all know, within the medical model are those engagements between patient and physician in which the patient is encouraged and expected to take an active, conscious part in the treatment of his distress—for example, in modifying his diet, in self-medication, elective surgery, in bringing about changes in life patterns (smoking, recreation, responding to genetic counseling), etc. For that matter, in the nonmedical model, sometimes called the psychological model, there are approaches as authoritarian, if not more so, as any of the traditional medical models, e.g., behavior modification treatment and the interpretive judgments in psychoanalytic therapy. Therefore, in both models we can find the idea of patients passively accepting medication, advice, explanation of drugs, surgery, etc., but there are also other instances in which the patient must consciously and actively assume responsibility for behavioral change.

Clinical experience teaches us to be aware of the limits of usefulness of the Parsonian model of the sick role. This model was designed for those who are acutely physically ill and has not been useful in understanding patients with chronic medical and surgical illnesses or most psychiatric patients (4).

I am somewhat puzzled by Dr. Halleck's criticism of psychiatric consultation, and I wonder what relationship his comments have to the internship preparation of the psychiatrist. Psychiatric consultation is a skill to be learned during the residency period, and evidently those he criticizes did not have adequate preparation for their task.

On reflection, I am left with the impression that Dr. Halleck's negative view does not concern the internship exclusively but rather reflects serious reservations as to the usefulness of medical education in general in the professional training of the psychiatrist. If this is so, I would hope that he would clarify his position and explain more fully his reasons for these reservations.

I agree with one of Dr. Halleck's concluding remarks, when he insists that whatever changes we might make in psychiatric education should be informed, intelligent, and appropriate to the tasks of the psychiatrist as we perceive them today and as we may anticipate them to be tomorrow. The internship, no more than any other educational program, should never be considered a sacred cow. The curriculum is an instrument, a means to an end, never an end in itself, and is changed, as it should be, by the introduction of new knowledge and the acquisition of new skills. Furthermore, the internship cannot be dealt with in isolation because it will be affected by college and medical school preparation and, in turn, will affect materially the specialty training that follows it.

Surely there must be by now sufficient evidence to warrant significant revision of both undergraduate and graduate medical educational programs. It must be clear that the various experiments with the curriculum in the undergraduate period (particularly in those schools that established three-year programs) have led to diminished clinical experience and scholarship in recent medical school graduates. In the past the fourth year of medical school provided advanced clinical clerkships in all the major clinical disciplines, but with the change to an elective year, most students were deprived of the opportunity for sustained responsible clinical assignments. Thus, the assumption of the Millis Report—that the undergraduate clinical clerkship

would be sufficient to transform the student into a physician—has not been fulfilled, nor has the expectation that there would ensue institutional responsibility for graduate medical education (5). As a result, a considerable number of students face serious problems of transition between their role as students and their entrance into the formal specialty training program. This is particularly true for psychiatry, but it is also true for those who are interested in careers in anesthesia, pathology, dermatology, physical medicine, radiology, and perhaps others. These students wish to obtain a full measure of at least a year of clinical internship before they embark on their specialty training but have serious problems doing so because internal medicine, pediatrics, and family medicine, which would be primary in the construction of such internships, have unusually great demands on their resources. All of this has led, as most of us know, to students being forced to make early career choices when they are insufficiently informed. As a result, students often have to lie to convince program directors in internal medicine, pediatrics, and family medicine of a commitment of which they are really unsure. I am vigorously opposed to forcing students to make premature decisions. On an earlier occasion I wrote,

> I am not one who defends the thesis that there should be unbroken continuity between collegiate education, medical school, and graduate specialty training. I have learned that growth is hardly ever regular or even, and I would warmly support the occasional discontinuity or psychosocial moratorium that enables the student to free himself from the restrictions of the convoy system to examine more carefully his objectives and to plan more intelligently for his future career. Whatever changes may take place in the future, let us try to give our students every opportunity to make intelligent and informed decisions in their choice of career and special interest. This will require curricular planning, with full measures of scholarship in all fields relevant to their professional aspirations. (6)

I am heartened to learn that these matters are under serious consideration by the Council on Medical Education of the American Medical Association (7). With others, I hope sincerely that this group can enlist the interest and support not only of psychiatry's fellow sufferers in the clinical disciplines who face similar problems with what has been called the flexible internship but also the assistance and cooperation of those responsible for the primary care programs in medicine, pediatrics, and family medicine. One hopes, too, that these matters will engage the attention of the faculties of our medical schools. Only if there is a concerted attempt to correct the current deficiencies in clinical scholarship in the undergraduate period and the obviously deficient transitional flexible internship for the clinical specialties noted can we hope to ensure adequate standards for professional education.

As must be evident, I consider the internship, together with collegiate and medical education obligatory experiences in the professional preparation of the psychiatrist. I would hope that proper measures will be taken to ensure full measure of the internship assignment in terms of clinical experience and high standards of educational supervision.

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BRIEF COMMUNICATIONS

Seclusion: Uses and Complications

BY MARLIN R. MATTSON, M.D., AND MICHAEL H. SACKS, M.D.

A total of 7.2% of the patients in a private psychiatric hospital were placed in seclusion during a one-year study. These 63 patients had a more frequent diagnosis of schizophrenia and manic-depressive illness, manic type, than a comparison group. Significant intergroup differences in age and length of hospitalization became insignificant when the seclusion patients under age 20 were excluded. Although seclusion was often used to protect patients, others, and property, the most frequent reason was to maintain a therapeutic environment. This study pointed toward a staff tendency to see the "problem" of the patient's behavior as being solved by seclusion rather than viewing seclusion as an intensive care environment in which the patient's behavior and treatment could be more carefully monitored.

THE SECLUSION ROOM is a regular part of most psychiatric units. Despite this, its use as a form of treatment for disturbed patients has received remarkably little attention. A computerized search of the psychiatric literature from 1964–1975 located only two relevant articles (1, 2), both of which viewed seclusion as an effective way of managing the destructive behavior of severely disturbed patients. Wells (1) reported that it was used with 4% of the patients admitted to a university hospital psychiatric unit but did not make any note of complications arising from its use. Fitzgerald and Long (2) commented briefly on the effects of staff disagreement on the use of seclusion, the importance of monitoring the patient's medical status, and the possibility that sensory deprivation might lead to a worsening of the mental state. One suspects that this paucity of reports on seclusion may relate to the discomfort of the psychiatric profession regarding its use. It is a topic that for personal, humanitarian, ethical, and legal reasons might lead to an avoidance of discussion and investigation. As part of a generalized review of the use of seclusion, we thought it might be useful to report our findings in order to stimulate discussion and establish directions for further study.

METHOD

A seclusion patient was defined as any patient who required the use of a locked seclusion room at any time during his or her hospitalization. All seclusion patients during 1975 were included in the study. The diagnosis, age, sex, marital status, length of hospitalization, reasons for seclusion, and complications of seclusion were recorded. These patients were then compared with a random selection of 160 nonseclusion patients during the same period. The hospital is a 104-bed private voluntary psychiatric division of a general hospital. It consists of six inpatient units, all of which are heavily staffed and involved in resident or medical student teaching.

RESULTS

During the period under study 63 patients (7.2% of the patient population) required seclusion. More than one-half of these patients (63%, N=40) were diagnosed as schizophrenic, compared with 38% of the nonseclusion group (N=61). The rest of the seclusion group received diagnoses of manic-depressive illness, manic type (17%, N=11), personality disorder (10%, N=6), and other (10%, N=6). In contrast, in the non-

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TABLE 1
Types of Behavior Leading to Seclusion in a Sample of 63 Patients

Behavior*	Number of Patients	Percent of Episodes
Behavior disruptive to therapeutic environment	63	34.4
Assaultive to others	46	25.1
Threatening or verbally abusive	14	7.7
Destructive to property	14	7.7
Dangerous to self	13	7.1
Other	33	18.0

*Some patients exhibited more than one type of behavior.

TABLE 2
Complications of Seclusion in a Sample of 63 Patients

Complication*	Number of Patients	Number of Episodes
Assaultiveness	19	32
No note with each 24-hour period by primary therapist	16	26
Self-injury	8	10
Destruction of seclusion room	4	5
Deterioration of mental status	4	7
Deterioration of physical status	3	5
None	33	

*Some patients exhibited more than one complication.

seclusion group 4% (N=6) received diagnoses of manic-depressive illness, manic type, and 14% (N=22) personality disorder. These diagnostic differences were significant ($p<.01$, chi-square test).

Comparisons of sex and marital status revealed no significant intergroup difference. However, intergroup age differences were significant ($p<.05$, chi-square test). In the seclusion group the percentages for the 10–19, 20–29, 30–39, and 40 and over age groups were 24% (N=15), 29% (N=18), 29% (N=18), and 19% (N=12). Percentages for the nonseclusion group were 10% (N=16), 36% (N=59), 21% (N=33), and 32% (N=52). If the 10–19 age group is excluded, the age difference no longer reaches significance.

Patients who required seclusion had a significantly longer length of stay (58 days) than nonseclusion patients (38 days) ($p<.05$, t test). This significant difference disappears if patients aged 17 and younger are excluded. Table 1 lists the types of behavior that led to seclusion. The complications occurring during seclusion are shown in table 2.

DISCUSSION

The use of seclusion in our hospital was greater than that reported by Wells (1) (4% versus 7.2%). This difference may be even larger, since we defined seclusion as a locked room, which excluded quiet room patients. The differences noted may be due to patient population, staffing patterns, and attitudes concerning the use of seclusion. Greenblatt and associates (3) have documented how changes in staff philosophy can have a dramatic effect on the frequency with which seclusion is used. The general therapeutic philosophy on our units might best be described as a combination of psychopharmacology and expressive therapies (individual, group, and milieu). Seclusion is usually a last resort in helping patients to establish control of their behavior.

Diagnosis and age had a significant effect on whether a patient would be secluded. Schizophrenic and manic-depressive, manic type, patients tended to be secluded more frequently than others. These findings are in the expected direction. Age also had a significant effect on whether a patient would be secluded, but only if the 10–19 age group was included.

The mean length of hospitalization for patients who were secluded was significantly greater than that for patients who were never secluded. However, if one removes the adolescent patients from the seclusion group, the difference between the mean length of hospitalization for the adult seclusion group and the nonseclusion group is no longer statistically significant. Since the treatment program for adolescent patients emphasized a longer period of hospitalization, the fact that a patient required seclusion did not seem to provide an indication of the length of time he or she would need to be hospitalized. Similarly, neither sex nor marital status was significant in distinguishing seclusion from nonseclusion patients.

Although a patient requiring seclusion was more likely to be schizophrenic or manic, the data in table 1 provide more specific information regarding the behaviors that actually led to seclusion. Assaultiveness and behavior disruptive to the therapeutic environment were the most common reasons for seclusion. Examples of the latter behavior included repeated slamming of doors, running up and down the corridor unclothed, and persistent screaming. Although such behavior is not usually threatening to either the patient or others, it is the most frequent cause of seclusion. Disruptive behavior, as well as threats of assaultiveness, are areas in which staff attitudes toward psychotic behavior are very relevant in determining the frequency with which seclusion is used. Other variables that may affect the decision to seclude the patient and which require further study are staff-patient ratios, availability of staff, general anxiety on the ward, and staff training.

However, we have found that placing the patient in seclusion does not entirely restore the therapeutic environment of a ward. The rest of the patients remain apprehensive and concerned about the patient who is in seclusion. Fears regarding a similar loss of control or the inability of the staff to prevent such behavior from occurring are common themes during this time. However, the use of seclusion does enable the unit to place the patient in a quiet environment where he or she can be more closely observed so that frequent re-evaluation of the adequacy of the treatment plan can be made. At the same time the opportunities to hurt

oneself, to hurt others, or to damage the environment are minimized. These factors have led us to think of the seclusion room as an intensive care room, since it seems to be more appropriate to the needs of the patient at the time he or she requires its use.

We had hoped in the chart review to evaluate the effect of the seclusion room itself on the patient's behavior but could find no data to support either the traditional belief that removing a patient from the stimulating activity of a ward to a "quiet room" has a calming effect or the contrary hypothesis that the resulting sensory deprivation might produce further deterioration. What seems to occur is that a patient who is in seclusion is rapidly medicated while continuing the behavior that placed him or her there in the first place. The sedative and antipsychotic effect of the medication, combined with the patient's tiring himself out, may be the principal results of seclusion. This would be more consistent with the view that the seclusion room is not a treatment in itself but simply a place where intensive treatment can occur—an intensive care room. This approach enables the staff to more realistically appraise the function of the seclusion room and to explain its use to patients.

A surprisingly high number of complications occurred while patients were in seclusion (see table 2). One interesting complication was the tendency for the patient's therapist to provide less documentation of his or her involvement in the patient's total care. This was evidenced by poor documentation in the medical record of 1) evaluation of patients going into seclusion, and 2) reevaluation at intervals thereafter if seclusion needed to be continued. Although this absence of documentation is not a complication in the strictest sense, it could result in complications by not providing other staff responsible for the patient's care with information regarding the patient's behavior and the proposed treatment plan. These lacunae in the charts of seclusion patients could be related to a tendency toward the decreased involvement of some staff with the patient during this period or to uncomfortable feelings about placing a patient in seclusion. They seem to mirror the lack of attention to the secluded patient in the psychiatric literature.

We found that assaultiveness was also a common complication of seclusion, necessitating reevaluation of the patient's mental status and the adequacy of the current treatment plan. Seclusion alone is not sufficient to control assaultiveness. In over half of the occurrences, patients were assaultive to staff in the seclusion room. In slightly less than half of these episodes, the assaultiveness occurred while the patient was trying to leave the seclusion room when the door was unlocked by staff or, if the patient succeeded in leaving, during attempts to bring him or her back.

Self-destructive behavior was also a frequent complication. Despite staff precautions, some patients managed to take a sharp object into the seclusion room and others managed with great ingenuity to use the seclusion room itself to hurt themselves. Patients have unscrewed security light fixtures to expose electric wires and chipped tiles to obtain sharp-edged instruments. Of course, such assaultive or self-destructive behavior is often what caused the need for seclusion in the first place. However, it is clear that seclusion is not a "cure" but simply a more efficient way of enabling the staff to provide continued surveillance.

Those patients whose mental and physical status deteriorated in the seclusion room are worth further comment. One was a 53-year-old acutely schizophrenic woman who appeared to have a marked paradoxical response to chlorpromazine characterized by increasing confusion, agitation, and bizarre behavior that was not recognized immediately. The other patient was a 17-year-old boy who appeared to be acutely intoxicated and became assaultive when examined. Once in seclusion he became increasingly unresponsive. A sedative overdose was diagnosed and confirmed by gastric lavage. In both of these cases there was an unexpected organic complication that was not immediately considered once the patient had been secluded. In both situations there seemed to be a tendency to regard the seclusion room as a symptomatic treatment and the changes in behavior as temporary reactions to seclusion rather than as a progression of the symptoms that initially required the use of seclusion.

The danger in assuming that the seclusion room is a treatment in itself is that it may provide a false sense of security that the symptoms or their cause is being treated. This is also evident in our finding that many of the patients who deteriorated while in seclusion were not receiving adequate medication. The importance of repeated careful evaluations of the patient's mental and physical status, diagnosis (e.g., is there an organic component?), and the adequacy of current treatment once the patient is in seclusion must be emphasized.

CONCLUSIONS

In conclusion, we have found that 7.2% of the patients in a private psychiatric hospital were placed in seclusion. These patients had a more frequent diagnosis of schizophrenia and manic-depressive illness, manic type, than the nonseclusion group. Significant intergroup differences in age and length of hospitalization became insignificant when the under-20 age group was excluded. Although seclusion was often used to protect a patient from himself or herself, from hurting others, and from being destructive to property, the most frequent reason was to maintain a therapeutic environment. Staff attitudes and experience seemed to be a critical factor in this determination. The study of complications of seclusion pointed toward a staff tendency to see the "problem" of the patient's behavior as being solved by seclusion rather than viewing seclusion as an intensive care environment in which the patient's behavior and treatment can be more carefully monitored.

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Marijuana Use in Schizophrenia: A Clear Hazard

BY DAROLD A. TREFFERT, M.D.

The use of marijuana as the independent variable produced a serious exacerbation of a psychotic process in four schizophrenic patients whose illness was otherwise well controlled with antipsychotic medication. Each patient served as his own control—the sole substance abused was marijuana and antipsychotic medication intake remained constant. Each time marijuana use at moderate levels began, there was exacerbation and deterioration. The author suggests that marijuana use is a special hazard to schizophrenic patients and that physicians should alert such high-risk patients to the possible untoward interaction between their illness and marijuana.

CONTROVERSY CONTINUES as to the degree of significant adverse medical effects of marijuana on various systems in the human body (1, 2). Medical/psychiatric studies thus far show that there is a temporal relationship, at least in some cases, between cannabis use and the occurrence of psychotic episodes (3–5). These episodes may be 1) toxic psychoses as a result of acute cannabis intoxication (6–12), 2) conditions resembling mania and dementia as a result of long-term cannabis abuse (10–13); or 3) paranoid psychoses resembling paranoid schizophrenia in the course of long-term cannabis abuse (13–15).

Questions remain regarding the number of cases (16), the dose relationship, if any (16), and whether marijuana causes a psychosis in an otherwise healthy person or whether it merely precipitates a latent psychosis in a predisposed person who already has significant premorbid psychopathology (5, 16, 17). While those controversies continue, surprisingly little attention has been paid to the important clinical observation that patients who are psychotic, irrespective of etiology, run the risk of relapse and exacerbation of an otherwise well-controlled illness by use of even moderate amounts of marijuana (3, 4).

Thacore and Shukla (18) found that 25 patients with cannabis psychosis responded swiftly to short-term antipsychotic treatment, and relapses occurred only when the patients reabused cannabis. Treatment was directed at preventing further abuse. To the already psychotic patient the use of marijuana in even moderate doses presents a clear medical hazard

I will describe four patients with well-documented schizophrenia in which the use of marijuana as the sole independent variable produced exacerbation of the psychotic process in an otherwise well-controlled illness.

CASE REPORTS

Case 1. Mr. A, a 23-year-old man, entered a general hospital psychiatric unit in June 1976 after severely mutilating himself with a razor blade. His first psychotic episode had occurred in January 1975, when he abruptly became profoundly withdrawn, stopped eating, could not sleep, and spent hours in bed in absolute silence with covers over his head to shut out all possible stimuli. He developed well-crystallized delusions, bizarre behavior, and a fulminating thought disorder. These symptoms followed his first involvement with smoking marijuana (two to three joints a day for a period of 2 months). He had no drug history and was not involved with any other drugs. There was a strong family history of mental illness—his paternal grandfather had committed suicide at age 40 and his maternal grandfather and a maternal uncle had been hospitalized for many years for psychiatric illness.

Mr. A, blatantly psychotic, catatonic, delusional, and periodically agitated, was hospitalized at that time (January

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1975) for approximately 2 weeks. He impulsively left the unit and threw himself in front of an automobile, seriously injuring himself. He was then transferred to a state hospital, where he remained for 4 months. He was released on fluphenazine enanthate and was maintained at home on an outpatient basis. He was generally active and alert and resumed his drawing and painting, which he was involved in prior to his first psychotic episode. He participated in athletics, slept well, and did some socializing.

In May 1976, 2 weeks prior to his second admission to the hospital, Mr. A began using marijuana heavily (at least three times daily on a regular basis) and was on what he described as a "2-week trip." He used no chemicals other than marijuana, used no alcohol, and continued to take his psychotropic medication. It was following the "2-week stoned trip" that Mr. A mutilated himself. At admission he was frightened, startled, and nearly mute; showed remarkably flattened affect; and was hallucinating and delusional. The self-mutilation occurred as part of an extensive delusional system involving God, the devil, and bisexuality.

The patient was hospitalized for 3 months and responded to a combination of electroconvulsive therapy and high doses of psychotropic medication including haloperidol, trifluoperazine, chlorpromazine, and amitriptyline. With regular medication and without marijuana, the remission has been maintained over 18 months. Mr. A attributes his relapse to the resumption of marijuana use even though he had been taking his psychotropic medications regularly.

Case 2. Mr. B, a 21-year-old Marine, began using a variety of chemicals, including marijuana, lysergic acid diethylamide (LSD), mescaline, and heroin, while overseas. He admitted to heavy use of cocaine and LSD. In July 1975 he was shooting methylenedioxyamphetamine (MDA) at a party, and also was taking pentachorlophenol (PCP). Shortly thereafter he was hospitalized with a "toxic psychosis," which included bizarre and manic behavior, severe agitation, a profound thought disorder, and a well-crystallized delusional system. There was obvious liver impairment as well, with SGOT levels of 1280 units. He was hospitalized on the psychiatric unit of a general hospital for approximately 4 weeks and responded slowly but regularly to high doses of fluphenazine decanoate, thioridazine, and haloperidol. His fragmented thinking slowly cleared, and his confusion and the delusional system disappeared.

He was discharged reasonably symptom-free but on high doses of psychotropic medications. The delusions and hallucinations had disappeared completely. Mr. B did well for 3 months but then began to smoke marijuana "to help his depression." Nevertheless, he continued his psychotropic medications regularly as prescribed. Following the resumption of daily marijuana smoking, his condition deteriorated and his delusional system promptly returned, requiring rehospitalization in February 1976. He remained in the hospital approximately 3 weeks, during which time his symptoms again were relieved; he then returned to outpatient status.

During the months following, Mr. B followed the prescribed medication regimen but periodically resumed the use of marijuana (no other chemicals or alcohol). Each resumption of marijuana use was correlated with a deterioration of his condition—a return of an obvious thought disorder, ideas of reference, preoccupation, staring, and withdrawal to bed for hours at a time. When counseled about the need to avoid marijuana, he would stop use of the substance and his symptoms gradually would clear. After several such episodes he agreed with the observation that marijuana was toxic to him; for the next 6 months he did not use marijuana and his psychotic symptomatology did not recur. He continues to take his psychotropic medications regularly, is working, and is free of his ideas of reference and delusional material. He is much more active, socializes well, and shows no evidence of a thought disorder.

Case 3. Mr. C was first hospitalized for approximately 5 months at age 15 for treatment of a severe schizophrenic illness characterized by catatonic symptoms, profound withdrawal, and disorganized thinking. He was maintained on antipsychotic medication and was seen on an outpatient basis over a period of approximately 36 months. At age 18 he required rehospitalization following discontinuation of medication and experimentation with, and then daily use of, marijuana. That hospitalization lasted nearly 9 months before the agitation, paranoid delusional system, and bizarre behavior subsided sufficiently for Mr. C to be released on intramuscular fluphenazine decanoate.

Mr. C did well for approximately 4 months. However, he resumed smoking marijuana heavily 2 weeks prior to his third hospitalization; at the time of his admission he turned in his "grass," pipe, and a supply of marijuana to the admitting doctor. For those 4 months between hospitalizations Mr. C was maintained on intramuscular fluphenazine decanoate. At admission he was frightened, anxious, paranoid, agitated, and again showed obvious thought disorder. He had a series of electroshock treatments to which he responded favorably; after 6 months he was released on piperacetazine and lithium carbonate. As an outpatient, Mr. C did quite well; he resumed work, became more socially active, and showed no evidence of his delusional system or thought disorder. His affect remained somewhat flattened. Mr. C continued regular intake of his prescribed medications; however, he resumed the use of marijuana in order to establish better social relationships with his friends, who were using marijuana on a regular basis. He slowly began to deteriorate again and experienced withdrawal, ideas of reference, agitation, and insomnia. He admitted that he had been using marijuana again in order to feel less depressed and more socially acceptable. Over the next 18 months Mr. C had episodes during which his illness deteriorated, and each of these was correlated with the resumption of marijuana, in spite of regular intake of psychotropic medications. In February 1976, the patient began to use marijuana very heavily and became hyperactive, explosive, uncontrollable, and finally had to be transferred to a maximum security hospital. He remained in that setting for approximately 4 months. After the delusions and agitation ceased, he returned home on lithium and trifluoperazine, which he has continued regularly. He has maintained his remission for approximately 4 months, and since he has concluded that the marijuana was responsible for his deterioration, he has not resumed its use.

Case 4. Mr. D, a 24-year-old man, was first seen in July 1973, after a period of progressive depression and disorganization. In the 3 months prior to admission he had been increasingly confused and disoriented, with disorganized thinking. At the time of admission he was disorganized, his associations were loose, and there was an obvious thought disorder. He was agitated, sobbed and cried uncontrollably, and experienced a great deal of sexual preoccupation and bizarre ideation. Mr. D indicated that he had been using psychedelics heavily and had taken LSD or mescaline on more than 100 occasions. He also admitted to heavy use of marijuana and the use of amphetamines. He responded fairly

promptly to fluphenazine decanoate and trifluoperazine and was followed on an outpatient basis.

After 12 months the medications were gradually decreased. Mr. D finally discontinued both the medication and outpatient contacts. He began graduate school 12 months later and also resumed the use of marijuana. He indicated that he was smoking approximately three to four times per week but was using no other drugs or chemicals. He became increasingly agitated, developed a fairly elaborate delusional system, and went to New Jersey to meet with the president of the United Nations. Mr. D became acutely agitated on the plane to New Jersey and continued to have delusions; after an unplanned stop he was hospitalized at a county receiving hospital and was frankly delusional and psychotic. When he finally was transferred to a hospital near his own home, Mr. D was preoccupied and distant. He spoke slowly and deliberately and talked about his ideas of reference and a fairly expansive delusional system. He indicated that he had resumed the use of marijuana. Mr. D was placed on psychotropic medications and responded well to them. His thinking cleared, he became less preoccupied, and he was discharged on a combination of fluphenazine decanoate, haloperidol, and diazepam.

DISCUSSION

I have not presented these four cases to draw any conclusions regarding the relationship between onset of the original psychosis and the use of marijuana, although there was a temporal relationship between the use of marijuana and the onset of psychosis in each patient. It should, however, be noted that other drugs were involved in the original onset in at least two of the cases and that in one case there was a strong familial incidence of major mental illness.

Instead, these cases illustrate that in schizophrenic or otherwise psychotic patients whose illness is well-controlled with medication, the use of marijuana is a clear hazard, irrespective of etiology of illness or involvement of drugs in onset. Patients who use marijuana run a risk of exacerbating an otherwise well-controlled illness. In each of the four cases the patient served as his own control; i.e., marijuana was the only substance used, and each time the patient resumed use of marijuana at moderate levels (on his own volition and against medical advice), his illness was exacerbated. Abstinence from marijuana led to improvement and stabilization.

All four patients are still under treatment, and each has become convinced, through persuasion and his own observation, that marijuana use is clearly correlated with recurrence of psychotic symptoms. Each of the four patients now abstains entirely from marijuana use, and remission has been sustained. One patient describes marijuana as "ungluing" him, and another talks about it being "toxic" to him. Nevertheless, during rehabilitation each has felt a temptation to use marijuana to deal with the depression frequently felt after a major psychotic episode and/or to reestablish better social relationships, particularly if prior to hospitalization the patient was involved in a peer group where marijuana use was frequent. The schizophrenic patient is at high risk with respect to marijuana use. Perhaps some persons can safely use marijuana, but schizophrenics cannot. This special risk of marijuana use to the 500,000 schizophrenic persons in the United States has obvious and far-reaching implications.

The schizophrenic patient must be alerted to the special hazard of marijuana use for the same reasons that the patient on tranylcypromine must be alerted to the special hazard of foods with high tyramine content or the patient with porphyria to the special hazard of barbiturates. It is the physician's responsibility to alert all high-risk patients about the possibility of experiencing untoward interactions between their illness and the substances they use.

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Popping and Snorting Volatile Nitrites: A Current Fad for Getting High

BY LEONARD T. SIGELL, PH.D., FREDERIC T. KAPP, M.D., GREGG A. FUSARO, E. DON NELSON, PHARM.D., AND RUSSELL S. FALCK

In certain social circles a variety of volatile nitrites are being used extensively to expand creativity, stimulate music appreciation, promote a sense of abandon in dancing, and intensify sexual experience. Soon after amyl nitrite (the prototype of this group) became a prescription drug, legally produced substitutes appeared and were sold as room odorizers. Analyses of eight of these substitutes showed them to contain isomers of butyl or amyl alcohol and a corresponding nitrite. The authors describe the context of use of these products and their desired and adverse effects.

AMYL NITRITE and other volatile nitrites have become popular street and over-the-counter drugs that are inhaled to alter consciousness, enhance meditation, stimulate dancing, and intensify sexual experience among certain groups of the population. They are examples of mankind's never-ending search for behavior-influencing agents that will produce a state of ecstacy with the utmost enjoyment of all desires and passions. Although a variety of psychological and physical approaches to this end have been and still are in vogue, such as transcendental meditation and yoga, the most popular means to achieve an altered state of consciousness and hoped-for expanded creativeness and hyperacuity of sensation has been the use of chemical substances. The list of such substances is endless, varying from ancient man's use of the poppy and fermented fruit juices to such current customs as smoking marijuana; snorting cocaine; mainlining heroin; ingesting downers, uppers, hallucinogens, and deliriants; sniffing glue; and, recently, inhaling volatile nitrites.

Our interest in the effects of and potential for harm associated with acute or chronic inhalation of volatile nitrites came about because of questions posed to two of us (L.T.S. and E.D.N.) who are involved with the Cincinnati Drug and Poison Information Center. The center is a hotline program that responded to 36,000 phone calls during 1976 (80% from the public). Several callers (especially users, staff of community mental health centers, and psychiatrists) wanted information about the contents of these products and their possible adverse effects.

In order to obtain information that would allow us to respond to these inquiries we conducted a literature search, analyzed the contents of several samples, and collected additional information from several users of these agents. (We submitted this paper to the *Journal* on September 9, 1977.)

BACKGROUND

Amyl nitrite has been used medically since 1867 to provide relief from attacks of angina pectoris (1). It is packaged in small, thin glass capsules covered by cloth webbing that can be easily crushed between the fingers and the fumes inhaled when the patient suffers anginal pain. In the late 1960s sales of amyl nitrite rose sharply due to its nonmedical use as an agent for getting high and as an aphrodisiac. It acquired the street name "poppers" because of the sound created when the capsules were crushed just before inhalation of the amyl nitrite fumes.

Since amyl nitrite was made a prescription drug in 1968, other liquid volatile nitrites have been marketed in bottles containing 10–30 ml and selling for $3–$10 each. Until recently male homosexuals indulged in these drugs more than other groups did. They would inhale the vapors just at the time of sexual orgasm, claiming that this enhanced their sensation. One author claimed that "poppers" are inhaled during anal intercourse by the passive partner, presumably to relax the anal sphincter and thus facilitate intromission (2), but they are also used by either partner in association with any sexual act.

During the past two years the use of these drugs has increased considerably among heterosexual men to supposedly enhance orgasm, as well as among both sexes, often in groups, just for getting high. Indulgers often take them in social situations, particularly to promote a sense of abandon in dancing, expand creativity, stimulate music appreciation, and enhance meditation.

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Common settings in which these agents are used include the bedroom, parties, backrooms of pornographic bookstores, pornographic theaters, bars, and dance floors. Some users have told us that a few discotheques use special lighting effects to indicate that they are about to spray nitrite fumes over the dance floor.

The snorting of volatile liquid nitrites for hedonistic purposes in recent years has created a large business estimated to total $50 million a year. Sales have reportedly averaged 100,000 bottles a week in one city alone (3). These substances are advertised in underground newspapers and flashy sex magazines, purportedly to be used as room odorizers to produce such supposedly aphrodisiac odors as the smell of a gymnasium. Since their labels state that they are not to be inhaled, they are sold legally over the counter in various pornographic shops, novelty or "head shops," or by mail order under such suggestive trade names as Heart-On, Jac Aroma, Locker Room, Bullet, Rush, Aroma of Men, Toilet Water, Ban Apple Gas, and Crypt Tonight. Unlabeled preparations produced by small manufacturers are also found in many cities. To their users they are all known as either "poppers" or "locker room." They are inhaled directly from the bottle or by way of single or double nasal inhalers.

The pleasurable sensations that are perceived from the inhalation of the volatile nitrites as well as from the other chemical agents used to affect mood, perception, thought, or behavior result from the combined effect of the pharmacology of the specific agent, the individual's expectation of what it will do for him or her, and the environment or social setting in which it is used. The consequences of drugs taken for purposes of affecting the mental apparatus are strongly influenced by the reputation they have among their advocates as to what effects they are supposed to have on one's psyche and behavior.

Most of the users we talked with were willing to discuss freely their experiences with these agents. Some expressed preferences for specific products, citing either a favored aroma or a stronger effect. Some reported experiencing prolonged pulsatile headaches and dizziness. Others claimed that they were no longer able to perform sexually without the use of these drugs.

CHEMISTRY AND PHARMACOLOGY

To determine the contents of currently available volatile nitrite preparations, we analyzed six brand-name products and two unlabeled street samples. The results indicated that six of the samples contained an isomer of butyl alcohol, two contained an isomer of amyl alcohol, and all had a corresponding nitrite.

We conducted an extensive search of the biomedical literature and found a paucity of clinical reports describing adverse effects from acute and/or chronic inhalation of these agents. Because of amyl nitrite's 111-year history of medical use in relieving symptoms of angina, far more is known about its systemic effects than about the effects of isomers of amyl and butyl alcohol and butyl nitrite.

Butyl nitrite was investigated in the late 1800s as an alternative to amyl nitrite for patients with angina (4). It has essentially the same therapeutic effects as amyl nitrite but has not been used clinically to any extent. Both amyl and butyl alcohol are used as synthetic food flavoring materials, the former in fruit and liquor flavors and the latter in rum, liquor, and butter flavors. Data, primarily from animal studies, suggest that small quantities of these alcohols do not produce toxic effects.

The following is a summary of the published effects of inhaling the ingredients in these products (5-8).

Central nervous system: light-headedness, weakness, nausea, ataxia, delirium, headache (both short-lived "nitrite headaches" and prolonged pulsatile headaches), syncope (following profound vasodilatation), tolerance, sedation, and anesthesia. (The last two effects are from reports of animals exposed to high concentrations.)

Cardiovascular: profound hypotension and cutaneous flushing followed by reflex vasoconstriction and tachycardia, and transient ECG changes (e.g., inverted T waves and depressed ST segments).

Hematological: methemoglobinemia (documented after oral and parenteral administration and repeatedly mentioned as a possibility after inhalation, but no reports were found when it occurred in association with the hedonistic use of these substances) and normocytic normochromic anemia after exposure in an industrial setting.

Ocular: increased intraocular pressure (reported but quite unlikely) and supraorbital pain.

Pulmonary: irritation (from reports of animals exposed to high concentrations).

We could find little information about the doses and duration of exposure necessary to produce the more serious adverse effects. Most of the toxicology literature on butyl nitrite and amyl and butyl alcohol refers to concentrations in the air breathed by industrial workers.

Research regarding N-nitrosation reactions (9) raises the issue of whether repeated use of these products could increase the risk of developing cancer. Inhaled nitrites could interact freely with endogenous trivalent nitrogen compounds to produce nitrosamines. Some nitrosamines are known to be carcinogens. It remains to be determined whether nitrosamines are formed after inhalation of these preparations. If so, are they carcinogenic, and how much exposure by inhalation would be required to produce them in amounts sufficient to increase the risk of cancer?

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Comparing Arrest Rates of Mental Patients and Criminal Offenders

BY HENRY J. STEADMAN, PH.D., DONNA VANDERWYST, M.A., AND STEPHEN RIBNER

The authors compared the arrest rates of former mental patients with those of criminal offenders released in the same jurisdiction. The data for all ex-patients and all offenders released in Albany County, New York, in 1968 and 1975 support the contention that it is the relationship between prior and subsequent arrests that explains the increasing crime rate of ex-patients and the three to six times higher rate of arrest of released offenders. Since the three-quarters of former patients with no arrest records were arrested about as often as the general population and substantially less often than offenders, care is warranted in drawing inferences from overall mental patient arrest rates.

IN THE PAST DECADE the literature dealing with the arrest rates of former mental patients (1–10) has burgeoned. This interest appears to be linked to concerns that expansion of community treatment programs has placed or kept in the community more persons who are violent or apt to be involved in criminal activity. In addition, this growth of research literature has been fueled by the finding in all studies published since 1965 that former patients are more often arrested for certain offenses than the general population. In all of these studies inferences have been drawn from differences observed between the patient rates and those of the general population in the states, counties, or catchment areas from which the patient samples were taken. Given our recent analysis of New York State data (11) suggesting that prior criminality rather than mental illness may be the root cause of these differences, it may be more instructive for the development of social policy to compare patient arrest rates with those of criminal offenders released in the same jurisdictions.

Despite the increasing number of studies about arrest rates of ex-mental patients, none has provided such comparisons. In this report, we will examine the experiences of two groups of released patients and two groups of released offenders in Albany County, New York, as a first step in determining of what use such comparisons might be in addressing the volatile issues of the criminal behavior of former mental patients.

METHOD

In order to make some preliminary assessment of the stability in any identifiable trends, we selected two groups of released mental patients and two groups of criminal offenders. We studied all Albany County residents released from New York State psychiatric centers in fiscal 1968 and fiscal 1975; there were 307 in 1968 and 204 in 1975. The criminal offenders studied were all the offenders released in fiscal 1968 and 1975 from the Albany County jail and all Albany County residents released in calendar 1968 and 1975 from state

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TABLE 1
Arrest Rates for Albany County Released Mental Patients and Criminal Offenders and New York State General Population

Group	1968 Arrests per 1,000		1975 Arrests per 1,000	
	Total	Violent Crimes	Total	Violent Crimes
General population	27.53	2.29	32.51	3.62
Ex-patients	77.98	2.05	111.18	12.35
Criminal offenders	267.84	22.63	695.00	87.50

TABLE 2
Arrest Rates for Albany County Released Patients and Criminal Offenders and New York State General Population, by Number of Prior Arrests

Group	Arrests per 1,000	
	In 1968	In 1975
General population	27.53	32.51
No prior arrests (patients)	27.63	37.89
One prior arrest		
Patients	52.50	120.00
Offenders	151.20	378.00
Multiple prior arrests		
Patients	297.82	302.40
Offenders	275.07	729.91

prisons. There were 167 offenders released in 1968 and 252 in 1975. All mental hospitalization records for both the mental patients and offenders were obtained from the Department of Mental Hygiene's data processing unit. All criminal records for both groups were obtained through the collaboration of the New York State Department of Criminal Justice Services.

The offender groups in both years were predominantly male (93% and 90%, respectively) compared to the released mental patient groups, which were 52% and 61% male. Likewise, the offenders tended to be younger and more often black than the mental patients. Since the purpose of this paper is to examine the comparative arrest rates of all released mental patients and all released offenders in the same jurisdictions, the potential impact of these group differences in the observed rates will not be examined. Our intention is simply to answer the question of how the rates of arrest of ex-patients and offenders compare.

FINDINGS

As is shown in table 1, as expected, in both 1968 and 1975 the released mental patients overall had substantially higher arrest rates than the general population. For both years, however, the offenders' rates were much higher than the mental patients'. In 1968 the mental patients' overall arrest rate was about three times that of the general population, but the offenders' rate was about three and a half times that of the mental patients. Also, in 1975 the mental patients' total arrest rate was about four times that of the general population, but the offenders' was six times that of the mental patients. For the most part, the same trends exist for violent crime except that in 1968 the mental patients were arrested somewhat less often than the general population. In both years the offenders were arrested much more often for violent crime than the mental patients. Because of the infrequency of violent crimes among the former patients, the limited size of our two samples did not permit more detailed analyses except for total arrest rates.

While the total rates reported in table 1 are important—particularly the consistently higher rates of arrest for offenders than ex-patients—there is a critical relationship for both the mental patients and offenders that these rates mask. As was found in our earlier work (11), among the ex-patients there were dramatic differences in arrest rates depending on whether they had been arrested prior to the current hospitalization. As is evident in table 2 this same relationship existed in the two patient samples from Albany County and among both the 1968 and 1975 offender groups. The ex-patients who had no arrests prior to their current hospitalization were arrested about as often as the general population. For both 1968 and 1975 those mental patients and offenders who had one arrest prior to their release were arrested substantially more often than the general population, and those with multiple prior arrests had exceedingly high arrest rates after release. Since all of the offenders had at least one prior arrest at the time of their releases, there are four combinations of year and number of prior arrests (one or multiple) according to which mental patients and offenders can be directly compared. In three of the four comparisons the offender group remains higher, but in the multiply arrested 1968 releasees the corrections group is lower. Because the trend toward higher arrest rates is completely consistent with a higher number of prior arrests, it seems that the relationship between prior and subsequent arrest explains the overall differences reported in table 1 in the offender and mental patient rates rather than something else associated with the offender.

In most comparisons between the arrest rates of mental patients and the general population, the mental patients will be more often arrested. This is primarily because a much higher percentage of those persons currently being released from state mental hospitals have been arrested previously than is the case among the general population, and it is true that in any population there is a very strong relationship between having been previously arrested and being subsequently arrested. Likewise, then, when the arrest rates of mental patients are compared with those of released offenders, it is not surprising that the offenders have consistently higher rates of arrest than the mental patients. The offenders all have been arrested at least once and we have seen that among mental patients with one prior arrest their rates usually slightly exceed those of the general population. Also, a much higher proportion of the offender group has multiple prior ar-

rests, which greatly increases the probability of future arrests.

DISCUSSION

These data drawn from one county in New York for two time periods would seem to support the conclusion that as the composition of state mental hospitals has changed to include more persons with prior police records, the rate of arrest of former mental patients has increased and now exceeds that of the general population. What the comparative data on the criminal offenders released at the same time into the same areas provide for the first time is documentation that, as the proportion of any population with previous criminal records increases, so too will their rate of future criminal activity. It would appear that the use of the arrest rate data of former mental patients in policy discussions revolving around the controversial issues of deinstitutionalization and the "dumping" of mental patients must be more specifically formulated in terms of subgroups of mental patients and the changing composition of the populations being released. Those mental patients who have not been arrested—approximately three-quarters of all released patients in New York—are arrested about as often as or less often than the general population. It is the multiply arrested mental patient who is often arrested after release. Thus, when a geographically comparable group of offenders is used for comparison, the rates of released mental patients become much less intimidating, and it becomes evident that there is a need for more appropriate points of reference than the general population for comparing the rates of ex-patients given the changing composition of state hospital clientele.

The issue is not so much that there are more mentally ill people at risk for criminal activity in the community; more accurately, the problem seems to be that there are more criminals in mental hospitals in the first place.

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Supplemental Security Income and the Sick Role

BY H. RICHARD LAMB, M.D., AND ALEXANDER S. ROGAWSKI, M.D.

Supplemental Security Income (SSI) has given the psychiatrically disabled a dependable source of income and helped make it possible for them to live outside state hospitals. The change to SSI has had an important effect on delabeling and decreasing the stigma of receiving Aid to the Disabled. However, the secondary gain inherent in SSI may interfere with social and vocational rehabilitation efforts. The authors present recommendations aimed at making the SSI system more conducive to social integration and rehabilitation.

FOURTEEN YEARS have passed since eligibility for Aid to the Disabled (ATD) was extended to the mentally ill. Over three years have elapsed since this program was transferred to the Social Security Administration and renamed Supplemental Security Income (SSI). Has a social, political, economic, and psychological phenomenon as important as this one received the scrutiny it deserves? The extension of eligibility for ATD, now SSI, to the mentally ill in 1963 had a major impact on psychiatry. What have been the benefits and what have been the adverse effects of this eligibility?

BENEFITS OF THE ATD TO SSI SHIFT

The beneficial consequences have been impressive. First and foremost, a source of funding became available that made it possible for the mentally ill who are unable to support themselves through work or private funds to move out of state hospitals and be maintained in the community. Previously, state governments had been willing to bear the cost of state hospitals, making only feeble and scattered attempts to provide funding for community alternatives. The frequently adverse consequences of prolonged state hospitalization were evident and were even made known to the public on occasion through newspaper exposés (1). However, the fear of the mentally ill and the public's apathy and desire to isolate them in locked and usually distant institutions prevailed. The advent of the phenothiazines and the community mental health movement made it more feasible for the mentally ill to live outside of the massive, impersonal institutions, but only the availability of ATD funds enabled resourceless persons, who had spent or would have been destined to spend many years in state hospitals, to be placed in a variety of community living arrangements. These ranged from good to poor, from independent living to board and care homes to convalescent hospitals. In some cases ATD payments made these patients more acceptable to their own families; before this financial support was made available the patients had sometimes been regarded primarily as an economic and emotional burden. Although there were many indignities to be suffered in the process of being approved for ATD, the end result was a dependable source of income for the mentally ill. We are convinced that many thousands of patients could have been discharged from state hospitals years earlier had ATD been available to them. Furthermore, the extension of welfare and income maintenance to the mentally ill meant that their needs were at last recognized; prior to this, ATD was available only for the physically disabled.

From the beginning, there were concerns about potential problems with this process, particularly in terms of officially labeling this group "totally disabled" and "mentally ill" and thereby stigmatizing them. Because of this, although the program was still called ATD, the abbreviation no longer stood for "Aid to the Totally Disabled" as it had initially but simply for "Aid to the Disabled." To indicate the possibility of recovery, a periodic review of eligibility was required by the regulations although it was usually performed only in a perfunctory way. While these changes were a step in the right direction, we think the term "disabled" was impressed on the minds of recipients, who generally continued to feel labeled and stigmatized.

The change to SSI, administered by the Social Security Administration, has had an important effect on delabeling and decreasing the stigma of ATD (2). A recent study (3) revealed a number of important effects of the shift from ATD to SSI. Most of the subjects in this study referred to themselves as being "on Social Security" or "on Social Security insurance," indicating a delabeling effect. Their financial support no longer clearly labeled them as psychiatrically disabled, and most no longer considered themselves to be on

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welfare. The term Social Security had a more normative sound to the recipients. Thus, SSI has resolved or partially resolved some important problems of the mentally ill. But have new problems been created?

There has been a mass exodus from state hospitals, and tens of thousands of state hospital patients have been discharged. However, it has come to be recognized that an appreciable number of them are still not really integrated into community life (4). They live impoverished, inactive lives "in the community," and are open to exploitation by a variety of persons who take advantage of their helplessness (5). Community mental health professionals have by and large neglected them, preferring to focus on preventive programs and on treating attractive, verbal neurotics and individuals with character disorders. Recently, new interest has developed in treatment and rehabilitation programs for long-term, severely ill psychiatric patients. A component of such programs is usually social and vocational rehabilitation designed to enable these patients to "join in the mainstream of society" and experience the satisfactions of participation and even production.

SECONDARY GAIN IN SSI

To what extent does the aspect of secondary gain inherent in SSI interfere with such an approach? Although much has been written about compensation neurosis, it is noteworthy that little has been said publicly about the corresponding, although more subtle, problems inherent in the SSI program. Perhaps we do not mind paying for mental illness as long as it keeps the patients quiet and out of sight. It is the thrust of this article that serious consideration should be given to all aspects of the impact of SSI on the psychiatrically disabled. Let us turn first to problems of vocational rehabilitation.

An event in Missouri highlights the disincentive effects of SSI. When ATD was made readily available for psychiatric patients, one-third of those enrolled in sheltered workshops precipitously quit (Hilary Sandall, personal communication, May 1976). Unfortunately, no follow-up study was done and we do not know how these persons fared in the long run. However, this event made the problem clear. Large sums of money are being appropriated for vocational rehabilitation for the mentally ill, but at the same time there is undermining of their already weakened resolve to overcome their apprehension and try to deal with the everyday demands of life, such as self-support and achievement of a measure of independence.

Frequently, although by no means always, the severely mentally ill have a reduced work capacity. For many, the routine demands of work and social relations are major stresses. So much effort goes into their struggle with illness that they have little energy left to deal with a work situation. Handling interpersonal relationships with supervisors and coworkers is a major stress. The very thought of work [redacted] it carries the risk of yet another failure. [redacted] or at least protective work setting and an adequate preparatory period of vocational rehabilitation need to be made available for many of these precariously compensated people.

Those who believe in the value of vocational rehabilitation stress that heightened self-esteem issues from experiencing oneself as productive, as making a contribution to society, and as achieving at least partial self-support and independence. Work therapy is recognized as being fully as important as talking therapy. Having no reason to get up in the morning and no structured day to look forward to causes great voids in the lives of the severely mentally ill. Rewarding and gratifying use of leisure time is equally important. A combination of work and play is both normative and curative.

How does this square with a system in which continued financial support at a relatively low standard of living is contingent upon the client's maintaining a sick status? When one includes Medicaid, food stamps, the supplement to the basic federal grant supplied by a number of states, the exemption from taxes, and the possibility of pooling one's resources with another recipient, the standard of living may not be so low. It is not surprising that a large percentage of the mentally ill remain "disabled" and totally (or almost so) dependent on SSI. Most of these persons could be rehabilitated only into low-paying sheltered employment or low-level jobs even if they succeeded in overcoming their fear of venturing forth into the world of work. Moreover, they fear that such employment will result in a reduction of their already marginal standard of living and fear that they will lose one of the cornerstones of what little security they have, their SSI status (6). The secondary gain of illness, already a problem with many long-term psychiatric patients, is thus further reinforced.

Case Example

Mr. A, a 26-year-old man, has been hospitalized three times with a diagnosis of paranoid schizophrenia. His social worker suggested that he apply for SSI during his second hospitalization, and he has now been on SSI for 2 years. He had held a number of low-level jobs, but none for more than 6 months and none in the last 2 years. He is now seeing a psychiatrist and takes antipsychotic medication on a regular basis. He was referred to a vocational rehabilitation counselor who felt Mr. A could handle a low-pressure job, providing that he remained in treatment. The counselor arranged for him to be interviewed for a job as groundskeeper at a golf course. The foreman there handles his employees well and has supervised ex-mental patients in the past with good results. The prospect of being a groundskeeper appealed to Mr. A. He liked outdoor work, and the job would involve minimal demands to interact with other people. He found staying home all day difficult and depressing; time hung heavy on his hands. He did not know how to answer when people asked what he did. Mr. A was interviewed and hired, but later that day he called the boss and quit the job. Why? Later he talked with his counselor about his fear of failure.

Despite reassurance from both his counselor and the Social Security office, he could not be convinced that he would not permanently lose his SSI if he accepted the job; he remembered well the bureaucratic red tape and interminable delays involved in getting SSI initially and then again when he was discharged from the hospital and had to be reinstated. Besides, Mr. A told his counselor, his net pay from the job (at minimum wage) would be little more than his SSI check (which includes the California supplement) plus food stamps. He would be taking quite a chance, and he was frightened. At this writing, 2 years later, nothing has changed. Mr. A refuses to see his vocational counselor and is depressed and immobilized.

The message given to the patient by the SSI system is fully as important as the financial remuneration. Essentially, he is told "you are sick, disabled, and unable to work." Rehabilitation may be mentioned, but usually it does not come through to the patient with any real conviction.

The SSI system thus does not counteract the pull toward regression and dependency. Its purpose is income maintenance, not helping the person act or feel like a contributing or significant member of society. In terms of his source of income, the person perceives that society expects little of him. If the recipients of SSI choose to do so, they have the means to live an undemanding life with family or in a board and care home. There is little incentive to participate in a treatment program, either social or vocational. For a significant proportion of schizophrenic patients who have ego strength sufficient only to cope with minimal demands and social stimulation (7), this may be entirely appropriate. However, such a system undermines the already reduced motivation of many mental patients, both schizophrenic and nonschizophrenic, who could benefit from rehabilitation programs.

WHAT CAN BE DONE?

A first step would be to screen persons very carefully for entry into the SSI system and send them in this direction only if they are truly disabled. A major problem is the pressure from local jurisdictions, cities, and counties to divert persons from general relief or general assistance (funded entirely by local money) to SSI (funded primarily by federal and state money). Persons who may need general relief only for a limited time of crisis find themselves propelled into a system where, despite the perfunctory yearly SSI review, they may remain for years or for life. Sometimes the mere fact that a person is in psychotherapy seems sufficient to a welfare department eligibility worker to initiate an SSI application for that individual, even though he may have functioned well in the recent past and is only temporarily disabled. Such inappropriate entries into the SSI system need to be reevaluated, and primary consideration should be given to the long-range interests of the patient.

Another way to reduce the occurrence of chronic regression in the SSI system is to make yearly SSI reevaluations more meaningful. Currently they consist of an essentially mechanical financial eligibility check, with little concern for the patient's nonmonetary needs. Instead, each patient should be carefully evaluated by rehabilitation professionals who would offer the opportunity of social and vocational rehabilitation and explore its applicability to the specific person, with sensitive assessment of his capabilities and limitations. Patients who are not likely to withstand the stress of such activities should not be pressured or made to feel guilty about not participating in them. Further, the opportunity to receive rehabilitation should be seen both by the patient and the Social Security Administration as a benefit, like Medicaid, not as a requirement, and the patient's participation —or nonparticipation— in rehabilitation programs should in no way affect his eligibility for SSI.

To minimize the disincentive effects of SSI and the sick role, the law should be liberalized so that SSI recipients are allowed to keep a greater share of earnings from employment; under the current arrangement, any significant amount of earned income is deducted from their SSI checks.

At this time, we cannot predict which of our patients will benefit from a social integration program and which might react adversely to such efforts therefore, we suggest that every person at the point of application and approval of SSI be given every opportunity to participate in a comprehensive treatment and rehabilitation program. Whatever services are indicated should be made available: individual and group psychotherapy, social and vocational rehabilitation, training, sheltered work settings, employment assistance day treatment, linkage to therapeutic housing programs, and social services. Patients should be monitored to ascertain how they respond to treatment and to inducements to greater social and occupational participation. If clients react adversely, manifest a recrudescence of symptoms, or seem refractory to active help, the professional should at any point in the process be able to reset their goals and accept the clients limitations and possibly their need for a passive, dependent lifestyle that will make minimal demands on them. With this perspective, professionals will leave the door open to treatment and rehabilitation opportunities in the future. In the meantime, they will offer the patient those services that he can or will accept, including the option to call at times of crisis or great stress. Efforts must also be made to upgrade residential facilities and bring treatment and supportive services to patients who are not ready to come to them.

Finally, we advocate discussion and reassessment of the unintended adverse results of a social policy that was conceived in a humanitarian spirit and has brought about so many favorable developments for the mentally ill. The topic is often avoided because so many professionals fear that even the raising of questions will support the arguments of those extremists who would abolish social programs because they consider

any social dependency immoral, even that brought on by psychiatric disability.

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The Mind-Body Dichotomy Reified: An Illustrative Case

BY MARK SCHOENBERG, M.D., MORTON G. MILLER, M.D., AND CONSTANCE E. SCHOENBERG

Cartesian dualism, the splitting of human existence into mind versus body, is at times reified with unfortunate consequences. The authors report a case in which a social policy formulation based on such diagnostic dualism resulted in the denial of health-related facility placement to a patient. They argue that this denial was prejudicial and illogical and that benefits should be related to a functional disability rather than to diagnosis.

THE CONCEPT of psychic dualism—of psyche as a separate entity from soma—has haunted psychiatry since its inception. We would like to report a case[1] that illustrates the reification of this concept in a matter of public policy and discuss the importance of such reification.

CASE REPORT

Mr. A was 38 years old when we saw him. He was born in New York State in 1938. He had a possible history of use of instrumentation at delivery. A diagnosis of mental deficiency as well as mucus colitis was reportedly made when he was 18 months old. At the time, he was known to bang his head. At the age of 3 he was brought to a major university teaching hospital and diagnosed as mentally deficient. Institutionalization was recommended when he was 4, and he was admitted to a private institution, where his IQ was found to be 47.

One year later he was transferred to a state facility with the diagnosis of mental deficiency, undifferentiated, imbecile; his IQ was 35. He was described as clean, tidy, and able to dress himself. He lived in a succession of state facilities for the mentally retarded until he was 21. At that time he was transferred to a state psychiatric hospital with auditory and visual hallucinations, hyperactivity, assaultive behavior supposedly in response to command hallucinations, and periods of withdrawal. A diagnosis of psychosis with mental deficiency was made, and treatment with neuroleptics was instituted. He was given phenytoin sodium (Dilantin) and phenobarbital for 3 years, although no specific mention was made of seizures. At the age of 30 he was said to be no longer psychotic. At the age of 33 he was discharged to a foster home and to outpatient clinic treatment. In 1975 his neuroleptics were stopped, and his psychotic symptoms did not recur.

The patient's family had no recorded history of mental retardation or major mental illness. Physical examination revealed that the patient was thin and had multiple burn scars from dropped cigarettes. There were no focal neurological findings, stigmata of chromosomal damage, or evidence of metabolic defect.

The mental status examination revealed that the patient was disoriented to time, place, and person. His speech was limited and concrete, and he had a markedly limited fund of knowledge. However, he could hold a coherent conversation on simple subjects. There were no Schneiderian first-rank symptoms, no hallucinations, no delusions, and no apoph-

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[1]This presentation was abstracted from the rather sketchy records of 30 years of institutionalization.

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anous phenomena. His affect was restricted, and his mood was apathetic.

While in the foster home Mr. A became increasingly confused and disorganized. He also started to have fecal and urinary incontinence. After a negative workup for organic causes, he was started on imipramine, 25 mg h.s., which improved but did not eliminate his enuresis.

After a home visit, a social worker judged the foster home unable to provide adequate supervision and a request was made for an application of transfer to a facility with both a health-related and a nursing home component. The request was based on the patient's continuing problems with hygiene and self-care, urinary and fecal incontinence, and worsening disorientation. The Department of Social Services denied a request for placement with the following note: "Deteriorating mental status does not justify Health-Related Facility status." Reevaluation was requested and disapproved on the basis of the following policy:

1. Placement of mental hygiene discharges in a . . . Health-Related Facility shall be prior approved by the local social services district Medical Director.
2. Patients with exclusively psychiatric diagnoses will not be approved for such long-term care.
3. A physical condition must exist which warrants care.
4. Not appropriate for . . . H.R.F. [health-related facility] because they [patients such as Mr. A] are primary [sic] mental patients. These facilities have no commitment to care for such patients, and there is no proof that they can take care of these patients.
5. We must assume priorities—physical vs. mental.

This decision was appealed. Several months later, the patient was admitted to a general hospital with stasis edema and cellulitis of the legs. He was then placed in a health-related facility on the basis of this physical condition. The appeal was denied on its merits.

DISCUSSION

Most of us speak and act as if our existence could be neatly divided into mind and body. We recognize that many schools of thought do not postulate a mind-body dichotomy, but we observe that most followers of these various schools usually behave in ways that imply or assume such a dichotomy. The field of psychiatry is polarized into psychodynamics against psychobiology. Psychology is polarized into humanists and social psychologists versus behaviorists, neuropsychologists, and psychobiologists. Psychiatric social work is usually identified with a psychic or mind position (attribution or labeling theory, psychological or social etiology theory). Caseworkers, however, are often relegated to a somatic position when their function is purely adjunctive to medical treatment. The lay public continues to stigmatize mental illness as different from and more shameful than physical illness. The legal profession, which is responsible for most of the policy making in our society, has been greatly influenced by Szasz, who, according to Roth (1), argues against the disease model of psychiatric illness by postulating a fixed mind-body split.

We would argue that the mind-body split is a false dichotomy. Human mental function, conscious and unconscious, is integrally connected with the function of the brain in the milieu of the body, and the integrity of brain and body is inextricably related to mental function. Evidence from the fields of genetics (2–4), neurophysiology (5), physiology (6, 7), and epidemiology (8, 9), as well as neurotransmitter hypotheses and stress-diathesis and multifactorial theories, all point to the influence of soma on psyche. Conversely, the elucidation of the function of the hypothalamic-pituitary axis, the theoretical basis of somatopsychic medicine, as well as specific works on life stress (10), psychosocial dwarfism (11), and emotional influences on physical disease (12), all point to the consequence of psyche and soma. It is also worth noting that *DSM-II* (13), the supposed basis for mental diagnosis, contains many conditions that are physically based, such as organic brain syndrome, psychoses associated with cerebral conditions, and psychophysiological reactions. It is on this basis that we reject the concept of a mind-body dichotomy.

The case presented is even more poignant because, even if we postulate a mind-body dichotomy, Mr. A most likely suffered from a physical illness. The severity of his illness, its early onset, the possible occurrence of seizures, his visual hallucinations, and his increasing disorientation all point to an organic syndrome. In addition, the patient's need for nursing care was due to his physical problems of incontinence and inability to take care of himself. For this reason, the denial of care on the ground that the patient had a mental condition strikes us as illogical and prejudicial.

Current proposals to accelerate community placement of individuals with mental retardation (the "Willowbrook Decree") and chronic mental illness will exacerbate the consequences of the inconsistency in current social policy formulations exemplified by the case of Mr. A. In our experience, individuals with a primary diagnosis of mental retardation with a history of even one psychotic episode and subsequent hospitalization in a state mental hospital are doubly stigmatized. Frequently rejected by community programs for mental retardation because of their history of mental illness, they are likewise often deemed not suitable for programs geared to the needs of individuals with functional psychoses.

Federal policy provides that services rendered under Medicaid should not discriminate by diagnosis. In the case cited, clinical facts were ignored in favor of a philosophical construct of mind-body dualism. The patient was penalized for his failure to fit neatly into a "clean" category. Continued deterioration of his mental status would almost certainly have resulted in his rehospitalization in a mental hospital. Although this result is clearly not consistent with the precept of least restrictive alternative, it looms frequently as the inevitable consequence of these dichotomous policies.

We would recommend that social policy reflect the unity of mind and body. "Mental" patients should not

be denied benefits available to patients with "physical" illness, and "organic" patients should not be excluded from the programs of psychosocial rehabilitation available to "mental" patients. As a matter of social policy, programs and benefits should be geared to patients' functional disabilities—not to their diagnoses.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Mental Disturbances During Bromocriptine and Lergotrile Treatment of Parkinson's Disease

BY MICHAEL SERBY, M.D., BURTON ANGRIST, M.D., AND ABRAHAM LIEBERMAN, M.D.

In recent years there has been increasing support for the hypothesized role of dopaminergic hyperactivity in schizophrenia (1). Schizophreniform psychoses characterized by paranoid ideation, delusions, inappropriate behavior, and, in some cases, hallucinations and disorders of thinking and affect have been observed after the administration of a variety of drugs that have a common denominator of dopaminergic facilitation (2). Organic confusional syndromes characterized by temporal and spatial disorientation, agitation, and delirium have also been observed (3, 4).

Bromocriptine and lergotrile are newly developed dopamine agonists that have shown promise in the treatment of Parkinson's disease (5–7). Early clinical trials demonstrated mental disturbances as a side effect of both of these drugs (5–9). In view of this we thought it would be of interest to report our recent experience with a larger series of parkinsonian patients treated with these agents.

Method

Between 1974 and 1977, 81 patients with advanced Parkinson's disease who gave written informed consent were treated with either bromocriptine or lergotrile (7, 8). Some received trials of both agents; 66 were treated with bromocriptine (mean dose=33±2.6 mg) and 53 received lergotrile (mean dose=39±3.1 mg). Bromocriptine or lergotrile was added to each patient's regimen at an initial dose of 5–10 mg/day. The daily dose was increased by 5–10 mg each day. During the course of the study, increased abnormal involuntary movements (AIMs) made it necessary to reduce the dose of L-dopa by 10% in both drug groups. The dosages of amantadine and/or anticholinergics were kept constant throughout the study.

Patients with moderate or marked organic mental syndromes were excluded. Patients with mild organic mental syndromes or histories of depression or psychotic behavior were included if their psychiatric condition was stable. All such patients underwent an EEG and a psychiatric evaluation before they were committed to the study.

Among the 66 patients treated with bromocriptine 17 developed mental changes. In 14 patients the mental changes necessitated discontinuation of the drug; in 5 cases the changes were severe enough to require additional psychiatric consultation. In 3 of the 17 patients the mental changes were transient and disappeared when the dose of bromocriptine was reduced. Nine of the 17 patients had exhibited similar changes on L-dopa. Among the 53 patients treated with lergotrile, 13 developed mental changes. In 12 patients the mental changes necessitated discontinuation of the drug, and additional psychiatric consultation was required in 5 cases. In 2 of the 14 patients, the mental changes were transient. Eight had exhibited similar changes on L-dopa. The 10 patients with severe mental changes on bromocriptine or lergotrile form the basis of this report. Statistical analyses were performed using Student's one-tailed t test at the 95% confidence level.

Results

Comparative data on the 10 patients whose mental changes on bromocriptine or lergotrile were severe enough to require additional psychiatric consultation

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TABLE 1
Clinical Data on 10 Patients Receiving Bromocriptine or Lergotrile

Patient	Age (years)	Sex	Parkinsonism Duration (years)	Parkinsonism Stage	L-Dopa Treatment (years)	AIM	Psychiatric History	Bromocriptine Dose (mg)	Lergotrile Dose (mg)	Mental Changes*
1	72	M	25	4	8	None	Mild organic mental changes	25		OCS
2	72	M	13	4	9	None	None	15		OCS
3	73	M	8	4	8	Minimal	None	80		OCS
4	52	M	8	3	8	None	Depression	50		SP
5	53	F	12	3	7	None	Depression		50	SP, periods of catatonia
6	63	F	14	3	8	Marked	Hallucinations with amantadine		15	SP
7	64	F	13	4	9	Marked	None	30		SP
8	58	M	8	3	7	Marked	None		50	Florid paranoid psychosis, mild confusion
9	74	M	11	4	5	Minimal	None		60	Personality change
10	64	M	5	3	4	Minimal	None		85	Personality change

*OCS=organic confusional syndrome, SP=schizophreniform psychosis. All schizophreniform psychoses occurred in a clear sensorium.

are summarized in table 1. Further clinical details of drug response patterns are available from the authors on request.

Among these 10 patients, 3 developed an organic confusional syndrome and 4 showed a schizophreniform psychosis consisting of paranoid ideation, delusions, and hallucinations in a clear sensorium. One patient showed mental changes with characteristics of both syndromes. In this patient the organic confusional features were mild, and paranoid features (delusions of being poisoned, filmed, and tape-recorded) dominated the clinical picture. The remaining 2 patients showed more subtle changes in behavior and personality without confusion that were noted by families but not by the hospital staff.

The mean age (±SEM) of the 3 patients exhibiting the organic confusional syndrome was 72.3±0.3 and that of the 6 patients with nonconfusional syndromes was 61.6±3.3, a significant difference. In addition, 2 of the 3 patients who developed mental changes in a clouded sensorium had moderately abnormal pretreatment EEGs consisting of frequent 3–7 Hz slow waves bilaterally. In contrast, only 1 of the 6 patients who developed mental changes in a clear sensorium showed an EEG abnormality, and this was only minimal, consisting of 5–7 Hz slow waves accentuated over the left temporal region. The patient who showed both mild confusional and florid paranoid symptoms had a minimally abnormal EEG, with infrequent 5–7 Hz slow waves bilaterally.

There was no correlation between the type of mental disturbance and the duration or severity of parkinsonism, the duration of treatment with L-dopa, or the development of abnormal involuntary movement on L-dopa. One of the patients who developed an organic confusional syndrome on bromocriptine had a mild organic mental syndrome before treatment was initiated, and 2 of the patients who developed schizophreniform psychoses had previously exhibited depression sufficiently severe to require psychiatric intervention but not hospitalization.

Discussion

In this group of patients there was a relationship between antecedent organic mental changes and the development of organic confusional syndromes on lergotrile or bromocriptine. Not only was the group with the organic confusional syndromes older, but 2 of these 3 patients had a mild organic syndrome before they were given bromocriptine or lergotrile. Two of these patients also had moderately abnormal EEGs before they were given the drugs. This finding of an increased incidence of organic confusional syndrome in patients with an underlying dementia is consistent with previous observations. In 1971, Goodwin and associates (3) reviewed all the published studies on L-dopa in Parkinson's disease in which the nature of psychiatric side effects was specified (predominantly patients without dementia) and found confusion or delirium reported in 35 of 1,045 patients (3.35%). In contrast, Sachs and associates (10) administered L-dopa to 15 parkinsonian patients with specifically documented severe dementia, and all developed organic confusional syndromes.

Although our 10 patients were receiving drugs other than bromocriptine and lergotrile, the temporal relationships between bromocriptine or lergotrile administration and the emergence of symptoms suggest that these drugs were responsible. This impression is supported by the fact that such reactions were noted in

previous studies with these drugs (5–9) and by the known capacity of a variety of other dopaminergic agonists to induce similar psychiatric disturbances.

Thus, two main conclusions emerge from these observations. First, lergotrile and bromocriptine, like other drugs that facilitate dopaminergic activity, can in some patients act as specific psychotomimetic agents producing paranoid schizophreniform psychoses. This particular finding constitutes additional support for the hypothesized role of dopaminergic overactivity in schizophrenia. Second, these drugs, like many other agents, can also produce in some patients a nonspecific organic confusional syndrome, with age and preexisting dementia as predisposing factors.

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Tardive Dyskinesia Reinduced by Lithium Carbonate

BY BERNARD D. BEITMAN, M.D.

Reports concerning the short-term effect of lithium salts on the symptoms of tardive dyskinesia include a number of apparently contradictory findings. In studies of 6 and 15 patients, respectively, Reda and associates (1) and Gerlach and associates (2) found mild improvement in dyskinetic movements. However, in two separate studies of 10 patients each, Simpson and associates (3) found no significant change. In a single case report Crews and Carpenter (4) described an aggravation of tardive dyskinesia at toxic doses of lithium, whereas Gerlach and associates (2) reported complete remission of dyskinetic movements in a patient at toxic doses of lithium.

These studies have involved interaction of lithium and tardive dyskinesia over periods of 12 weeks or less. The following case report suggests that lithium may reinduce a dyskinesia that has been quiescent for many years.

Case Report

The patient, a 56-year-old woman, had had recurrent depressive episodes since 1960. These episodes were characterized by dysphoric mood, lack of energy, 3–4 hours of sleep each night with early morning awakening, decreased appetite, and weight loss. She was treated with most of the tricyclic antidepressants, numerous courses of ECT, neuroleptics, monoamine oxidase inhibitors, and minor anxiety agents. Only short courses of ECT were consistently effective. However, the patient became afraid of possible organic impairment resulting from ECT. After reading about the effectiveness of lithium in certain types of depression she came to the psychiatric medication clinic at the University of Washington seeking a lithium trial.

Of note in her medication history is the fact that she took thioridazine, 25 mg b.i.d., from January 1967 to March 1973. She discontinued the medication because she began to develop facial contortions: "My mouth was moving. All the time grimacing, my lips moved a lot, and I couldn't sit with a composed expression on my face." Within 2 weeks after she stopped taking the thioridazine, the oral-facial dyskinesia ceased.

The patient was stabilized on lithium carbonate during December 1975 at a dose of 300 mg t.i.d. with a serum lithium level of 0.9 mEq/liter and experienced no recurrence of depression when she was taking the drug. She decided to stop taking lithium in March 1977 "to see if it was the lithium or my belief in it." Within a week she began to redevelop symptoms of depression. After a few weeks of increasing discomfort she resumed the lithium and regained her normal mood. In June 1977 she began to experience a mild oral dyskinesia that seemed to her very similar to the dyskinesia associated with thioridazine. Her neurologist reported "occasional grimacing and spontaneous facial movements which increased when specific tasks involving the extremities were requested." The patient described a feeling that her "lip-

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weren't right, they were tight," in addition to the uncontrollable grimacing.

During a short trial of trihexyphenidyl the dyskinesia ceased immediately; later, it returned when the anticholinergic was discontinued. After being stabilized on 2 mg of trihexyphenidyl t.i.d. for 3 months, she experienced a mild recurrence of symptoms. In order to test the effects of the medications on her dyskinesia within the limits she would tolerate, she was asked to stop taking the trihexyphenidyl for 2 days and then to discontinue the lithium for 4 days. Without the anticholinergic, the dyskinetic movements worsened, and when both drugs were discontinued, they worsened further. On resumption of both medications, the dyskinesia was much reduced but still present. The most effective course of treatment has yet to be defined.

Discussion

The physical signs of the movement disorder, its time of appearance, and the exacerbation of dyskinetic movements on withdrawal of the implicated medication are characteristics of neuroleptic-induced tardive dyskinesia. Two interrelated explanations may be offered for the role of lithium in this case. First, lithium may simply unmask certain pathological conditions (5), suggesting that the previous neuroleptic administration had created an abnormal neurochemical substrate on which the lithium acted to cause the clinical picture. Second, lithium salts may reduce dopamine activity in a manner parallel to but weaker than the neuroleptics. For example, lithium may accelerate the reuptake of dopamine by synaptosomes, thereby reducing the total amount of dopamine (6). This neuroleptic-like activity of lithium might explain the short-term amelioration of tardive dyskinesia by lithium (1, 2) as well as the appearance of dyskinetic movements later in lithium therapy, as occurred in the case reported here. There have been no reports of lithium alone being associated with the appearance of tardive dyskinesia, so the best explanation may be a weak neuroleptic-like activity combined with a preexisting pathological substrate.

In reviewing this case report one must also consider the possibility that the dyskinetic movements might have recurred without the influence of lithium carbonate. Unfortunately, too little is known about the natural history of those tardive dyskinesias which remit on withdrawal of the offending medication. The temporal association of lithium with the reappearance of the dyskinetic movements suggests a causal connection. This connection implies that lithium, like neuroleptics, might have to be discontinued to prevent the dyskinesia from becoming chronic.

It should be noted that trihexyphenidyl was used instead of, for example, deanol because evidence is mounting that there are at least two pharmacologically distinct subtypes of tardive dyskinesia. In one type there may be a relative dopamine excess, while in the other there may be a relative cholinergic excess (7). The existence of multiple pharmacologic subtypes may also explain the reported variety of responses to lithium salts. At present, the only way to differentiate the subtypes is through pharmacologic trials with potentially helpful medications, including deanol and the anticholinergics.

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Successful Treatment of Thalamic Pain with Apomorphine

BY DENNIS P. MILEY, M.D., ALAN A. ABRAMS, M.D., J. HAMPTON ATKINSON, M.D., AND DAVID S. JANOWSKY, M.D.

Evidence exists suggesting a role for a catecholamine neurotransmitter, dopamine, as a moderator of analgesia and pain perception. L-Dopa, a dopamine precursor, elevates pain thresholds (1), and amphetamine, an indirect dopamine agonist, increases morphine analgesia (2). Also, in animal models, opiates stimulate dopamine biosynthesis (3). Apomorphine, a ligand for dopamine receptors, is structurally related to dopamine and acts directly as a dopamine receptor agonist (4, 5). Based on the hypothesis that apomorphine might have analgesic properties, we tested the effect of apomorphine on thalamic pain.

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Case Reports

Case 1. A 24-year-old man with dystonia musculorum deformans was hospitalized because of intractable pain. Two bilateral cryothalamectomies (2 and 3 years previously) ameliorated the patient's movement disorder, but a severe left-sided dysesthesia developed after surgery. Over a 2-year period, attempts to achieve analgesia with opiates, anticonvulsants, neuroleptics, and tricyclic antidepressants were unsuccessful, as was implantation of a dorsal column nerve stimulator.

At the time of his admission, the patient was bedridden, complaining of excruciating left-sided pain. Mild dysarthria, spasmodic torticollis, myoclonus, and severe left arm flexor spasm were present. Sensory examination demonstrated increased threshold to vibration, pinprick, and touch, as well as dysesthesia in the left arm. EEG findings were unremarkable. The patient's pain was refractory to intramuscular meperidine in a dosage of 200 mg every 3 hours.

Meperidine detoxification was accomplished by methadone substitution and withdrawal, and the patient was sedated with chlorpromazine. Thalamic pain syndrome was independently diagnosed by the neurology service on the basis of sensory loss associated with hyperpathia, intractable pain, and movement disorder.

Sequential drug trials were initiated for pain management. Drug trials were evaluated by 1) pain estimate and visual analogue scores as described by Sternbach (6), 2) behavioral observations recorded daily by both nursing and physician staff, 3) the McGill-Melzack Pain Questionnaire, and 4) the Profile of Mood States (POMS), all administered three times weekly.

Chlorimipramine, 5 mg/kg p.o. for 2 weeks, led to an intensification of pain complaints, deterioration in mood, manifest agitation, and a fourfold increase in chlorpromazine requirements from 300 to 1200 mg p.o. q.d. Therefore, chlorimipramine was discontinued and amitriptyline and chlorprothixene were started. Shortly thereafter, the patient had a left aversive seizure. An EEG demonstrated an irritative right anterior and parasaggital focus, so diphenylhydantoin was started. His severe, constant pain was unabated so amitriptyline and chlorprothixene were stopped after 10 days. Transcutaneous nerve stimulation afforded no relief.

Treatment with up to 5 mg/kg p.o. q.d. of apomorphine for 10 days resulted in a decrease of approximately 40% in pain estimate and visual analogue scores over baseline (from a weekly average score of 80/100 to 45/100), associated with enhancement of vigor (POMS vigor raw score increased from a pretreatment score 10 to 17 on apomorphine, and the T score increased from 48 to 59, $p<.01$ for both comparisons). There was also some alleviation of depressed mood: the POMS depression raw score decreased from a pretreatment score of 20 to 12, and the T score decreased from 49 to 43 (p=n.s.). The patient's chlorpromazine requirement decreased to 200 mg q.h.s., given for sedation. By the end of the 10-day study period, the patient could function independently, walk with a cane, and actively participate in recreational and physical therapy. As his pain complaints diminished, his self-care and interpersonal relationships improved. Apomorphine-induced pain reduction was not reversed by naloxone, 0.2 mg I.V.

Case 2. A 55-year-old woman was transferred from another hospital for treatment of thalamic pain secondary to a right cerebrovascular accident suffered 4 months previously. Drug trials (carbamazepine, pentazocine, and diphenylhydantoin) and transcutaneous nerve stimulation had failed to moderate the patient's pain. Her medications at the time of transfer included flurazepam, diazepam, pentazocine, phenobarbital, and diphenylhydantoin.

The patient complained frequently of intense pain and exhibited extreme lability of mood. Left-sided hemianopsia and palsies of cranial nerves 7, 11, and 12 were present. Hemiplegia, hyperreflexia, ankle clonus, and a flexor plantar response were also present on the left. Appreciation of all sensory modalities on the left was profoundly diminished, with hyperpathia to light touch and cold.

Analgesic and psychoactive medications were stopped. She had had cortical seizures in the interval since the cerebrovascular accident, so the anticonvulsants were continued. An EEG demonstrated right hemispheric grade 2 dysrhythmia. A trial of chlorpromazine treatment for her thalamic pain was aborted because of severe anticholinergic side effects. Subsequently, the patient had another seizure, so carbamazepine was restarted and diphenylhydantoin was discontinued because of subtherapeutic serum levels.

Apomorphine, 0.5 mg subcutaneously every 6 hours for 3 days, was given in a double-blind, crossover design. When compared with placebo (subcutaneous saline solution) apomorphine resulted in resolution of spontaneous pain complaints, reduction in subjective estimates of pain intensity and frequency, a predominance of pain-free intervals, and increased tolerance for physical therapy. Cognitive deficits precluded quantification of pain rating and mood state.

Discussion

Conventional analgesic treatment routinely fails in the management of thalamic pain. Phenothiazines may afford some relief (7) and diphenylhydantoin is sporadically effective (8). Stereotaxic thalamotomy is of benefit, but in the majority of cases pain recurs (9). In the patients we have described, despite pharmacologic management (including phenothiazines and diphenylhydantoin) and prior neurosurgical intervention in the first case, sustained analgesia was not achieved before or during hospitalization until apomorphine was added to the treatment.

Apomorphine has been used medically for its central emetic effect. Advantage has also been taken of the drug's dopaminergic potentiation in the treatment of Parkinson's disease (10) and neuroleptic-induced movement disorders. Although it is a morphine derivative, apomorphine lacks intrinsic narcotic analgesic effects. Autoinhibition and reversible azotemia at high doses limit the clinical use of apomorphine (10); however, neither of these side effects was observed in these cases.

Although it is possible that drug interactions with apomorphine resulted in analgesia or that changes in mood may have altered pain tolerance, we believe that apomorphine was helpful in controlling these patients' chronic pain. Since the pain was unresponsive to multiple treatment modalities, it is unlikely that the apomorphine effect was a placebo response. Further studies of apomorphine's usefulness in the treatment of thalamic pain are in progress.

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Intravenous Diazepam in the Treatment of Neuroleptic-Induced Acute Dystonia and Akathisia

BY DINSHAH GAGRAT, M.D., JAMES HAMILTON, M.D., AND ROBERT H. BELMAKER, M.D.

Dystonia and akathisia are two major extrapyramidal side effects of neuroleptic treatment. Dystonia, characterized by acute spasm of a muscle group, usually occurs during the first few days of neuroleptic treatment. Although it may remit spontaneously, dystonia is considered a medical emergency, and treatment with intravenous diphenhydramine is usually recommended (1). A major danger of diphenhydramine treatment is urinary retention or exacerbation of glaucoma or cardiac arrhythmias in predisposed patients (1).

Akathisia is an inner feeling of restlessness that may be accompanied by pacing and inability to sit still. It usually develops after weeks of neuroleptic treatment but is often seen earlier with the more modern high-dosage or parenteral neuroleptics. Standard treatment has been to administer anticholinergics orally, which does not take into account the often acute nature of akathisic restlessness. In addition, anticholinergics are often only partially effective in this syndrome (1) and carry the risk of the side effects mentioned above.

Diazepam has been anecdotally reported to be effective in neuroleptic-induced dystonia and akathisia (2–6). However, controlled study is necessary because these syndromes are often self-limiting or responsive to suggestion. Recent knowledge of the interactions of dopamine, acetylcholine, and GABA in the basal ganglia (7) make it seem logical that a GABA agonist like diazepam (8) might be equivalent to an anticholinergic in the alleviation of syndromes that result from dopamine receptor blockade. We therefore performed a double-blind trial of intravenous diazepam versus intravenous diphenhydramine in the treatment of acute neuroleptic-induced dystonia and akathisia.

Method

Subjects were selected from patients who developed acute dystonia or acute akathisia requiring immediate (intravenous) relief. Patients were excluded from the study if they were already on anticholinergic or benzodiazepine medication or had serious physical illness, especially glaucoma, prostatic hypertrophy, or cardiac or pulmonary disease.

Subjects were rated on a 4-point scale for dystonia or akathisia before the intravenous treatment and then 5 minutes, 15 minutes, 30 minutes, and 120 minutes afterward. The scale was defined as follows: 0=no dystonic or akathisic symptoms, 1=subjective feeling of muscle tightness or desire to contort (dystonia) or subjective inner restlessness (akathisia), 2=objective muscle spasm (dystonia) or objective restlessness (akathisia), and 3=severe objective muscle spasm (dystonia) or severe objective restlessness (akathisia). Solutions of diazepam (5 mg) or diphenhydramine (50 mg) were given slowly over 1 minute in syringes covered to conceal the different colors. Syringes were prepared according to a prearranged random order by one of us (R.H.B.) and administered by another author (D.G.), who was blind to the nature of the solution and who made the clinical rating observations. In addition to the symptom ratings noted above, D.G. rated drowsiness (absent, mild, severe) at each time point. After acute intravenous treatment the patient was usually started on oral anticholinergic medication for one to two weeks as clinically indicated according to the recommendations of Bassuk and Schoonover (1). Patients became eligible for re-study if the symptom re-

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TABLE 1
Effects of Diphenhydramine and Diazepam on Akathisia and Dystonia Severity Ratings

Symptom and Treatment	Number of Treatments	Mean Severity Rating*				
		Baseline	5 Minutes	15 Minutes	30 Minutes	120 Minutes
Dystonia						
Diphenhydramine	10	2.7	.70*	0.00	0.00	0.00
Diazepam	10	2.5	.70*	0.10	0.10	0.10
Akathisia						
Diphenhydramine	11	2.36	1.09*	0.82	0.55	0.64
Diazepam	9	2.22	1.44*	1.11	0.89	0.56

*All values for posttreatment severity ratings represent significant improvements over baseline ratings ($p<.01$, paired t tests).

turned after cessation of anticholinergics or if they had been treated for dystonia but later developed akathisia. Some patients were studied during more than one admission. Thus, a total of 40 intravenous treatments were given to 27 patients. Of these 27 patients who developed neuroleptic side effects and satisfied the other criteria for conclusion in the study, 26 were treated with haloperidol. This figure represents 30.6% of the patients treated with haloperidol in our unit over the period of study.

Results

Table 1 shows the effect of intravenous diazepam and diphenhydramine on the mean severity ratings of dystonic or akathisic symptoms. Both treatments are effective in both syndromes, with a significant reduction in symptom scale scores by 5 minutes posttreatment ($p<.01$, paired t test) that continued through 120 minutes. There were no significant differences in effectiveness between the two treatments at any time point. Drowsiness rating scores did not significantly differentiate between the two treatments (data not shown), although there was a trend for diazepam-treated patients to be drowsier.

Discussion

The present study suggests that intravenous diazepam is as effective as a standard anticholinergic drug in the treatment of acute neuroleptic-induced dystonia and akathisia. Three factors have made these neuroleptic side effects increasingly important: 1) increasing use of high-potency neuroleptics with considerable potential for inducing extrapyramidal disorders, 2) the spread of rapid tranquilizing techniques, which involve parenteral administration and rapid dosage increments, and 3) the realization that prophylactic use of anticholinergics may interfere with the efficacy of neuroleptic therapy (9) or cause another set of unwanted side effects. Safe and rapid treatment of extrapyramidal disorders has thus become increasingly important, especially for dystonias and akathisias, which are frightening to patients and may affect their future compliance with neuroleptic treatment (10). Diazepam seems to be an effective treatment for these syndromes and has the advantage of being familiar to psychiatric personnel, perhaps more so than diphenhydramine, and of being safe for patients with glaucoma or prostatic hypertrophy. Low doses of intravenous diazepam are sufficient, although the drug should be given slowly over the course of 1 minute to avoid danger of apnea. Although we did not see any instances of apnea with this low dosage, and apnea is very rare in patients who are not receiving other sedatives, clinicians should be aware of this possible risk when administering intravenous diazepam to patients with pulmonary disease, alcohol intoxication, or multiple sedative treatment.

Our study does not provide data on the use of diazepam orally over days or weeks in the follow-up treatment of patients with dystonia. For example, a patient with complicating severe prostatic hypertrophy would require follow-up treatment with diazepam because diphenhydramine and anticholinergics would be contraindicated. Our clinical experience suggests that it may be effective. Diazepam does not seem to act merely as a sedative in akathisia or as a muscle relaxant in dystonia as we have also seen cases of severe neuroleptic-induced akinesia, approaching stupor, that have responded to intravenous diazepam. Recent data on the interaction of diazepam with GABA in the brain suggest that the mechanism of action may be specific and central (8) and that diazepam may have interesting and important interactions with the neuropharmacology of schizophrenia.

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Hyperthermia Associated with Chlorpromazine and Full-Sheet Restraint

BY PHILIP GREENLAND, M.D., AND WILLIAM H. SOUTHWICK, M.D.

Disturbances of thermoregulation are well-recognized side effects of phenothiazine tranquilizers and constitute some of the most serious reactions to these drugs. There have been reports of both hypothermia (1), especially in elderly patients during winter months, and hyperthermia (2), generally associated with high ambient temperatures and/or excessive physical activity. A number of reports of fatal heat stroke in association with phenothiazine emphasize the clinical significance of these thermoregulatory abnormalities (3).

We report here the case of a patient in whom the use of full-sheet restraint led to profound hyperpyrexia. Chlorpromazine appeared to be the only drug the patient had received that had the potential for harmful effects on thermoregulation. To our knowledge, this association has not previously been recorded in humans.

Case Report

The patient, an 18-year-old man, was admitted to the psychiatry service of Strong Memorial Hospital because of acute agitation and combative behavior. He had been in excellent health until he became depressed, withdrawn, confused, and irritable 2 weeks before his admission. Five days before admission, he developed a blank stare and delusional, paranoid thinking. He also expressed considerable guilt about recent frequent marijuana use. He did not use any other licit or illicit drugs, including alcohol. On the day of admission he became quite agitated and combative and was brought to the emergency room by his family.

The family history was remarkable in that his father had died suddenly of a myocardial infarction almost exactly 2 years before the patient's admission. His medical and psychiatric history was negative and he had no known allergies.

The initial diagnosis was schizophrenia, paranoid type. The patient was given chlorpromazine, 400 mg/day, which was gradually increased over 7 days to 2000 mg/day. He was intermittently secluded during this time because of combative behavior, but no other drugs were used and he was not restrained. A complete physical examination could not be done during this time, but vital signs, including temperature, were consistently normal.

On the 6th day of his hospitalization a full-sheet restraint was ordered because of severe agitation and self-destructive behavior, most notably tongue biting and flinging his body against the walls of his room. The Posey "restraint net," made of heavy nylon mesh, exposed only the head, arms, and lower legs. He remained agitated and thrashed about despite the restraint. Five hours later, the patient was diaphoretic, and his axillary temperature was 104.6°F. He had received one 10-mg dose of haloperidol I.M. approximately 20 minutes before the fever was recorded. He was on no anticholinergics and ambient room temperature was 70°F.

An examination was remarkable only for the fever and diaphoresis in addition to continued agitation. He was placed in a cool bath and his temperature rapidly returned to normal. Cultures of urine, blood, and sputum were all negative; his white cell count was 10,000 with a normal differential; and a chest X ray obtained the next morning was normal. His T_4 was also normal.

The next morning he was again put in restraints as a precautionary measure, but he was not at all active and vital signs were normal. A complete physical examination was unremarkable. He remained afebrile and calm, and the restraints were removed until approximately 6:00 p.m. when he became agitated and was again placed in full-sheet restraint. At 8:00 p.m. his axillary temperature was 105.0°F. He was subsequently given 5 mg of haloperidol I.M., but he had not received the drug previously that day. Physical examination and all laboratory measures were unchanged, and a cool water bath again led to rapid resolution of his hyperpyrexia.

Throughout the remainder of his hospitalization the patient received no additional phenothiazines, and his agitation and psychotic behavior were well controlled on haloperidol and benztropine mesylate. Restraints were no longer required, and fever did not recur.

He was discharged after 49 days of hospitalization with the diagnosis of acute schizophrenic episode and possible schizophrenia, paranoid type. He has remained well during 3 months of follow-up on low doses of haloperidol.

Discussion

Thermoregulation in humans is thought to involve a complex interaction between central and peripheral regulatory mechanisms. Central mechanisms probably occur in the caudal hypothalamus (4) and are mediated by both dopaminergic and serotonergic receptors (5). Peripheral mechanisms include vasoconstriction and dilatation, piloerection, and sweating, leading to increased or decreased heat loss by convection, radiation, or evaporation (4). Hyperthermia occurs when heat production exceeds heat loss by these various mechanisms (4).

A number of drugs are known to disturb these thermoregulatory mechanisms (6–8). The phenothiazines may affect thermoregulation by both central and peripheral mechanisms, although the impairment of hypothalamic thermoregulation is probably the major pathogenic mechanism (5). Severe hypothermia and fatal episodes of hyperthermia have been recorded in patients treated with phenothiazine. The hyperthermia is invariably associated with elevated ambient temperatures and/or excessive physical activity in humid en-

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vironments (2, 8). The risk of hyperthermia with phenothiazines is increased when phenothiazines are given in combination with anticholinergics, which impair sweating and thus abolish a potent mechanism of heat loss (9).

The patient we have described demonstrated profound hyperpyrexia on two occasions shortly after he was placed in full-sheet restraint. On one occasion, after 7 days of high-dose chlorpromazine therapy a single intramuscular injection of haloperidol was given 20 minutes before the fever was recorded. The second febrile episode occurred when the patient was receiving chlorpromazine but no other medications. It would thus appear most likely that chlorpromazine was the drug involved, although haloperidol has also been known to produce thermoregulatory impairment (5). The significant and previously unreported feature of this case is the association of fever with restraint in a patient treated with a phenothiazine. The presumed mechanism of fever was a chlorpromazine-induced abnormality of central thermoregulation, combined with the inability to evaporate sweat due to the heavy nylon restraint, a situation analogous to that of a patient who is receiving both chlorpromazine and an anticholinergic. The agitated behavior appeared necessary for increased heat production since fever did not occurr in its absence, even when the patient was in restraint. That the stress of restraint in patients taking phenothiazines may lead to hyperthermia via central mechanisms alone has been suggested by experimental study of rabbits treated with apomorphine (10). However, full-body restraint did not produce hyperthermia in these animals, probably because it was also associated with sedation, which precluded excessive heat production. Hyperthermia did occur when these rabbits were put in neck restraints; those restraints were associated with aggressive behavior and therefore also increased heat production.

This case illustrates yet another potentially significant clinical situation in which hyperthermia may occur in association with phenothiazine therapy. Judiciousness in the use of full-sheet restraint in the agitated patient can now be added to avoidance of elevated ambient temperatures and cautious use of anticholinergics in patients receiving acute or chronic phenothizaine therapy. Recognition of this complication will expedite proper management and avoid inappropriate treatment of fever of obscure origin.

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A Single-Blind Study of ECT in Patients with Tardive Dyskinesia

BY GREGORY M. ASNIS, M.D., AND MICHAEL A. LEOPOLD, M.D.

Tardive dyskinesia is a neurologic disease caused by neuroleptic medication. The syndrome usually consists of mild involuntary movements (1, 2) but occasionally becomes medically disabling, causing dysarthria, dyspnea, cyanosis, or dysphagia (3).

Most authors agree that when patients develop tardive dyskinesia, the dose of antipsychotic medication should be lowered or discontinued as the patient's mental condition permits in an attempt to reverse the disease process and to avoid possible complications (4, 5). A problem arises, however, when a patient with tardive dyskinesia becomes acutely psychotic. When one treats such patients with neuroleptic medication he is using the agent that caused the patient's iatrogenic disorder, which may increase the likelihood of an irreversible disease.

The use of ECT in tardive dyskinesia patients who are psychotic is now being considered. However ECT's effects on the dyskinetic process are unclear. One early report suggested that tardive dyskinesia had resulted from ECT for 4 of 33 tardive dyskinesia patients assessed (6), which raises the possibility that

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ECT worsens tardive dyskinesia. However, recent reports of the beneficial effects of ECT on Parkinson's disease (7, 8), another extrapyramidal syndrome, raise the possibility of improving tardive dyskinesia symptoms by treating the psychosis with ECT.

This study was undertaken to evaluate the effects of ECT on the acutely psychotic patient with tardive dyskinesia.

Subjects and Method

Four women with tardive dyskinesia were admitted to an acute hospital ward because of psychotic decompensation. All patients had an oral-facial and limb dyskinesia that was first noted in the previous 6 months. Their prior neuroleptic use ranged from 2 to 8 years, with a mean of 3.5 years; their ages ranged from 43 to 69 years, with a mean of 56 years. All medications were discontinued 3 weeks before commencing ECT. If medication was absolutely necessary during the study, a minor tranquilizer was given.

Patients gave informed consent for the treatment and evaluation procedures. Bilateral ECT was given, preceded by intravenous sodium methohexital and succinylcholine HCl. Treatments were given three times a week. The total number of treatments ranged from 6 to 13.

Each patient's oral-facial dyskinesia was evaluated by the frequency count method of Kazamatsuri and associates (9). Patients were videotaped in a room alone for a 3-minute period. Every movement in the oral-facial region, such as tongue protrusion, lateral movement of the jaw, or lip smacking, was counted, and the number of movements per minute was obtained. Assessments were made at least twice during the baseline period, after every other ECT treatment (on the day of the treatment), and at 1 week and 2 months postECT. Recordings were made at the same time of the day and in the same setting. The movement frequency counts were done blindly from the videotapes by one of us (M.A.L.); tapes were shown in order. A 25% change in either direction was considered to be within the normal day-to-day variation and thus was considered as no change.

Results

As can be seen from table 1, the average oral frequency count of four patients did not change significantly during and after ECT from the initial baseline measurements. Patient 4 was the only one who showed a significant change during ECT (−27%) and after ECT (+50%). This patient had fluctuating pre-ECT baseline recordings varying as much as threefold, whereas the other patients' baseline recordings fluctuated only ± 20%.

At no time during or after ECT did a new area of the body develop a dyskinetic process, and no preexiotomy dyskinetic symptom disappeared.

Initially, all four patients benefited from a course of ECT and were discharged from the hospital. Patient 1 (manic-depressive illness, depressive type) after an initial 2-month follow-up was successfully treated with 1–2 ECTs every month for a total of 6 months. Patient 2 (schizo-affective schizophrenia, depressed type) was treated successfully with minor tranquilizers for an additional 6 months. Patient 3 (organic brain syndrome) and patient 4 (acute paranoid schizophrenia) deteriorated shortly after discharge from the impatient service (approximately 2 months). Both were then treated with neuroleptic medication.

TABLE 1
Mean Oral Frequency Counts

Patient	Before ECT	During ECT	After ECT
1	60.7	52.8	47.7
2	140.1	125.1	114.1
3	81.9	101.6	103.2
4	20.1	14.8	31.7
Mean	75.7	73.6	74.2
Mean excluding case 4	94.2	93.2	88.4

Discussion

The findings reported in this study suggest that ECT had a negligible effect on the dyskinetic process. In one individual patient the dyskinesia significantly worsened during the post-ECT period. This particular patient had as high as a threefold difference in dyskinesia ratings during the baseline period, whereas the other three patients' baseline ratings varied less than 20%. Gardos and Cole (2) recently reported on patients whose dyskinesia fluctuated markedly in a short period of time even when their dosage of medication was unchanged. The authors thought this might cause false positive or false negative results and suggested eliminating such patients from studies on dyskinesia (2). When patient 4 is removed from consideration, the finding that ECT has a negligible effect on dyskinesia appears stronger (table 1). This stands in contrast to recent work demonstrating that ECT has a beneficial effect in Parkinson's disease (7, 8).

A real dilemma exists on how to treat the psychotic patient with tardive dyskinesia. Although tardive dyskinesia is frequently irreversible, recent reports have suggested that stopping the neuroleptic medication in patients whose tardive dyskinesia is in the early stages can reverse the symptoms (7). The importance of making every attempt to reverse tardive dyskinesia is highlighted by a recent report suggesting an increased mortality in tardive dyskinesia patients (10). It is our hope that an alternative antipsychotic therapy can be used in psychotic patients with tardive dyskinesia which would not negatively influence their iatrogenic disease. This report suggests that ECT might be such a treatment for some patients.

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Post-ECT Asterixis Associated with Primary Hyperparathyroidism

BY MAURICE W. DYSKEN, M.D., AND ANGELOS E. HALARIS, M.D., PH.D.

Asterixis is a neurological sign commonly associated with hepatic encephalopathy. First described in 1949 by Adams and Foley (1), asterixis consists of quick, asynchronous, flexion-extension movements of the hands from an initial position of hyperextension at the wrists. These involuntary movements represent momentary lapses in the patient's ability to sustain a fixed posture and are associated with electromyographic evidence of periodic electrical inactivity. Asterixis may also appear in a variety of nonhepatic, metabolic encephalopathies and has been reported in patients with chronic pulmonary insufficiency, uremia, malabsorption syndromes, bromism, and magnesium deficiency (2). In this paper we will report on a patient who developed post-ECT asterixis, a complication that led to the detection of primary hyperparathyroidism.

Case Report

A 48-year-old woman with no previous psychiatric complaints entered the hospital with a 10-day history of manic behavior characterized by pressured speech, flight of ideas, hyperactivity, and preoccupation with religious concerns. She gave a positive family history for affective illness: both her father and brother had committed suicide during depressive episodes. Physical examination including neck palpation was entirely normal. Serum calcium was 10.8 mg/dl, which is at the upper limit of normal (8.8–11.0 mg/dl). Protein electrophoresis was entirely normal. She was diagnosed as manic-depressive, manic type, and started on 300 mg of lithium carbonate t.i.d. Because of persistent nausea and vomiting, lithium carbonate was discontinued at the end of 10 days and the patient was treated with 20 mg of trifluoperazine daily. Her elevated mood returned to normal over the next 3 weeks and she was discharged on 50 mg of fluphenazine decanoate every 2 weeks.

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The patient functioned well at home for approximately 10 days and then began to experience depression associated with anorexia, insomnia, fatigue, and suicidal thoughts. She was rehospitalized 2 weeks after discharge with a diagnosis of manic-depressive illness, circular type, and was given lithium carbonate, 300 mg t.i.d. She tolerated the drug this time because 250 mg of trimethobenzamide (Tigan) was given 30 minutes before lithium was administered. A steady-state plasma level of 1.04 mEq/liter was obtained on 1,200 mg/day of lithium carbonate. The trimethobenzamide was discontinued after 1 week without recurrence of nausea or vomiting. Because the patient's depression was quite severe and was characterized by marked weight loss and psychomotor retardation, ECT was started on the 10th day of lithium carbonate treatment. ECT was administered three times a week (Monday, Wednesday, and Friday) while she was maintained on lithium carbonate. A total of 10 ECTs was given: the first was bilateral and the subsequent ones were all unilateral over the nondominant hemisphere. Pretreatment medications included intravenous atropine, 0.4 mg; thiopental, 75 mg; and succinylcholine, 40 mg; as well as nasal oxygen. After the third ECT the patient showed increased interest in her appearance and in ward activities. Because of increasing agitation she was started on thioridazine, 10 mg t.i.d. three times a day and 50 mg at bedtime, and experienced relief of tension and less frequent periods of wakefulness during the night. Her depression continued to improve with each ECT. Immediately upon regaining consciousness after the 9th and 10th ECTs, she developed bilateral upper limb asterixis that lasted for approximately 2 hours.

Since the development of asterixis suggested a metabolic encephalopathy, a search for the underlying cause was initiated during the period of asterixis after the 10th ECT. Arterial blood gases and pH were normal. Assisted ventilation during the 10th ECT had appeared adequate. The patient had no history of liver disease, and liver enzymes and blood ammonia level were normal. Serum glucose, BUN, and magnesium were all within normal limits. The EEG showed generalized slowing, a pattern consistent with the presence of a metabolic encephalopathy. Skull films and a brain scan were normal. The plasma calcium, however, was above normal at 12.0 mg/dl with normal albumin, phosphorus, and alkaline phosphatase levels. Parathyroid hormone (PTH) level, determined from a sample of blood drawn 2 weeks after the

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10th ECT, was elevated at 44 μEq/ml and was associated with elevated calcium level of 11.5 mg/dl. A presumptive diagnosis of primary hyperparathyroidism was made. When we examined her chart for past calcium and phosphorus determinations, we discovered that 3 years before admission she had had a calcium level of 9.7 mg/dl and a low phosphorus level (2.4 mg/dl). Two years before admission her calcium level was 10.6 mg/dl and phosphorus was 2.6 mg/dl, and 1 year before admission the calcium was 11.3 mg/dl and phosphorus was 3.1 mg/dl. Because the patient had been medically asymptomatic, neck exploration for a parathyroid adenoma did not seem warranted at this time, and arrangements were made for the patient to be followed in the endocrine clinic after her discharge from the hospital.

Upon completion of ECT, the patient was started on desipramine, which was increased to 150 mg/day. She was discharged much improved on desipramine, thioridazine, and lithium carbonate. Thioridazine was discontinued 1 month later and desipramine was stopped 5 months later. The patient has continued on lithium carbonate for the past 18 months and has not experienced depressive or manic episodes. A repeat PTH level 1 month after discharge was 35 μEq/ml, with a calcium level of 10.5 mg/dl. These values remain compatible with a diagnosis of primary hyperparathyroidism (3).

Discussion

Since asterixis began immediately after the 7th and 10th ECTs, we conclude that ECT was necessary for the development of asterixis, but we doubt that ECT alone was sufficient to produce it. Other variables that may have interacted to produce the asterixis include the hyperparathyroidism and associated elevation in calcium, psychotropic medications, pre-ECT drugs, properties of the ECT stimulus, and post-ECT ventilatory assistance. However, the absence of any report in the literature describing post-ECT asterixis suggests that the hyperparathyroidism may have been the significant factor in the development of the asterixis.

Administration of lithium carbonate to animals and humans has been associated in some studies with no change in serum calcium (4, 5) and in others with an increase in serum calcium (6, 7). Mellerup and associates (6) examined lithium effects in thyroparathyroidectomized rats and concluded that the calcium elevation was independent of PTH secretion. It appears unlikely that lithium produced our patient's hyperparathyroidism. Calcium and phosphorus levels before lithium treatment suggest that hyperparathyroidism developed over the 3 years preceding hospitalization.

Recent studies have demonstrated a fall in urinary and serum calcium after ECT (8, 9). Carmen and associates (9) described decreases in both CSF and serum calcium after 3–5 treatments. They reported a single case in which the serum calcium decreased at 5 and 20 minutes post-ECT but returned to the pre-ECT level by 90 minutes. In another patient a modest increase in plasma PTH was measured 5 minutes after ECT, but the increased value was still within the normal range. The authors suggest that the increase in PTH is a secondary effect that results from the decrease in serum calcium. We wish to point out that in our patient both PTH and serum calcium were abnormally high, which suggests that the PTH increase was a primary rather than a secondary change. Follow-up calcium level determinations over the past 18 months have remained above normal, and phosphorus levels have continued to be below normal. These persistently abnormal laboratory values support a diagnosis of primary hyperparathyroidism.

The development of post-ECT asterixis focused attention on the possibility that an underlying metabolic disorder was present. To our knowledge, this is the first report not only of asterixis following ECT but also of an association between primary hyperparathyroidism and post-ECT asterixis.

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Pyroluria: A Poor Marker in Chronic Schizophrenia

BY RICHARD CRUZ AND WOLFGANG H. VOGEL, PH.D.

Carl Pfeiffer and associates (1) claim that 30% of schizophrenics have elevated urinary pyrrole excretion indicative of higher pyrrole levels in the body. This endogenous pyrrole can combine with the aldehyde group of pyridoxal phosphate to form a Schiff's base, which complexes with zinc. The result of these reactions is a syndrome of B_6 and zinc deficiency (1). B_6 is a cofactor in the synthesis of various neurotransmitters such as serotonin, norepinephrine, dopamine, and GABA, and deficiencies in these neurotransmitters could contribute to psychoses and neurologic problems. We have tested morning urine samples from nine chronic schizophrenic patients for urinary pyrroles (the "mauve spot") by thin layer chromatography (TLC) and have evaluated these patients for the clinical picture described by Pfeiffer and associates as characteristic of pyridoxine and zinc deficiency.

Method

The urinary pyrrole analysis used was Irvine's procedure as modified by Sohler and associates (2–4) to eliminate false positives due to the presence of phenothiazine metabolites.

Internal and external standards and blanks ensured accuracy in the determination of pyrrole. TLC spots were judged positive or negative by visual comparison with internal standards. The urinary pyrrole determinations were carried out on 10 healthy controls and on a group of 9 chronic schizophrenic inpatients at Jefferson Hospital. The controls (9 men and 1 woman) had an age range of 20–47 years, with a mean of 27. The schizophrenic patients (7 men and 2 women) ranged in age from 23 to 57, with a mean of 31. All patients were taking either phenothiazines or haloperidol. The diagnosis of schizophrenia was based on a history of a first psychotic break before age 35 and at least two hospitalizations in two different years, plus at least two of the following symptoms: auditory hallucinations, delusions, flat or inappropriate affect, and looseness of associations. We looked for the following symptoms and signs, described in detail by Pfeiffer and associates (1) as characteristic of B_6 and zinc deficiency: schizo-affective type schizophrenia, history of major stress preceding the psychotic break, inability to remember dreams, sallow complexion, photosensitivity, dermatitis, cutaneous stretch marks, unexplained fever and chills, morning nausea, chronic constipation, upper left quadrant pain, barbiturate intolerance, amenorrhea, impotence, paresthesias, tremor, shaking, muscle spasms, convulsions, malformation of the knee, white spots on nails, hypoglycemia and B_6-responsive anemia.

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The authors would like to thank Alan Sohler, Ph.D., Roy Clouse, M.D., and Robert Stolberg-Acosta, M.D., for their assistance.

Results

Table 1 shows a comparison of pyrrole test results and the Pfeiffer and associates clinical criteria for zinc and B_6 deficiency. Only 2 of the patients had positive tests for urinary pyrrole excretion. One patient could not remember dreams and the other patient had a history of constipation and amnesia. However 5 of 7 patients with negative urine tests had 3 or more of Pfeiffer's symptoms of B_6 and zinc deficiency. One female patient with a negative urine test had many of the symptoms described by Pfeiffer: her psychotic break was preceded by the stress of her father's death, she is schizo-affective and says her knees became "disjointed" during childhood, and she has sallow complexion, dermatitis, amenorrhea, and peripheral neuropathy. The patient's blood level of B_6 was normal (67 ng/ml).

Only 2 of the 10 controls were positive for pyrroles when tested again, they were found to be negative.

Discussion

It is our feeling that pyroluria is a poor marker in chronic schizophrenia for Pfeiffer's symptoms of B_6 and zinc deficiency. The comparable prevalence of pyroluria in our patient and control groups suggests that pyroluria is unrelated to chronic schizophrenia. Furthermore, the finding of both positive and negative test results in the same individual (2 control subjects) might suggest a dietary origin for pyroluria.

It is possible that Pfeiffer's observations were made on patients who had acute schizophrenic symptoms but whose overall syndrome is a disease of heme biosynthesis similar to porphyria. Pfeiffer and associates have found that patients with "pyroluric schizophrenia" have increased urinary coproporphyrin excretion (1) as do patients with porphyria (5). Patients with acute intermittent porphyria and patients with porphyria variegata may have psychotic symptoms, abdominal colic, and neurologic symptoms (5). Patients with porphyria variegata may also have symptoms of photosensitive dermatitis and barbiturate intolerance (5). Hamefelt and Wetterberg (6) found decreased blood concentrations of pyridoxal phosphate in patients with acute intermittent porphyria (AIP). No clinical improvement was found when two of these B_6-deficient patients with AIP were treated with low doses (100 mg/day) of pyridoxine. Pfeiffer and associates prescribe

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TABLE 1
TLC Results, B_6 Deficiency Symptoms, Medications, and Demographic Data*

Item	Patient 1	2	3	4	5	6	7	8	9
TLC test for pyroluria	+	–	–	–	–	–	–	+	–
Selected symptoms of pyroluria and B_6 and zinc deficiency									
Sallow complexion	–	–	–	+	–	–	–	–	–
Striae	–	–	–	–	–	–	+	–	–
Dermatitis	–	–	–	+	–	–	–	–	–
Photosensitivity	–	–	–	+	+	–	–	–	–
Unexplained fever and chills	–	–	–	–	+	–	–	–	–
Constipation	+	–	–	–	–	–	–	–	–
Irregular menstrual period or amenorrhea	–	n.a.	n.a.	+	n.a.	n.a.	n.a.	n.a.	n.a.
Amnesia	+	–	–	–	+	+	–	–	–
Tremor, shaking	–	–	–	–	–	+	+	–	–
Muscle spasms	–	+	–	–	–	–	–	–	–
Paresthesias	–	–	–	+	–	–	–	–	–
Convulsions, seizures	–	–	–	–	–	+	–	–	–
Malformation of knee cartilage	–	–	–	+	–	–	–	–	–
White spots on nails	–	+	+	–	–	–	–	–	–
Major stress preceding breakdown	–	–	–	+	–	+	–	?	–
Failure to dream or remember dreams	–	–	+	–	+	–	+	+	–
Schizo-affective	–	+	–	+	–	–	–	–	–
Medications	P	P	P	H	H	P	P	H	P
Demographic Data									
Age (years)	57	37	26	26	23	28	33	29	23
Sex	F	M	M	F	M	M	M	M	M
Race	W	W	W	W	B	B	B	W	B

*n.a.=not applicable, P=phenothiazine, H=haloperidol.

megadose of pyridoxine (2 g/day) for patients with pyroluria. There have been no reports on the clinical effects of such larger doses of B_6 on patients with a diagnosis of acute intermittent porphyria.

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"Experimental" Self-Mutilation

BY STEVEN L. DUBOVSKY, M.D.

Patients who habitually injure themselves without suicidal intent are often encountered on medical and psychiatric services. Wrist cutting is the most frequent type of self-mutilation (1, 2), but abrasion, burning, head banging, ingestion of medications and various objects, jumping from heights, hair pulling, insertion of foreign bodies into the urethra, self-enucleation, self-castration, and removal of the tongue have been reported occasionally (1, 3–5). The following case report represents a type of self-mutilation that as far as I

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know has not been reported previously and that provides some additional insights into this disturbing symptom.

Case Report

The patient was a 22-year-old man with congenital sensory neuropathy and an inability to appreciate pain, temperature, proprioceptive stimuli, and other surface sensations because he lacked peripheral sensory nerves (6). He did not know when he had hurt himself, so his injuries often went untreated, and he developed numerous chronic infections of his extremities that could be controlled only by amputation. The patient's care was further complicated because he ignored wounds or interfered with their healing by picking at them or refusing to change dressings. Angry confrontations by the surgical staff resulted in a worsening of this behavior. He eventually explained to the psychiatric consultant that if he tried to heal his wounds and failed, he would experience overwhelming disappointment and anger. He had therefore decided to make his wounds worse so that the outcome would at least be predictable.

By the time the patient was 21, three fingers on his left hand, two fingers on his right hand, and his right leg had been amputated, and he had developed chronic osteomyelitis in his left leg, which he knew would eventually require amputation. At this point, he began to cut his wrists and forearms systematically, always in different locations. He initially said that he had been depressed about the possibility of losing his remaining leg and thought that life would not be worth living without it. On closer questioning, however, he described an entirely different motive. He said that he had noticed that healing of his extremities was worse distally, and he wondered whether he would experience difficulty healing in his thighs after his left leg was amputated too, and in his arms after he lost his hands. He had decided to experiment on his arms in order to examine this question systematically. Because he thought that this would sound like a "crazy" reason to cut himself, he had decided to give a more understandable explanation, i.e., that he was suicidal. After he had satisfied himself that his wrist wounds healed quickly, this self-mutilation ceased.

A year after his left leg was amputated, the patient developed chronic osteomyelitis of his left index finger. Antibiotics did not seem to help and he asked his surgeon to amputate the finger. He was angered by the constant reminders of his body's inability to heal and felt that the finger was "only in the way." The surgeon did not feel that the patient was in a position to formulate an accurate diagnosis and treatment plan and refused to amputate. Three days later, the patient cut a hole in a shoe box and put the healthy finger inside the box. He stuck the infected finger through the hole and calmly sawed it off with a hack saw. He saved the finger in a plastic bag in the freezer for later "study."

Discussion

Unlike borderline or chronic schizophrenic patients who cut themselves to relieve feelings of depersonalization (1), lessen inner tension (7), solve genital conflicts (8), or to be reassured that they are alive by seeing or feeling their own blood, (1, 9), the patient's acts had specific "experimental" and "surgical" purposes. This unusual attitude seems to have reflected a wish to deny his inability to control his body by playing an active role in its destruction. He could then feel that he was losing body parts because it was his wish. He also acted as though only a part of himself (e.g., his finger) was incapable of healing itself and removed the helpless part by amputation.

Few psychiatrists can expect to encounter such unusual symptoms in a patient with such a severe medical disorder. However, the wish of patients who feel helpless or victimized to assert control over a part of themselves may play a role in many forms of self-mutilation.

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Am J Psychiatry 135:10, October 1978

The Catatonic Dilemma

BY IRA BRENNER, M.D., AND WILLIAM J. RHEUBAN, M.D.

High-potency neuroleptic treatment of schizophrenia can be complicated by the onset of putative extrapyramidal signs, which can be indistinguishable from catatonia (1–7). Clinicians are warned to stop medication for noncatatonic patients who become catatonic during treatment (1) because such patients are prone to serious complications, e.g., pulmonary emboli and pneumonia (7). The following case details our dilemma in treating catatonic schizophrenia and superimposed neuroleptic catatonia. To our knowledge, this combination, although it has been assumed to occur, has not been reported previously.

Case Report

A 19-year-old college student, previously in excellent health but with a family history of schizophrenia, developed increasing inability to concentrate, anxiety, anorexia, and indecisiveness several weeks before his admission. He became preoccupied with his sexual identity, and tension developed between him and his roommates regarding whether or not he should move out. He decompensated to the point that food had to be placed in front of him. He was referred to the emergency room, where he exhibited motor ambivalence, agitation, and loosened associations. He refused to be admitted and was given haloperidol, 5 mg p.o. The next day he agreed to admission; he was almost mute, with fixed staring, posturing, ideas of reference, thought rushing, and auditory hallucinations. Physical examination, CBC, SMA-12, SMA-6, EEG, CT scan, and a neurology consultation ruled out organicity. Haloperidol was increased to 30 mg/day, resulting in an acute dystonic reaction. Benztropine, 2 mg I.M., was effective in treating the reaction and 4 mg/day p.o. was added. His stupor worsened, and he demonstrated waxy flexibility, automatic obedience, rigidity, reaction at the last moment, abnormal compliance, and forced grasping. Haloperidol was increased to 80 mg/day on day 4 and amantadine, 200 mg/day, replaced benztropine. The patient became progressively more autistic and developed a "paralysis" of the left arm. He believed that moving his left side made him a woman and moving his right side made him a homosexual. Male aides made him agitated, and we wondered whether he might erupt into catatonic excitement. Amantadine was discontinued on day 7 and the patient regressed further. Urinary incontinence and food refusal coincided with family visits. The patient appeared to have difficulty with secretions and the gag reflex was decreased, although his lungs remained clear. Haloperidol was discontinued and IVs were started. By day 10, there was gradual improvement in motor activity and the patient began to eat, but his difficulty with secretions continued. His WBC increased to 16,000 and a chest X ray showed bilateral lower lobe infiltrates. Pulmonary consultants confirmed the diagnosis of aspiration pneumonia and felt that the psychiatric service had missed an underlying systemic illness that warranted further evaluation. The patient was then transferred to the medical ward for treatment.

The patient's rigidity, mutism, and posturing continued to improve gradually without medication. Penicillin, 10^7 U/day in continuous infusion, was started, and the infiltrates resolved uneventfully. He was transferred back to the psychiatric service 6 days later, at which time there was marked improvement in his rigidity and mutism. Hallucinations and thought rushing disappeared, but loosened associations, perplexity, denial of illness, and "paralysis" persisted. He also remained delusional about the consequences of moving his body. Thioridazine was instituted in a dosage of 75 mg/day, which was slowly increased to 500 mg/day over 3 weeks. At the time of discharge, his thought disturbance was improved and his motor involvement resolved, except for weakness of the left arm.

Discussion

The patient's conflict over movement demonstrated analogic action, which is seen in disorders of volition (8). Moving out of his apartment meant failure, which he equated with being a failure as a man and becoming a woman or a homosexual. He felt he would have to return home to his schizophrenic mother and brother, submitting himself to a lifetime of psychosis. Consequently, his immobility served the dual magical purposes of preventing his move and preserving his male image. The persistent weakness of his arm seemed to contain his psychosis, encapsulating his struggle over moving. Such volitional conflicts are characteristic of catatonic schizophrenia (8) and would not be seen in a pure neuroleptic-induced stupor.

As the patient's clinical course progressively deteriorated, the staff began to experience his dilemma, feeling helpless and indecisive. Distinguishing between the increasing drug side effects and the psychosis was very difficult. Did he have iatrogenic catatonia, or was his underlying schizophrenia refractory to treatment? Then, when he developed aspiration pneumonia, the staff experienced guilt followed by indignation and loss of esteem when the consultants implied we had failed and "must have missed the underlying illness." Although he improved, the patient remained psychotic and regressed when he returned to our ward. Consequently, the staff's feelings about "missing the diagnosis" were relieved, and the iatrogenic overlay became clear.

Catatonia accounts for more than 5% of first diagnoses of schizophrenia (9) and the incidence of neuroleptic-induced catatonia is estimated by some clinicians to be less than .5% (personal communication with Salman Akhtar, M.D., David Rosenman, M.D.,

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and William J. Rheuban, M.D.), so the probability of encountering this combination is quite small. Even though catatonic features do not appear to predispose patients to extrapyramidal effects such as akinesia and rigidity (10), high-potency drugs should be used cautiously in catatonics until methods are developed to predict those especially at risk. One possibility is assay of pretreatment urinary dopamine excretion, which is negatively correlated with the probability of developing such toxicity (10). If neuroleptic catatonia is indeed a severe extrapyramidal reaction, such measurements could identify the low dopamine excreters, who might comprise the high-risk group.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Safe Use of Haloperidol in a Patient with Cardiac Dysrhythmia

SIR: Often it is necessary to treat with antipsychotic medication patients who have cardiovascular disease. The hypotensive side effects of phenothiazines and thioxanthenes, a well-documented problem (1, 2), usually compel one to turn to another class of agents. Butyrophenones, primarily haloperidol, are often recommended because of their allegedly low rate of cardiovascular effects, both on blood pressure and the cardiac conduction system (2–4). However, evidence of haloperidol's safety is anecdotal and not well documented. It seems helpful, therefore, to report an example of the safe use of haloperidol to treat an acute psychosis in a 14-year-old boy with a severe genetic disorder of cardiac rhythm.

The patient was 13 years old at the time of his first cardiac arrest. He was admitted to the hospital 1 year later, after his third cardiac arrest, during which he had developed ventricular fibrillation, required electroconversion five times, and had also vomited and aspirated, which led to a severe pneumonia. He had had an older brother who died from a cardiac dysrhythmia and two younger brothers who were also suspected of having cardiac conduction abnormalities; there were no female siblings. He was diagnosed as having Romano-Ward syndrome (prolonged Q-T interval).

During his hospitalization he had multiple medical problems, including several episodes of cardiac dysrhythmia, sepsis, aspiration pneumonia, hypoxia, gastrointestinal bleeding, persistent hypokalemia, transient liver enzyme elevation, a positive direct Coombs' test, and agitated psychosis (presumably secondary to one or more of his other problems or medications) with apparent hallucinations. During his hospitalization he was treated at various times with cephalothin, digoxin, amikacin, chloramphenicol, intravenous lidocaine, propranolol, tocainide, cimetidine, carbenicillin, potassium supplement, amino-phylline, multi-vitamins, folic acid, antacids, and ferrous sulfate. He also received blood transfusions. His cardiac dysrhythmias included recurrent ventricular premature beats, ventricular tachycardia, supraventricular paroxysmal tachycardia, ventricular fibrillation, and multifocal bigeminy. He required a cardiac pacemaker.

During the second week of hospitalization the patient developed an agitated, delirious psychosis with apparent hallucinations. He was originally treated with a combination of a barbiturate, a narcotic, and a benzodiazepine; these provided sedation. However, when he was awake it was clear that the psychosis did not respond to this regimen. Therefore, haloperidol was administered in a previously described regimen (5), except that in this patient it was given orally through a nasogastric tube. Maintenance medication was 10 mg b.i.d. with further 5-mg doses as needed. The average daily dose was approximately 30 mg. The agitation, disorientation, and hallucinations resolved in about 3 days. Within 2 weeks haloperidol was stopped. By this time the patient's medical status had markedly improved. There was no return of psychotic or delirious symptoms throughout the remaining hospitalization or thereafter for at least 6 months (the time of last follow-up).

With the exception of a mild tremor that resolved with improvement of the medical problems, the patient had no untoward reactions to haloperidol. Specifically, there was no deterioration of his cardiac status. The successful use of haloperidol in this patient with a very severe cardiac rhythm disturbance supports previous statements that haloperidol is free of cardiovascular side effects and is the antipsychotic drug of choice in patients with cardiac rhythm disorders.

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Wedded Bliss Between Professionals?

SIR: I read with dismay the article "The Successful Professional Woman: On Being Married to One" (October 1977 issue) by Theodore Nadelson, M.D., and Leon Eisenberg, M.D., in which two male psychiatrists purport to describe the joys they have found in being married to women who are also psychiatrists. They conclude, "We consider ourselves the most fortunate of men." However, the tone of the paper is so cloying and effusive that only the most naïve (or psychologically insensitive) reader would take the message at face value.

What comes across to me is a hymn of praise not to their wives and their marriages, but to themselves for being such

great guys about putting up with the inconveniences and downright foolishness inherent in their wives carrying on as though they truly are their husbands' professional peers.

The most telling vignette is the one in which L describes, with obvious delight, the way he handled himself while attending a professional meeting in the role of spouse. His idea of social grace was to upstage his wife by delivering a life-of-the-party after-dinner speech in which the big joke was the absurdity of his being cast in the role of mere faculty spouse.

My husband is a radiologist. Over the years he's been my greatest source of support. At times we've had our tensions over who's pulling, or not pulling, his/her share of the spouse, parent, and professional loads. But never have I sensed in him the kind of competitiveness that I sense in the Nadelson and Eisenberg marriages. My husband has never felt it necessary to be the star at my professional gatherings. Nor has he sought to increase his list of professional presentations or publications by writing about how capably he has dealt with me over the years.

You show-cased the article as though you saw it as a paradigm for wedded bliss between professionals. I propose you would have done better to offer it under the classic women's magazine feature: "Can These Marriages be Saved?"

JEAN L. ROBERTS, M.D.
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Trimipramine and Relapses of Symptomatic Drinking

SIR: I have seen puzzling relapses of heavy drinking in patients given trimipramine who correspond to the diagnostic category proposed by Hagop S. Akiskal, M.D., and associates, "intermittent bipolar affective disorder" ("Cyclothymic Disorder: Validating Criteria for Inclusion in the Bipolar Affective Group," November 1977 issue).

The first, a 52-year-old man, was treated on an outpatient basis for social phobia with a variety of antidepressants, neuroleptics, and combinations of the two classes of drugs. For the past 8 years he had fared reasonably well, with only a few drinking bouts. Because of his history of chronic incapacitation and the clinical picture of agitated depression, trimipramine was started in a single oral dose of 100 mg at bedtime. He reported considerable gain in subjective well-being and demonstrated self-confidence that deteriorated into occasional paranoid vindictiveness within a few weeks. Almost simultaneously there were signs that he had resumed his secret drinking. Within about 6 weeks of the introduction of trimipramine, which was then at 150 mg/day, he was in an acute detoxification center because of legitimate fears for his own and the household's safety.

The second patient, an unemployed 35-year-old artist with a history of heavy periodic drinking, had been treated on an outpatient basis for almost 8 years with minor and major tranquilizers without too much success. Because of his underlying depression and definite periodicity, trimipramine, 75–100 mg/day, and lithium carbonate, 900 mg/day, were started. He reported a subjective increase in well-being and improved sleep within a few weeks, but within a month he slipped into an unusually heavy and prolonged episode of drinking that ended with hospitalization for a predelirious condition.

The third example was a 52-year-old laboratory technician who had been sober for almost 11 years. He had been treated for 6 years during which time he had at least two bouts of reckless spending and traveling that were controlled with phenothiazines. His presenting complaints were primarily neurotic, but he also showed a definite impulsive trait with ready vindictiveness and some paranoid attitudes. Upon recognition of the periodic pattern of the seemingly (hypo)manic episodes superimposed on chronic "anxiety-depression," trimipramine, 150 mg/day, was prescribed, and the patient reported that he felt better within a few weeks. However, he has since slipped back into fairly heavy drinking.

All of these men had a long-standing affective disturbance with discernible periodicity, compounded by dipsomania. Introduction of trimipramine to treat the anxious depression uniformly produced initial subjective improvement only to result in a return to heavy drinking within about 4 weeks.

Is this merely a series of coincidences? Do the bouts of drinking represent (atypical) manic episodes that could be triggered by any tricyclic antidepressant? Could we suspect a similar idiosyncratic reaction to trimipramine in all recidivist alcoholics? Or is it that the combination of intermittent bipolar disorder with a history of alcohol abuse provides an unfortunate metabolic background for the specific dibenzazepine drug?

ANDRZEJ KUBACKI, M.D.
Bathurst, N.B., Canada

Dr. Akiskal Replies

SIR: Dr. Kubacki's clinical vignettes are not presented with sufficient detail to enable one to decide whether they exemplify what my associates and I describe as "cyclothymic disorder." Dipsomania is a known clinical indication of bipolar disorder, occurring more commonly during the hypomanic or manic phase. Nevertheless, it is difficult to determine whether these relapses of drinking behavior represent a hypomanic "equivalent" in the absence of other clinical features suggestive of a bipolar disorder. From the available information, it would be more parsimonious to suggest that the patients reported were simply experiencing a return of pretreatment behavior.

Pharmacologically induced hypomania has been reported with the tricyclic drugs, monoamine oxidase inhibitors, L-dopa, sympathomimetic drugs, ECT, and sleep deprivation (1–3). The available evidence suggests that it is generally limited to bipolar depressives. The occurrence of this phenomenon with drugs or procedures that suppress REM sleep or mobilize limbic system catecholamines suggests that REM suppression or neurotransmitters involved in this process are integral to the switch process from depression to mania. However, drugs with relatively selective action on the serotonergic system (e.g., 5-hydroxytryptophan plus peripheral decarboxylase inhibitor or tryptophan plus an MAO inhibitor) have also been reported to induce hypomanic behavior (4). I am not aware of reports of trimipramine-related hypomania. However, since it suppresses REM sleep, like other tricyclic antidepressants, it would be logical to expect a pharmacologically induced shift from depression to hypomania upon trimipramine administration.

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HAGOP S. AKISKAL, M.D.
Memphis, Tenn.

Speech Blockage: Therapeutic Side Effect?

SIR: In their article "Speech Blockage: A Tricyclic Side Effect" (May 1978 issue) Alan Schatzberg, M.D., and associates stated that "to our knowledge this tricyclic side effect has not been described in the literature." This effect was described in my paper titled "Anxiety Revisited" (*American Journal of Psychoanalysis*, volume 37, pp. 299–308). On page 306 of the article I stated, "Some of my patients taking antidepressive drugs (tricyclics) have described forgetfulness and difficulty in word or idea finding; this may help break the circular associative circuit both in obsessive-compulsive symptoms and in depressions."

I did not emphasize this as a side effect but more as a therapeutic effect. I would assume that a therapeutic effect can blend into a side effect depending on the sensitivity of the particular patient to the effect, the quantity of tricyclic, the specific tricyclic (in any patient the response and side effects differ with different tricyclics), and other drugs taken simultaneously. Other variables that can cause episodes of time-related blocking are whether the tricyclic is taken on an empty stomach or as a single bolus. I have always been fascinated by so-called side effects that in a different context are called therapeutic effects (for a discussion, see reference 1).

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JACK SCHNEE, M.D.
Briarwood, N.Y.

Dr. Schatzberg Replies

SIR: We read Dr. Schnee's letter with great interest. It has been our feeling that tricyclic antidepressants may have significant effects on various cognitive functions including learning and memory (1). The symptoms described by Dr. Schnee appear similar to those described in our communication. We must point out, however, that many of these patients experienced the effect as a hesitance in speaking rather than an inability to think of a particular word. This phenomenon may be different from certain forms of anomia or aphasia, but it is conceivable that the various effects noted are all related under one umbrella. An important difference between our report and Dr. Schnee's is that our patients who experienced speech blockage or some memory disturbance were uncomfortable and complained that the symptoms interfered with their functioning.

It is difficult to cull from the literature all reports of drug side effects. However, to our knowledge Dr. Schnee's paper appeared while ours was in press. We also received an interesting letter from Dr. Francis Kane, who apparently described this phenomenon some 14 years ago (2), certainly before our paper was in press! Dr. Kane's report raises another dimension to this phenomenon. Cited are references to a syndrome of stammering, jerky speech, and involuntary jerks of the head and extremities (3, 4). These have occurred with imipramine-phenothiazine combination and with imipramine overdose. This phenomenon is similar to the myoclonic seizures on high-dose chlorpromazine-imipramine combination, described by Kramer and associates in 1962 (5). This phenomenon may be quite different from our patients', who did not show such myoclonus. Again, however, an argument could be made that the two phenomena are related in some way. At any rate, it is important for clinicians to be aware of less frequent untoward reactions to the tricyclics, and we were pleased to read the letters.

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ALAN F. SCHATZBERG, M.D.
Belmont, Mass.

An Educational Approach to Short-Term Hospitalization

SIR: In "Brief Versus Standard Hospitalization: The Differential Costs" (June 1978 issue). Jean Endicott, Ph.D., and associates presented a good case for brief hospitalization. This concept has been successfully used at the Mandala Center, a private psychiatric hospital in Winston-Salem, N.C., where the average length of stay is 18 days. The focus on short-term treatment uses a novel approach to mental illness.

The patients attend mandatory classes six times a week for 1 hour. Classes, which are both didactic and experiential, cover the following topics: "Thinking Yourself Out of Depression Using the ABCs of Rational Emotive Therapy" (RET), "Assertive Training" (AT), "Transactional Analysis" (TA), "Coping with Stress," and "Relaxation Training."

These five areas are taught each week, so therapists and patients talk the same language. The hospital bookstore sells self-help books dealing with each of these topics, and patients are encouraged to read them as part of their daily homework assignments. Families are encouraged to read the same books. Therapy sessions focus on the patients' problems along with the self-help techniques that they have learned. Because the average length of stay is only 18 days, the treatment is intense, with a focus on increased self-help skills to use outside the hospital setting. An example of this treatment approach follows.

A 37-year-old woman came to the hospital with extreme depression resulting from a poor marital relationship and boredom with her life. In addition to antidepressants, the treatment program called for daily education classes, activi-

ty therapy, individual sessions with the therapist, and group sessions with her psychiatrist. During individual and group sessions, the focus of therapy was on her negative cognitions leading to depression (RET) and her helpless "child" ego state (TA). Once a degree of insight was obtained, the action stage of therapy involved assertive techniques (AT) and relaxation exercises to help her cope with her anxiety. Her base of knowledge was the education classes, which the therapist and psychiatrist channeled to her personal situation. The patient was not only treated by mental health professionals but was also able to talk with some of the other patients about her problems because they all spoke the same "language."

The use of education groups can be a valuable adjunct to therapy; patients can learn basic coping strategies that are then made specific to their own personal problems. Follow-up with patients supports the assumption that this is one of the most helpful aspects of the brief hospitalization treatment program.

RICHARD BLUE, PH.D.
Winston-Salem, N.C.

CHAMPUS Payment Limitations

SIR: I would like to respond to the Letter to the Editor on "CHAMPUS Coverage" (April 1978 issue) by Jonathan G. Solomon, M.D. Dr. Solomon objects to CHAMPUS limitations of payment for five visits per week. It is hard to believe that this is not more than adequate reimbursement.

Analyzing the letter briefly, Dr. Solomon's emotionalism is evident in such words as "arbitrary," "unwarranted," "galling," and "intrusion." He states that his hospital (Bayberry Psychiatric Hospital) typically has provided 7–11 hours of therapy per week; undoubtedly the hospital has been reimbursed for this. It is hard to see justification for that kind of treatment and one wonders if anyone can afford to be ill. The only way one could pay for such therapy is through third-party payments, i.e., CHAMPUS, which Dr. Solomon is so vehemently berating.

It would seem he wants it both ways, and has been used to getting it so in the past. I can think of better places for my tax dollar than the situation Dr. Solomon describes at Bayberry Hospital.

E. WILLIAM HAUSER, M.D.
Newport Beach, Calif.

Dr. Solomon Replies

SIR: I plead guilty to the charge of emotionalism on the important issue of the intrusion of third-party carriers into the doctor–patient relationship. The thrust of my previous letter was to protest the arbitrary curtailment of insurance coverage by CHAMPUS for psychiatric patients. I strongly feel that psychiatric patients are entitled to medical care on the same basis as other medical patients and should not be discriminated against. Further, depriving psychiatric patients of mental health care is an unproductive attempt to effect cost-curtailment because they then overutilize medical facilities (1–2).

It is unclear what Dr. Hauser means by "that kind of treatment" but he seems to object to spending 7–11 hours a week with patients and feels that 5 visits a week should suffice in treating hospitalized patients. It seems peculiar to restrict the therapy portion of a 168-hour week to an arbitrary maximum of 5 hours. There is always the chance that CHAMPUS will discover that it is cheaper to establish a minimum of 4 hours weekly, and then perhaps none at all. The determination of the number of reimbursable therapy hours per week seems an unwarranted intrusion by third-party carriers who are not licensed to practice medicine. Their business is insurance; their primary interest to curtail costs. The clinician's primary goal is to provide quality health care. A conflict of interest may ensue.

There are safeguards in existence to prevent abuse of services. For example, at Bayberry Psychiatric Hospital there are overviews by an active utilization review committee, the Colonial Virginia Foundation for Medical Care, audits by the Joint Commission on Accreditation of Hospitals, Medicare and others, and chart reviews by insurance companies who can and do request photocopies of entire charts. Thus, only those patients who require intensive treatment are hospitalized.

In a previous communication (3) I expressed the view that a wide variety of community-based alternatives to hospitalization should be available. Unfortunately, CHAMPUS recently curtailed the benefits of partial hospitalization from 5 days a week to 2. So much for "The Paradoxical Underutilization of Partial Hospitalization" (4), a modality of therapy that is effective and less costly than residential treatment but which is undermined by the counterproductive policy of the carrier.

I recognize the importance of third-party carriers but protest a unilateral determination of medical practice. I feel the health care providers should be consulted with rather than informed of policy directives. We are not yet salaried employees of the government and may have contributions to make in establishing policies. Accountability should be a two-way street and is best served by an ongoing dialogue between the carriers and the health care providers, whose goal is to establish policies that maximize the quality and availability of care while restraining escalating costs. The practice of medicine is sufficiently challenging and arduous without an adversary relationship between the medical profession and the insurance industry.

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JONATHAN G. SOLOMON, M.D.
Hampton, Va.

Persistent Lithium-induced Nephrogenic Diabetes Insipidus

SIR: Nephrogenic diabetes insipidus is an unusual but important side effect of lithium carbonate therapy. It is typically associated with nontoxic serum levels of lithium, is thought to result from an idiosyncratic effect of lithium or the biochemical mediation of the action of vasopressin in the

renal tubular cell, and is generally thought to clear within three weeks of discontinuation of lithium therapy (1–3). A case we reported in the medical literature (4) and another case report (5) indicate that nephrogenic diabetes insipidus secondary to lithium does not always clear rapidly and may, in fact, persist for up to 20 months after lithium has been discontinued.

We are drawing attention to this because, to our knowledge, persistent lithium-induced nephrogenic diabetes insipidus has not been reported in the psychiatric literature. Awareness of its existence may be of considerable clinical value in the management of lithium-associated polyuric and polydipsic states that do not clear with termination of lithium therapy, especially in view of the recognized efficacy of thiazide diuretics in such circumstances.

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TREVOR R.P. PRICE, M.D.
PAUL J. BEISSWENGER, M.D.
Hanover, N.H.

Tardive Dyskinesia or Not?

SIR: I consider the article "A Survey of Tardive Dyskinesia in Psychiatric Outpatients" by Gregory M. Asnis, M.D., and associates (December 1977 issue) to be sufficiently misleading as to require some discussion. The central issue is whether the condition the authors evaluated was in fact tardive dyskinesia. The bulk of the evidence indicates that it was not.

Tardive dyskinesia, although not precisely defined, is generally considered to be a dyskinesia associated with long-term use of neuroleptics. Typically, it worsens on neuroleptic withdrawal, improves on reinstitution of neuroleptics, and is often worsened by anticholinergics. It must be distinguished from a variety of other dyskinesias, some drug-related, some not.

The paper by Dr. Asnis and associates reports no association between years of neuroleptic use and the presence of dyskinesias (in fact, the dyskinetic group had nonsignificantly fewer years of use). This suggests that the dyskinesias involved were not tardive dyskinesia; if a dyskinesia results from long-term neuroleptic use, one would expect a correlation between presence of dyskinesia and years of use.

Similarly, the fact that age did not correlate with the presence of dyskinesia and the trend (not statistically significant) suggesting that the use of antiparkinsonian agents was associated with a lower incidence of dyskinesia indicate that the condition under investigation was not tardive dyskinesia.

This issue is of considerable importance. Studies of tardive dyskinesia should carefully define the condition in order to obtain homogeneous populations. Factors such as response to neuroleptic withdrawal and reinstitution and response to anticholinergics or to dopamine agonists such as amphetamines can help to identify relatively homogeneous subgroups of dyskinetic patients and perhaps to define tardive dyskinesia more precisely. The approach used by Dr. Asnis and associates lumps together patients with dyskinesias of various etiologies, which will retard any efforts to understand the pathophysiology underlying tardive dyskinesia. Moreover, calling these various dyskinesias "tardive dyskinesia" and claiming a 43% prevalence of tardive dyskinesia in psychiatric outpatients will alarm many psychiatrists unnecessarily. Certainly, psychiatrists should be aware of the possibility of tardive dyskinesia, but the dyskinesias discussed by the authors have no proven relationship to classically described tardive dyskinesia, which is often irreversible and hence of considerable concern.

Finally, mention might be made of some of the difficulties involved in the use of videotapes as described in the article. The authors report excellent reliability, but this must be distinguished from validity. Their raters clearly agreed on whatever they were measuring, but there is no evidence that what they measured was tardive dyskinesia. Moreover, the person making the videotapes apparently was not blind to whether a patient was dyskinetic or not. Naturally, when videotaping a dyskinetic subject, one would attempt to demonstrate the dyskinesia as clearly as possible and would look for moments of dyskinesia to videotape. This would not be the case with control subjects, although some dyskinetic-like symptoms would be expected occasionally in normal controls. Therefore, the apparent distinction between dyskinetics and normals might be an artifact of the selective attention of the person doing the videotaping.

JEFFREY A. MATTES, M.D.
Forest Hills, N.Y.

Dr. Asnis Replies

SIR: Dr. Mattes suggests that there are commonly accepted associated findings for tardive dyskinesia, i.e., years of neuroleptic use, age, and concurrent use of antiparkinsonian medication. Because our survey did not support these associations, Dr. Mattes suggests that we were not evaluating tardive dyskinesia. However, the literature includes conflicting evidence on the role of these factors in tardive dyskinesia (1). The explanation of these controversial findings in the literature is far from clear, although diversity of study populations may have been a factor.

In our study, we carefully reviewed recent medical history, physical examinations, laboratory assessments, and family history to rule out other causes of dyskinesia. No cases of endocrinopathy, heredodegenerative disease, or other medical illnesses could be identified that might have presented as a dyskinetic syndrome. I agree with Dr. Mattes that further studies are needed to understand the pathophysiology of tardive dyskinesia; this was not the intent of our survey. It is naïve to believe that various pharmacologic challenges will separate tardive dyskinesia from other dyskinesias. Within the syndrome of tardive dyskinesia, systematic evaluations with dopamine and acetylcholine agonists and antagonists have revealed varying responses, indicating a heterogeneous disorder (2).

In terms of the evaluation process, Dr. Mattes suggests

that the person who did the videotaping might have "tipped" the raters, since he had already decided whether the patient had dyskinesia. We doubt that this was the case. The technician who recorded the evaluation did not know which patients had dyskinesia and recorded every subject in a standardized way. Most dyskinetic patients had dyskinetic movements not only during the structured part of the exam but also during the 1- to 2-minute observation period, when only the patient was present and videotaped, and all raters concurred on dyskinesia during the latter period. The point is highlighted by the fact that one patient with tardive dyskinesia demonstrated dyskinetic symptoms only during the observation period in which all three raters independently concurred.

Finally, Dr. Mattes is concerned about psychiatrists being alarmed "unnecessarily" by our report. We found that 10% of the dyskinetic population, or 4% of the survey population, had moderately severe dyskinesia; the remaining cases of dyskinesia were of minimal or mild severity. It has been suggested that dyskinesia of minimal or mild severity and of possible recent onset may be readily reversed by discontinuing antipsychotic medication or lowering the dosage (3, 4). For this reason, careful examinations to detect early signs of dyskinesia should be a routine part of psychiatric evaluations of patients treated with neuroleptics. Until the clinician has more guidelines as to which patients are most vulnerable, every patient taking neuroleptic medication must be viewed as susceptible to tardive dyskinesia. Part of good patient care should be an "overconcern" for the development of this syndrome.

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GREGORY M. ASNIS, M.D.
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Positive Side Effects of Lithium?

SIR: The article by Sahayl J. Nasr, M.D., and Robert W. Atkins, M.D., on "Coincidental Improvement in Asthma During Lithium Treatment" (September 1977 issue) in association with the observations of Peter L. Putnam, M.D. (Letter to the Editor, March 1978 issue) concerning remission of dermatologic symptoms in two patients with eczematoid dermatitis treated with lithium prompted us to report another possible "positive" side effect of lithium observed in two patients with seborrheic dermatitis.

The dermatologic symptoms of a 63-year old manic-depressive man cleared completely 1 month after initiation of prophylactic lithium treatment and have not reappeared during a 5-year follow-up. A similar effect was observed in a 32-year-old schizophrenic woman with severe seborrheic dermatitis who was treated successfully with low doses of lithium for her premenstrual tension syndrome. It is worth noting that remission of both premenstrual and dermatologic symptoms was achieved with a lithium dose of only 300 mg/day, for one week before expected day of menstruation.

We are currently pursuing the issue further by conducting a double-blind investigation on the effect of low doses of lithium carbonate on nonpsychotic patients with seborrheic dermatitis.

G.N. CHRISTODOULOU, M.D.
A.G. VARELTZIDIS, M.D.
Athens, Greece

Mental Health Services in HMOs

SIR: In "The External Provision of Health Maintenance Organization Mental Health Services" (June 1978 issue) David J. Muller, M.D., offers unsubstantiated opinions about HMOs. My experience as a director of integrated mental health services at the Harvard Community Health Plan (HCHP) since 1969 leads me to believe that the comparison of alternative models must be addressed objectively.

How are "quality," "flexibility," and "personal care" superior in Dr. Muller's clinic? He does not cite norms of staff training or experience, individual or group performance standards, supervisory practices, evaluation of treatment outcome, quality control mechanisms, or organized consumer input—features of many HMOs. Contractual negotiations seem an insufficient substitute.

Although I find many statements in the article objectionable, two oversimplifications require response. The first relates to cost: If "cost to the HMO" is less (no numbers are given), what about cost to the member?

In stating, "When the benefits are used up, there may be insufficient motivation to pay out of pocket and continue therapy," and "The initial sessions may therefore have been wasted," Dr. Muller ignores the vast literature demonstrating effectiveness of brief treatment. With staff motivation, treatment can be planned so that most patients can be treated within insurable limits. At HCHP, where extended psychotherapy is available, less than 1% of patients seen require treatment beyond the benefit limit of 20 sessions a year. A system using prepaid benefits to introduce patients to fee-for-service treatment will be inexpensive to the HMO and profitable to the clinic but expensive for the member.

Capitation payments go farther if fewer "heads" show up. Current estimates suggest that 10%–15% of the population may require psychiatric services (1). A cost/benefit assessment should include numbers referred and seen, cancellations, and no-show rates. At HCHP, 12% of members who use health services in a year come to the mental health service. Combined cancellation and no-show rates are less than 10%, and over 82% of patients referred show up. Other external services report similar experience (2). Comparison figures from Dr. Muller would be of interest.

The second area I would like to address is liaison. A number of authors have urged shared responsibility for mental health care (1). In a medical setting, where doctors and nurses see many psychological problems, this must include willingness to diagnose and treat common disturbances as well as readiness to refer when necessary. The notion that "at least monthly conferences" with HMO physicians will adequately support such efforts is naïve. At HCHP, approximately 30% of medical patients receive a diagnosis of emo-

tional disturbance during a year; half are treated by the medical provider, half are referred. Mental health staff support this function by spending 10%–20% of their time in liaison activities and joint programs. Physical contiguity facilitates this.

Dr. Muller's arguments for an autonomous, cohesive mental health group, with control over staff appointments and training, programmatic decision making, and a portion of the budget, are compelling. These factors characterize our system and are essential. I agree with his concern about softening the medical model. Insofar as mental health professionals can make such a contribution, this calls for a greater presence, visibility, and influence in the medical setting, not for leaving it.

In summary, I believe that generic conclusions about HMOs should be withheld pending data that permit meaningful comparisons.

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MICHAEL J. BENNETT, M.D.
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Dr. Muller Replies

SIR: I would like to reemphasize a major point of my paper. Any feature that the HMO wants in its mental health services can either be incorporated into its own internal department or specified in the contract with an external group. This could include many of the considerations Dr. Bennett emphasizes in his letter, such as staff training and experience, supervisory practices, evaluation and outcome studies, quality control, organized consumer input, extensive liaison services, and as much presence, visibility, and influence as desired. With these and other features equalized, I feel the autonomy and incentive of the external group give it a real advantage. Having been on both sides of the fence, I can state this definitely. Cost to the member is the same with an internal or an external group. When the HMO coverage is exhausted, the member must pay out of pocket regardless. I thank Dr. Bennett for his comments.

DAVID J. MULLER, M.D.
Denver, Colo.

Using Phylogenetic Mechanisms in Classification

SIR: We would like to comment on "A New Method of Classification for Psychophysiologic Disorders" by John G. Looney, M.D., and associates (March 1978 issue). The dilemma of classifying psychophysiologic disorders has been one of choosing between a characterologic determinant responding to an environmental situation and the resulting dysfunction of a target organ.

We propose an alternative solution. In our attempts to establish the biological underpinnings of psychophysiologic disorders, we have been concerned with the phylogenetic mechanisms that underlie their emergence. We see these as innate responses that are now inappropriate but were once adaptive.

For example, an irritable bowel syndrome becomes flight reaction, abortive and partial, manifested by increased evacuation of large bowel and bladder; spasms of the neck muscles become dominance/submission conflict, manifested by spasms of the muscles that raise and lower the head; angina pectoris becomes mock death reflex, abortive and partial, manifested by spasms of coronary arteries; and chronic indigestion becomes flight reaction, inhibited, resulting in chronic anxiety and manifested by impaired secretions of the upper alimentary tract.

We even have a place for the hypochondriac within this schema: impaired filtering of vegetative sensory stimulation (target organs are skin, heart, abdomen, etc.), cognitively elaborated as "cancer," "tuberculosis," or unspecified illness.

This is but a sampling. We are aware that this novel approach could be considered too controversial to use as a classificatory scheme; nonetheless it permits the use of ethologically verifiable instinctual response systems, placing the symptoms into a biological context, alludes to an open-ended stressor that sets the mechanism into action, and allows the avoidance or inclusion of metapsychological concepts depending on whether the emphasis is on a medical or psychological model.

A. DAVID JONAS, M.D.
DORIS F. JONAS
London, England

Correction

In the August 1978 issue there was an error in "The Prevalence of Schizophrenia: A Reassessment Using Modern Diagnostic Criteria" by Michael Alan Taylor, M.D., and Richard Abrams, M.D. The reference to the study by Weissman and associates (cited as reference 14) in the last sentence of the first paragraph of the Inpatient and Outpatient Studies section (p. 946) should appear in the Population Studies section. The last sentence of Inpatient and Outpatient Studies should therefore refer to 8 different admission prevalence figures (range of 0.6%–6.7% is unchanged).

The staff regrets this error.

Am J Psychiatry 135:10, October 1978

BOOK REVIEWS

The Treatment Techniques of Harry Stack Sullivan, *by A.H. Chapman, M.D. New York, N.Y., Brunner/Mazel, 1978, 229 pp., $13.50.*

Some of Sullivan's followers will not like it, but Sullivan is revealed in Chapman's new book as a closet Adlerian. Perhaps neo-Adlerian would be a more acceptable term for Sullivan, who all too often is given the ill-fitting label of neo-Freudian. Sullivan was neo-Adlerian to a degree—not entirely. He reflected the contributions made by the behavioral sciences of his era: the social behaviorism of George Herbert Mead and the American pragmatic philosophers; the study of face-to-face relationships done by Charles Horton Cooley, Robert E. Park, and the rest of the Chicago school of sociologists; the linguistic and cultural relativism of Edward Sapir and other anthropologists; and the rejection of libido theory and other metapsychological efflorescences in Freudian orthodoxy by "cultural" psychoanalysts such as Karen Horney and Clara Thompson. Unlike Adler, Sullivan steered clear of Marx and socialism and stayed close to the liberal capitalist ethos. Like Adler (and Adolf Meyer, for that matter), Sullivan preferred the least ornate explanation that could be given of what goes on between therapist and patient.

Sullivan and Adler both put a premium on the recent past rather than the remote past. Sullivan was not afraid to deal with thinking as well as feeling because human beings do have heads. Like Adler, Sullivan saw the importance of self-esteem and fear and the compulsive tendency that patients have to write scripts of jargon and obfuscating self-glorification when they are upset. However, Sullivan tried to bring peace instead of the more Adlerian sword of confrontation when he dealt with shy, overly intellectualized, fearful human beings who were patients. He strove to have the patient more comfortable by the end of each session. Sullivan acknowledged, as did Adler and Freud, the importance of formations of self-esteem during infancy and childhood and how these early attitudes of self regard or patterns of interpersonal coping could recur in bad times during later life to intrude and distort contemporary relations of individuals. If calling these "parataxic distortions" seemed cumbersome at the time of Sullivan's promulgating the term, today it seems fully salutary and reasonable to have the idea if not the term. Again, like Adler, Sullivan originated ideas that have permeated psychiatric thought, Sullivanian or otherwise. One trusts that the benefits derived by patients outweigh the neglect so many show in paying tribute to Sullivan, who did the initial working through of many important views about the means and techniques of therapy.

Never wholly consistent about it, Sullivan pontificated that intrapsychic identity is fictive, that only interpersonal relations are real, and that a person is defined by his other security operations plus his or her dynamisms. He played down the individual side of humanism but played up sociability and relatedness. Thus Sullivan is a prominent figure in social psychiatry and relationship therapy.

Chapman's codified presentation of the essence of Harry Stack Sullivan's treatment techniques has great virtue merely because it simplifies the murky prolixity of the original. It is a condensation and refinement that will be very useful for all nondoctrinaire psychotherapists willing to think above all of the mentally ill patient's rights to love, care and improved social functioning. The patient is put ahead of all theoretical expectations in the Sullivanian approach. The patient is treated gently. The patient needs competent professional help but is unfailingly respected as a person. The therapist also has "needs" or values that cannot be overlooked, however much the therapist's yearnings have to be curbed in a professional career as a psychotherapist, Sullivan said. Chapman conveys Sullivan's tenet that simple humanness furnishes the underpinnings of a professional, technical, expert, almost acrobatic interaction between a therapist and patient—so that the therapy enterprise is both interactive and real while it is professional, delimited, and artificial.

A clear writer who gets right to the point, Chapman has us by page 2 into the ways Sullivan taught to induct a patient into the collaborative role required in psychotherapy. From there (until the flagging chapter 10) the pace is brisk, the tone bright, and the message elucidating. The present tense use of "Sullivan says" gives one pause occasionally but only momentarily because the reader recalls that Sullivan has a vital influence that endures. When questioned about his use of the present tense in referring to George Bernard Shaw, as if Shaw had not died in 1950, Archibald Henderson (the mathematics professor named by Shaw to be his official biographer) answered, "Shaw never dies." So it seems also to be in Chapman's attitude about Sullivan.

Sullivan's therapy-related views are succinctly delivered—views about the psychiatrist as a participant observer, his stress on the interpersonal or on self-esteem, his concepts of dynamisms and security operations, his admonitions that therapists be thrifty about time, his setting up the goal of enlarged awareness of interpersonal functioning, his insistence on details and examples and allowing very slight desensitizing anxiety (adumbrating behavior therapy operations), and his seeing "consensual validation" as a process instead of an event of accord or agreement.

Sullivan rejected some of the grosser and more implausible psychodynamic formulations based on medical mechanics as the font of metaphors. However, he kept to an airy physics vocabulary when he stated his postulates about energy transformations in human motives ("dynamisms"). Sullivan held that security operations can be healthy as well as unhealthy, but his view of man was not much less struggle-filled than that of Sigmund Freud. Sullivan also saw patients as striving, coping, defending, and fighting to ward off malignant forces that impinged on their lives. Sullivan hardly foreshadowed Maslow by dwelling on human potentialities or on the human drive to self-actualization; instead, he was clinical and problem focused. A little romantic optimism inhered, nonetheless, in his view that a person freed of distortions can unleash "a tendency toward health."

Although Chapman portrays Sullivan correctly as a therapist who knew that oppression exists, it seems to me that both Chapman and Sullivan err when they approvingly furnish examples of a therapist asking a patient about the motives or feelings of a third party (pages 9, 11, 14, 15, 29, and throughout): For example, Do you think your father found this gratifying? Was your mother fearful? Was your mother afraid of arousing irritability in other people? Did your mother think you were the only person who did poorly in school? What else was happening in your mother's life to make her so irritable? In my opinion it is a cardinal rule that a patient may feel exploited by others, but a falsifiable hypothesis for deriving consensual validation does not pull in questionable speculations about the motives and goals of people not present in the consulting room. It is always easier to talk about Fred Martin's problems than one's own (p. 43).

As a fine Sullivanian himself, Chapman adheres to common sense and brevity to create a work that is laden with literary virtues and is a practical aid to psychotherapists. Chapman is disinclined to overstate his case or to overpraise Sullivan. He does not vulgarize Sullivan either. Chapter 11 holds a refreshing synopsis of Sullivan's life and writings and a genuinely useful, not overpowering bibliography is included at the end of the book. Chapman's writing is gifted, pure and simple. The good design and typography of the book serve to give a dash of lagniappe to an extremely worthwhile primer on psychotherapy in Sullivan's mode.

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Megavitamin Therapy: In Reply to the American Psychiatric Association Task Force Report on Megavitamins and Orthomolecular Psychiatry, *by A. Hoffer, Ph.D., M.D., and H. Osmond, M.B. Regina, Sask., Canada, Canadian Schizophrenia Foundation, 1976, 123 pp., $3.50 (paper).*

The history of medicine is scarred by a variety of controversies that have served more to smear reputations than to further our understanding of illness and its manifestations. The megavitamin treatment of schizophrenia is a superb example of such a controversy. The American Psychiatric Association appointed a task force that reported on the use of megavitamins for the treatment of schizophrenia. The task force concluded that there was no compelling scientific evidence that megavitamins were useful (1). This report was quite influential in diminishing the interest, let alone enthusiasm, of most American psychiatrists for the use of vitamins in high doses for the treatment of schizophrenia. Hoffer and Osmond, two of the leading proponents of this approach, have written a rebuttal of approximately 50 pages. This is supplemented by another 70 pages of appendixes that the authors feel support their position.

The tone of the volume is at best polemical and frequently deteriorates sharply to a level that has no place in scientific discourse. There is nothing to be gained by my adding more acrimony to that which has already entered the published literature. The authors argue that the task force members were biased and dishonest. They argue that the task force "tailored the report in order to bolster their biased conclusions." Motivations are repeatedly impugned, and it is suggested that at least some members of the task force were essentially servants of the pharmaceutical industry.

To those who are interested in controversies in medicine or have a particular interest in the treatment of schizophrenia this volume may be of interest. It can be read intelligently only by someone familiar with the literature and the task force report. Admittedly, megavitamin therapy has not been refuted because it is not scientifically possible to do so. One can only fail to show a value, one cannot demonstrate a lack of value. Clinicians must make up their minds on the basis of available evidence as to the most appropriate way of treating their patients. In a heterogeneous disorder such as schizophrenia there may well be a subgroup of patients for whom this approach will be useful. It would be of particular interest to know if this hypothetical group represents a fraction of a percent or is substantial in size. Such questions cannot be addressed in the present climate of charge and countercharge.

Competent and conscientious people have designed studies that failed to show a value to megavitamin therapy. Other colleagues who are equally competent and conscientious have reported studies that show positive results. Most workers in the field have concluded that the data do not support the use of megavitamin therapy except as an experimental technique. This position can be rejected by referring to its proponents as members of the Establishment. Equally pointless criticism can be made of those who support the use of megavitamin therapy. I hope that practitioners will have enough good sense to be guided by the facts rather than by the cleverness of the barbs. The major conclusion that can be drawn from this whole sorry controversy is that science is indeed a fragile activity that is most sensitive to injury by intense affect and certain human motivations.

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Readings in Psychotherapy with Older People, *edited by Steven Steury, M.D., and Marie L. Blank, M.S.W. Rockville, Md., National Institute of Mental Health, Division of Special Mental Health Programs, Center for Studies of the Mental Health of the Aging, 1977, 233 pp., no price listed (paper).*

The editors of this work modestly state that their book is "offered as support and encouragement to therapists working with the elderly." What they deliver is a book that injects much-needed optimism into geriatric psychotherapy. In the preface to this compilation, Dr. Gene Cohen says, "The topic of aging generally stimulates much discussion about the need for breakthroughs in knowledge and techniques. However, we already know a great deal about . . . older people." He goes on to describe the need for information to be assembled to facilitate application of expertise that can alleviate much of the mental suffering of older people. This charge sets the tenor of the book, which focuses on the dynamics of aging and psychotherapy for the elderly within a historical framework.

The book contains papers by 26 contributors, 21 of whom are physicians. The papers fall into three broad categories: psychodynamics of aging, issues in psychotherapy for the elderly, and psychotherapeutic techniques. Each paper has been previously published, so for the reader familiar with the

literature this work provides a pleasant review and for the neophyte there will be new insights and useful suggestions of where to begin in therapy. The book would have been strengthened if the papers had been organized into a format with a clearer purpose and providing more depth to the material.

The papers chosen for this book uniformly reflect genuine optimism and enthusiasm for geriatric psychotherapy as well as warmth toward older people. These attitudes require much insight, training, and experience on the part of geriatric psychotherapists. These qualities are unquestionably possessed by the contributors to this work. The authors skillfully achieve their purpose of sensitizing therapists to the needs of the elderly and to the huge and frequently unrecognized potential of psychotherapy for the elderly.

Although there is little material in the book that can be readily applied by therapists who are not already knowledgeable in the field of aging and experienced in psychotherapy, there is a richness of information in this readable and rather short publication that could be of considerable value to therapists of all backgrounds who work with the elderly. This publication will probably become a standard reference in recommended reading for programs designed to teach or provide therapy for older people. Its primary purpose and value is in providing an honest, sensitive, and concise account of aging and selected issues in therapy. In doing this, the editors make a timely, implicit case for less pessimism and more psychotherapy in treating old persons.

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Mind Control, *by Peter Schrag. New York, N.Y., Pantheon Books, 1978, 317 pp., $10.00.*

Mind Control is a lesson in polemics and a handbook for the antipsychiatry movement. If one accepts the definition of "hatchet job" as a maliciously destructive critique, we have an excellent example in this book. A master of hyperbole, Schrag cleverly selects the unusual dramatic event. Stopping just short of accusation, he cites an incident in a manner that suggests the purpose was nefarious and the episode typical. He is a master of guilt by association. Newspaper articles are cited as equal in evidence to scientific publications in refereed journals. Sixty pages of footnotes are arranged to suggest a careful scientific investigation.

In this book the culprits who would control your mind are psychiatrists and other mental health types. The scenario begins with a multitude of psychiatric labels and cleverly suggests that they could be applied to you and me and anyone. It moves on to the fears that computers and data banks engender. With such instruments readily available, the secrets in one's dossier are available to anyone at the punch of a button. It paints the picture of clever forces used to control individuals by subtle behavioral modification. The right combination of reinforcement will soon achieve the response desired. If that does not work, aversive methods will teach you not to do something as effectively as conditioning will promote positive action.

Schrag claims that there are more mental health workers in America than policemen. That is an interesting comparison. The mental health empire he describes achieves the "outreach of an octopus," developing liaison with schools, law enforcement, and all manner of agencies, all of it "backed by extensive advertising." If this is not enough, the acknowledged overuse of medication becomes even more frightening when one suggests that it is standard procedure for physicians to sign pads of blank prescriptions for others to dispense medications. The injectible forms of medication offer even more intriguing possibilities since their effects last for weeks. Schrag suggests that "if one can inject the recalcitrant . . . there will be no need to put Thorazine in drinking water." Here is a classic example of his technique, since the statement that chlorpromazine (Thorazine) will not have to be added to drinking water suggests that there was serious thought to do so.

Labels, psychological stimulation, and drugs are only the beginning. Schrag then points to the possibility of involuntary hospitalization. He suggests that the judicial hearing is a quick and cozy exercise based on a 10-minute psychiatric evaluation and 1.6 minutes with the judge. Once the victim is hospitalized, the horrors of ECT and psychosurgery are available. The book hints that patients are threatened with ECT if they do not behave. This treatment "burns and blasts out the electrochemical brain data." As if this prospect were insufficiently frightening, the threat of permanent mind control through psychosurgery is cited. After all, the author states, "hospitals are full of examples of individuals who were mutilated or whose intellectual capacities were destroyed by the lobotomies of the forties and fifties." The prospect becomes more vivid as he cites the only (to my knowledge) report of ECT behavior control from a project in Viet Nam, or a proposal made by one person to screen 8-year-old children for delinquent tendencies and incarcerate hard-core individuals. He further suggests that electrodes implanted in the brain could permit remote control of behavior. The problem is that this is not presented as science fiction and will be read by many as evidence of the real intent and professional activities of all psychiatrists. The technique is that mentioned in the first paragraph—clever use of a single unusual incident to suggest that it will become the standard for all psychiatric practice.

What does Schrag propose to do about the problems that psychiatrists call mental illness? He suggests that there would be no such problems if we had full employment and a shift of resources from therapy and "medical" interventions to the improvement of the social conditions that caused the problem in the first place. He insists that any psychiatric intervention be limited to serious cases (whatever they may be). He proposes that the symptoms of psychosis are simply idiosyncratic behavior or diversity of belief and that we should tolerate them as differences and not as deviance. He suggests absolute prohibition of treatment for unwilling individuals and elimination of any commitment laws. Public funds should be removed from the control of doctors and institutions to the control of "clients," who can then choose whether to use them for formal therapy, housing, or "simply a month at the beach which is likely to be at least as therapeutic as a month in the state asylum."

Lest the reader dismiss this book as "far out" or unusual, he or she should be reminded that similar arguments resulted in the passage of legislation that seriously restricted the use of ECT (even for voluntary patients) in California and other states. There has been a spate of proposals that mental illness is but a myth. Television programs suggest that psychiatrists use their patients sexually or incarcerate them unjustly. The patient suffering from mental disorder is hampered by stigma and prejudice against mental illness. The psychiatrist is pointed at as the opponent of freedom of thought and the proponent of mind control by a variety of evil schemes. Psychiatrists ought to read this book as a mat-

ter of education. Get to know what is said about you. It is no wonder that Mrs. Carter and the President's Commission on Mental Health have pointed to the goal of public education as a major requirement for an effective program for dealing with mental illness.

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The Mental Health Industry: A Cultural Phenomenon, *by Peter A. Magaro, Robert Gripp, and David J. McDowell, with the assistance of Ivan W. Miller III. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 259 pp., $17.95.*

This is at once a highly disciplined book and a highly imaginative one; in neither dimension is the adjective used pejoratively. The authors are also ambitious in that they set out to cover a number of profound concepts, introduce detailed proofs of each thesis, and still add daring new ideas aimed toward solving the troubles they identify. The book is about many things, each intertwined with the others.

The authors briefly present the perspective that what we recognize as madness is sorely dependent on cultural conceptions. The idea that social expectations and demands define the deviant as mad is repeated more times than are examples offered to illustrate. Most readers can interpolate, however, and the bibliographical leads are excellent.

The text then turns unexpectedly but on second thought appropriately to a very precise discussion of two modern ideas of treatment for mental disease, milieu therapy and behavior modification. An excellent technical review of research data in both fields illustrates how inept most such research is when measured by actual effectiveness for the individual. The authors show that most research amounts to a lifeless default of any attempt to assess effectiveness beyond lip service to the established mode of thinking about things psychiatric.

The middle section of the book describes a vivid and often (for me, at least) uncomfortably detailed view of the mental health industry as it exists in America today. The authors insist that they are not interested in attacking their colleagues but are merely describing the current scene from a cultural perspective. Like psychoanalytic interpretations offered out of the goodness of our hearts, this kind of benevolent gift is sometimes experienced by the recipient as if it were hostile. I suspect many readers will feel the same about this book.

The central idea is something like this: since cultural structures have created our treatment systems, both the theoretical and the institutional structures have become solidified in order to perpetuate their own beliefs. The individuals involved in this process could be mental health bureaucrats, behavior therapists, or psychoanalysts. Mental health professionals are thus ultimately totally invested in maintaining the status quo, immersing themselves in the more pleasant patients who fit their particular scheme and ignoring the rest. Researchers also fail to address the essential problems of treatment effectiveness, attending instead to tiny details of behavior variations so they can produce publishable papers to enhance their own status quo. The public, the body politic, and the practitioners do not demand effective research because it might introduce change into their small smug world.

The book's section on the personalities of the providers who stifle research and narrowly hold to theoretical or institutional fixtures is especially choice. My 35 years in psychology and psychiatry have not been so secure that I felt no discomfort in reading this part of the text. Of course, the authors are more accurate in helping me perceive the flaws of my peers than in reminding me of my own flaws. They arouse most unpleasant memories of bitter jurisdictional battles in which I have unhappily participated.

The authors then shift focus once again. Although still in the mainstream of mental health politics, they expound a possible solution for this complex problem. Using the example of the GI Bill of Rights, according to which vouchers were in effect issued to individuals who could then attend any approved and monitored school, the authors suggest a similar plan for psychiatric treatment. In this last part of their presentation they provide a great deal of specific information and quite specific recommendations to implement their thesis. Specific predictions are made as to the probable results of this shift of economic perspective.

The authors feel that if funds were available to the individual for therapy of mental problems the effect would be free choice of practitioners and sharply increased demands on practitioners for therapeutic results (as opposed to pleasing the mental health commissioner). These practitioners in turn would demand from psychologically oriented researchers more practical research. Reading this I suffered at the agonies that would come if such a path to the platonic ideal were followed, a point not missed by the authors. The idea of individual enterprise comes as no shock to a conservative midwesterner. In fact, it seems a most familiar if often maligned point of view. Interestingly, the authors themselves are even a little apologetic: "For those of us coming out of a liberal background, the encouragement of a treatment system based on the concept of free enterprise seems a bit paradoxical" (p. 216). This is just one paradox that will greet readers of this book.

If your mind is set and you are ensconced in a pattern of practice beyond change, do not bother to read this book. You will only include the authors with other nonbelievers. If you are able, although perhaps not eager, to accept profound challenge as a researcher, practitioner, consumer, or part of the body politic, then read this book—not as gospel, of course, but as the provocateur it is.

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Autistic Children: Teaching, Community and Research Approaches, *compiled and edited by Barbara Furneaux and Brian Roberts. Boston, Mass., Routledge & Kegan Paul, 1977, 193 pp., $12.00.*

One gets the distinct impression from the beginning of this book that there is still some controversy over the causation, diagnosis, and management of infantile autism, or Kanner's syndrome. This book promulgates the view that education is the most important factor in the management and treatment of the autistic child, along with active involvement of the parents in the care of the child, which is in direct opposition to Bruno Bettelheim's idea of rigid exclusion of the parents from the entire process of management, especially from the therapy of the autistic child. The authors stress that "the condition of the autistic child is not static nor are they a homogeneous group" and that no one type of professional can successfully deal with all the aspects of what they term the "autistic condition."

Brian Roberts' description of the conditions of autism and theories of causation in chapter two is instructive. Roberts examines closely Kanner's behavior criteria and some of Eisenberg's work and discusses the causation theories of Deslauriers and Carlson, which he feels to be most promising. In chapter three Furneaux talks at length about differential diagnosis and distinguishes among autism, childhood psychosis, childhood schizophrenia, and subnormal children. She lists the clues that the teacher can observe in a child's responses for the purpose of diagnosis: 1) Does the child respond? 2) How complete is his or her response? 3) Is his or her response appropriate or bizarre? 4) What is the normal speed of his or her responses? 5) Has the child the ability to follow through his or her responses in an appropriate manner? 6) Can he or she go from one thing to another when responding? 7) How well and for how long does he or she retain information? 8) Can he or she apply the information to a new set of circumstances? One phenomenon noted by some teachers is that an autistic child gives a bizarre response, but a subnormal child gives a slow response.

In chapter five Furneaux continues to stress the importance of the teacher in beginning the educational process for the autistic child. The teacher must define objectives and decide on an approach on the basis of considerations of the personality of the child and the extent of the child's remoteness and must constantly reevaluate methods for effectiveness according to the response of the child. The author seems to feel that the teacher can often successfully "lead from behind" and get the child going in a positive direction by means of close observation and patient attention to the child's individuality.

In chapter seven, which deals with research and teaching of autistic children, no clear methodology is outlined. Approaches to teaching include use of psychodynamic techniques, in which success is difficult to measure, and behavior techniques, which are easily measured but are difficult to duplicate outside the prescribed setting. The authors of this chapter note the common failure of behavioral techniques acquired in one situation. They conclude, however, that "not enough research has been done to enable the decision to be made as to which of the available approaches to the treatment and education of autistic children—the psychodynamic approach, behavior modification, or the use of a structured environment—has the most to recommend it."

This book gives a good basic understanding of the complexity of autism and how to differentiate it from other conditions. It tells succinctly what is being done currently in therapeutics and in education and what research is being contemplated. The authors are careful to document all their findings with a great deal of actual clinical material. It is worth reading and will make a good basic reference book, especially for teachers and for others helping in the care of autistic children.

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Clinical Aspects of the Rapist, *edited by Richard T. Rada, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 225 pp., $15.50.*

This latest book in the Seminars in Psychiatry series is a timely as well as an important contribution to the professional literature on sexual assault. In the past five years there has been a dramatic increase in the number of publications on rape with a primary focus on the victim. As Rada points out, interest in the rapist has been confined to media coverage of sensational sex crimes committed rather than any consistent and systematic pursuit of the subject. The women's movement is credited with raising society's consciousness to the many factors of the problem. Now that the mental health, social service, law enforcement, medicine, and justice systems are aware of the increased reporting of the crime, the issues of prevention and control of rape are especially salient. There is a definite need to understand the offense, the offender, and the victim.

The focus of the book is on the rapist, defined as an adult male who sexually aggresses against an adult female. The strength of the book is in its multidisciplinary approach. Issues raised regarding the rapist are dealt with from many viewpoints. The stated purpose of the book is to present under one cover previous knowledge and new developments in the biology, psychology, sociology, and treatment of the rapist. This goal is accomplished.

The book begins with Rada's historical survey of the sexual psychopathy literature, starting with Krafft-Ebing's 1886 publication and continuing with writings from Freud, Ellis, Abraham, Ferenczi, and Stekel. The American literature began with Karpman's publication concerning a case of obscene letter writing in 1923 and expanded to include major works by Karpman, Gebhard, MacDonald, and Fain. A review of the key concepts in sexual psychopathy—perversion and sadism—includes a discussion of current trends in ego psychology as well as psychological conflicts over issues of power, control, and dominance.

One controversial issue among clinical researchers is the significance of sexuality in the commission of a rape. Rada cautions that we are still in the early phases of developing techniques to quantify sex drive and desire in rapists. A second equally controversial issue is the etiology of sex offenses. Various speculations have been offered and discussed: psychoanalytic theory (inordinate castration anxiety and acute oedipal conflicts caused by childhood witness experience), learning theory (first sexual experience as aversive experience reinforced through masturbation), egopsychology (interaction between conscious and unconscious attitudes of parents and subsequent attitudes and behavior of their children), psychodynamics (hostility toward authority figures), and sexual inadequacy (fear of sexual contact with women).

Following the introductory chapter is an in-depth discussion of biological, psychological, and social factors of the rapist. Rada is judicious to caution the reader not to assume that there is sufficient evidence to favor one point of view over another. This approach is quite refreshing given the amount of research in this field.

The psychological motivation for rape, according to Rada, is the desire for power, control, and dominance. The means for obtaining this control is the commission of the rape offense because sex represents to the rapist the best way to deprive a woman of her personal control. The differentiating factor in rape is the mode by which this control is effected (e.g., for some the manner of raping is sexual, for some it is primarily aggressive, for still others it is primarily humiliating). Rada describes the mode of raping on a continuum from sexual to aggressive with the middle point being sadistic.

An interesting conceptualization by Rada presents rape as a defense against feelings of sexual inadequacy, dependency needs, and homosexual wishes; rape as a displacement of hostility onto the victim; and rape as a symptom of a madon-

na-prostitute complex. Rada also reviews various psychological studies relative to early life experience of the rapist, intelligence and physical characteristics, dream material, sexual adequacy, marital relationships, and alcohol and drug use. The psychometric studies on sex offenders have many methodological problems. However, the studies are carefully reported and useful as background information, and some of the tests may be useful to the psychiatrist preparing diagnostic and disposition statements.

Contributions from sociology to the understanding of rape have until recently been derived primarily from criminology, a subdivision of sociology. It has been the emergence of the newer field of victimology that has prompted sociologists to focus attention on the subject. In an excellent chapter, Park Elliott Dietz lucidly summarizes the available literature on rape and sociological theories (crime as normal, anomie theory, differential opportunity, the delinquent subculture, role theory, and social learning theories). Dietz also reviews in detail empirical studies on rape and rapist behavior (variables of social status, race, and religion, pornography, temporal and spatial patterning, relationship between victim and offender, group rape, and cross-cultural perspectives). In a concluding section to his chapter Dietz introduces a thought-provoking area of injury control research that is a developing branch of public health. A schema is presented to depict the rape countermeasures (offender behavior, victim behavior, social and physical environment) designed to reduce the frequency or severity of injuries (by phases of prerape, rape, and postrape). Dietz advises that there be emphasis on social control of rape, believing that eradication of rape would require radical changes in our class structure, the social and spatial mobility of individuals, and the right to privacy. He states that rape is ineradicable and believes an important direction for future research is the reduction of frequency and severity of victim trauma.

There is a chapter on a classification system for rapists that Rada has found useful in clinical work and courtroom testimony. It focuses on descriptive psychiatric categories of the rapist and includes five different types: the psychotic rapist, the situational stress rapist, the masculine identity conflict rapist, the sadistic rapist, and the sociopathic rapist. Drawing from his research and literature review, Rada addresses biological aspects of the rapist (genetic neuroanatomical, neurophysiological, and psychoendocrinological factors) and organic methods used in treatment of the rapist (castration, psychosurgery, drugs, and ECT).

Chapter six, "An Integrated Treatment Program for Rapists," is essential reading for anyone interested in or currently treating rapists. This behaviorally oriented treatment model, designed and implemented by Abel, Blanchard, and Becker, introduces many new approaches in terms of research on rapists. The research project draws its sample from referrals and nonconvicted offenders, which counters the criticism that only convicted offenders are ever studied. This chapter helps to synthesize the abundance of material from the previous chapters. The clinicians identify five major areas of treatment: 1) establishing an empathetic relationship with the rapist, 2) confronting him with the fact that he is responsible for his rape behavior, 3) heterosocial-heterosexual skills training, 4) increasing arousal to adult women, and 5) decreasing arousal to the urge to rape. Detailed material from research and clinical practice is given for each of these areas, and the specific focus is on the techniques developed to reduce deviant sexual arousal (electrical aversion, covert sensitization, and chemical and olfactory aversion). The methods to develop heterosexual arousal (aversion relief, systematic desensitization, social retraining, classical conditioning, fading, and masturbatory conditioning) are also described. Most helpful are the case illustrations of the techniques developed.

The last chapter, on legal issues, is a dramatic departure from the style and content of the preceding chapters. Until this point, the chapters are clear, concise, and extremely well documented. Suddenly in chapter seven, the reader is told that one of the roles of the psychiatrist may be to determine the veracity of the victim's character. Attention then shifts to the concept of consent and corroborating evidence laws (including Wigmore's 1970 statement that the judge should ensure that a qualified physician take a social history of the victim and conduct a mental examination). Sadoff states that the psychiatrist should render an opinion as to whether the victim is fabricating the rape, covering up for her sexual conduct, or fantasizing the rape. The title of chapter seven is "The Psychiatrist and the Rapist," but no guidelines are given as to the types of questions or assessment the psychiatrist might follow in determining the rapist's mental state and the impact of his social and sexual history or fantasy life. If Sadoff wished to address issues relating to rape, he might have cited some of the aftermath symptoms that are useful in documenting trauma and that might help in preparing testimony for either prosecution or defense. To compensate for this chapter, I would refer readers to Elaine Hilberman's book (1) for a sensitive and well-documented account of victim trauma and legal issues.

This book is a major source of the current state of knowledge in regard to men who sexually assault women. Rada and associates have written a carefully documented book and have made an important beginning in addressing this issue from a clinical perspective. Because it is not the final word, several research questions need to be asked: What are the biopsychosocial aspects of sadism? What is the impact on children who witness sexual as well as aggressive activity? Why is rape the symptom-choice? How early can rape behavior begin? When can it be detected and how can it be treated? If rape control is to be a pragmatic concept, what research can the mental health fields bring to bear on this problem? In summary, this book is a basic text containing a wealth of information about the complex and multidetermined nature of forcible sexual assault.

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Current Psychiatric Therapies, Vol. 17, 1977, *edited by Jules H. Masserman, M.D. New York, N.Y., Grune & Stratton, 1977, 337 pp., $32.50.*

This series has held up well as a result of the energy and scholarship of the editor and the wide interests represented by the contributors. Volume 17 contains 11 sections covering administrative problems, the problems of childhood and adolescence, dyadic and group psychotherapies, psychopharmacology, alcoholism, various psychophysiological therapies, institutional problems, and some cultural considerations.

Gibson's essay on the rights of staff is a refreshing start. This chapter is especially reassuring to those of us who have

felt impaled on the horns of such therapeutic dilemmas as the patient's rights to have treatment and to refuse treatment. At least we have one voice raised in defense of psychiatrists' rights.

The editor contributes his usual introduction, in which he again demonstrates his facility with words and his preoccupation with them. After a whimsical beginning he provides a thoughtful and discerning discussion of the diagnostic and therapeutic problems that complicate our lives, opting for a reasonable return to common sense in both areas. Seymour Pollack's chapter, "Psychiatric-Legal Problems of Office Practice," is in this commonsense vein and should be required reading for every resident in psychiatry. I was especially impressed by Pollack's insistence that modifying diagnoses in order to effect certain social results is a questionable practice; the inevitable end-product of this must be a pervading distrust in diagnosis and diagnostician.

An interesting variant from the usual approach to such matters is Leonard Woodcock's address, "Psychiatric Treatment in Tommorrow's Health Care System." Since the United Auto Workers (UAW) has been a pioneer in the field of obtaining adequate insurance coverage for mental illness, it is fitting that Mr. Woodcock, former president of UAW, be chosen to discuss this topic; he does it well.

The section on childhood and adolescence includes chapters by Cohen on "Minimal Brain Dysfunction," White on "Psychotropic Drugs in Child and Adolescent Psychiatry," and Nadelson and Notman on "Treatment of the Pregnant Teenager and the Putative Father." The lattermost is a topic that might have had little significance a few years ago, but the authors point out that the reported incidence of coitus before the age of 15 has risen from 3% for girls in 1953 to 19% in 1973, with a concomitant increase in medical, social, and psychiatric problems.

In the section on dyadic psychotherapies there is less concern than in previous years with psychodynamics and more with the practical aspects of the therapeutic process, the importance of medical orientation (by Forsythe and associates), planned interruptions in psychotherapy (by Mendel), and the treatment of the chronic patient (by Mendel and Allen). Sayre provides a cautious and informative review of contemporary radical therapies, with a word of warning that "pilgrims of self-discovery may find that there is nothing substantial to be discovered."

The authors of the chapters in the section on family and group therapies offer straightforward discussions of group therapy of the traumatic neuroses and schizophrenia. The inclusion of incest in this section is certainly appropriate in view of the far-reaching effects of this problem in the family and even in the surrounding social structure. Speck and Rueveni offer a case illustration of their social network intervention technique; I was impressed by the energy and salesmanship involved in achieving the cooperation and attendance of 60 participants not in the crisis family.

In the section on psychopharmacology there are papers on "Placebos in Psychiatric Therapy," "L-Tryptophan: An Effective Hypnotic" (an interesting new possibility), good reviews on "Tricyclic Antidepressants in Outpatient Therapy" and "Drugs and Depression: Dosage and Blood Level Monitoring," and an interesting argument for the development of a specialized affective disorder clinic under the title "The Lithium Clinic: A New Model for the Delivery of Psychiatric Services." Subspecialty clinics certainly have proven to be useful in other fields of medicine; it might be interesting to see if they could be equally useful in psychiatry.

The increasing concern over tardive dyskinesia is reflected in two chapters, one on its prevention and one on its treatment. The section on alcoholism offers a good overview of the problem by Ewing, a description of the use of a day hospital by Savitz and Kolodner, and some notes on disulfiram by Rainey.

In the section on psychophysiological therapies are found discussions of such interesting new developments as autoacupressure and multiple-monitored ECT. The final chapter is a comprehensive review of the history and present status of psychosurgery. The author of this review is correctly critical of the lack of really objective evaluation of treatment results, pointing out that anecdotal reports of "improved functioning" and personal happiness are meaningless. The review is so comprehensive that it is a little surprising that no mention was made of Lindstorm's ultrasonic technique.

The section labeled Institutional is a delight to the pragmatic reader. One has a constantly recurring sense of déjà vu while reading Schwab's history of the evolution of a general hospital psychiatric unit. We have all lived through periods when the psychiatrist was seen as "unhappily removing from the medical system" and often having attitudes that were "harmful to the community." It is pleasant to note that after describing the intervening evolutionary process the author concludes that "psychiatric and medical diagnosis, rational psychosocial therapy and pharmacotherapy and aftercare planning are the significant features of our program." Treffert's plea for a sane asylum is both provocative and practical, although he admits that the name may have to be changed to be really accepted. Oberleder's directions for hospital staff care of the aging will be supportive and reassuring for nurses and others who are distressed by the slow progress and recurring problems found in this population.

In the final section, Itil and Reisberg raise some interesting questions about cultural differences in the prescription of and the patients' response to psychotropic medication. Of course, part of the difference could stem from cultural differences in expectations. Easson's discussion of orientation in psychiatric diagnosis and therapy in the Soviet Union contains some interesting and perhaps puzzling statements. For example, it is noted that most of the doctors in the Soviet Union are women, although "an increasing number of men is going into the medical profession." This is not surprising because it is also reported that most of the senior clinicians are men, and they make the final decisions.

The editor and contributors of this volume should be proud of this useful and informative product.

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Second Thoughts: Selected Papers on Psychoanalysis, *by W.R. Bion, D.S.O., B.A. New York, N.Y., Jason Aronson 1977, 166 pp., $10.00.*

Dr. Bion is a renowned British psychoanalyst, clinician and theoretician who is teaching and practicing in Los Angeles. *Second Thoughts* is a collection of papers that were published between 1950 and 1962 and collected in one the present form with a final chapter of commentary—the second thoughts of the title.

Although these papers are mostly concerned with the psychoses, their scope is not limited to this topic, and no psychotherapist can disregard their importance to his or her work. Particularly is this true when they are read together with the commentary. Bion's skills in clinical observation,

his logic, and his original, illuminating points of view are worth the difficulties of confronting his closely packed and sometimes idiosyncratic style.

Bion's theoretical background is Kleinian, which will create a further intellectual hurdle for some readers, but again, one that will repay the effort. I acknowledge that much of Kleinian theory is confused and unconvincing for me. David Rapaport (1) stated baldly that the so-called theory of object relations of this school was an "id mythology." Nevertheless, the Kleinians number among themselves an astonishingly large group of outstanding clinicians. I have long puzzled over this fact and have concluded, tentatively and nonpejoratively, that the Klein school probably does not mysteriously and ab-initio attract for training great numbers of potentially outstanding clinicians; rather, the openness and proximity to fantasy of much of their theory permits and even assists greater access to primary process and to empathic perceptiveness of the psychic events within the patient and between patient and analyst.

Notable examples of this clinical perceptiveness occur in Bion's papers "On Arrogance" and "Attacks on Linking." One of the difficulties that psychoanalysis has had in promulgating its views is that, as Bion points out, it deals with experiences that are not available through the senses. Anxiety, for instance, is devoid of shape, size, color, or smell; the fact that the anxious patient may flush or sweat does not discredit this statement. One comes to understand the basic data of the patient, including what is verbalized by him or her and conceptualized from his or her associations by the analyst, by means of the empathized and cognitive effects that are internalized and integrated by the analyst.

It is largely true that the analyst knows his or her patient by means of what the analyst perceives and knows of his or her own internal and internalized processes as they operate in parallel with those of the patient. To tolerate in this way the stresses of the patient, to contain the patient's split-off intolerables and to remain balanced, would seem to be an occasion for the patient to feel gratitude. On the contrary, it is often the occasion for envy and hatred in the heated regression of the transference. Many therapeutic endeavors, analytic and otherwise, founder on the misunderstanding of this fact.

In "On Arrogance" Bion writes of certain patients whose analyses demonstrate "references to curiosity, arrogance and stupidity which are so dispersed and separated from each other that their relatedness may escape detection" (p. 86). He recognizes that this points to a past psychic "disaster" and to its imminent repetition in the transference through the failure of the analyst to "contain" and be a receptacle for the patient's projective identifications. I cannot summarize this further within the limited space of a book review; it is a brilliantly evocative and useful paper, deserving of clinical study and testing.

There will always be individuals who either fail to recognize that much of human experience is not reducible to physical facts or, if they do acknowledge an area of "the psychic," will deny that it is susceptible to scientific study. In recent years there has in addition been an unfortunate tendency to polarize so-called humanistic and scientific approaches to the study and treatment of mental ills. Often, such polarization has existed more in the minds of the polemicists than in the external facts. Bion's discussions of psychoanalytic patient-centered, humanistic treatment may help in dispelling the notion that psychoanalysis and humanism are incompatible.

Bion reconfirms in these and other writings the psychoanalytic employment of "freely hovering attention" and says much more that is useful about how the analyst works. He refers to the sharpening of intuition by means of the exclusion of "memory and desire" in the analyst's operations. Curiously and interestingly, he comments in this context on how the gap is to be bridged between reading the papers in this book and the psychoanalytical experience: "My suggestion is that the papers should be read in the same conditions as those in which a psycho-analysis should be conducted—without memory or desire. And then forgotten. They can be re-read; but *not* remembered" (p. 163; his italics).

This is not an expression of eccentricity; it is an operational statement that is courageous and useful. It also illustrates through the difficulties inherent in its execution some of the problems in applying the science of psychoanalysis and in teaching the finest state of its art.

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Research on Smoking Behavior. NIDA Research Monograph 17, *edited by Murray E. Jarvik, M.D., Ph.D., Joseph W. Cullen, Ph.D., Ellen R. Gritz, Ph.D., Thomas M. Vogt, M.D., M.P.H., and Louis Jolyon West, M.D. Rockville, Md., National Institute on Drug Abuse, 1977, 377 pp., no price listed.*

This book is a compendium of papers presented at a conference on smoking behavior held in Los Angeles in June 1977. It upholds the high quality of previous publications in the NIDA Research Monograph series. Although there is a certain amount of repetition and rather loose organization, the editing is generally excellent, and what is repeated is of such consequence that it deserves to be reinforced.

The content is concerned with the fundamental aspects of tobacco smoking, which, with alcohol abuse, can be viewed as the most dangerous form of addictive behavior that society must contend with. Interestingly and perhaps significantly, both these substances are legal, easily available, and persistently and cunningly advertised (pushed).

The book is conveniently divided into four sections: Epidemiology, Etiology, Consequences, and Treatment. Each of these contains papers of extremely high caliber as well as useful discussion and session overview chapters. Taken together they provide a comprehensive account of the distribution and patterns of smoking behavior, biopsychosocial variables underlying the tobacco use syndrome, tobacco-related diseases, and prevention and treatment issues.

In spite of the attention given to smoking since Wynder and Graham's 1950 report linking tobacco smoking, especially cigarettes, with the induction of bronchiogenic carcinoma, there has been a stubborn persistence of the smoking habit. Leonard Schuman describes some important trends in the patterns of smoking in the United States, showing a gratifying but disappointingly modest decline in recent years. Of great concern is the fact that women have shown far less prominent declines in cigarette smoking than have men, and teenage girls show a continuous increase in smoking behavior. Also, both males and females are initiating their smoking

habit at earlier ages. This does not augur well for the future and emphasizes the Sisyphean nature of the task of preventing this addiction.

The reasons why people smoke remain unclear, and the etiology section serves to highlight the multifactorial nature of the dependency, as is the case in other addictive behaviors. Murray Jarvik makes a strong case for the central role of nicotine in cigarette smoking, and Dorothy Green reviews the factors that have been identified as contributing to cigarette use. Obviously, there are many of these, found in varying combinations in individual smokers, but pleasurable relaxation, stimulation, reduction of negative feelings, craving or psychological dependency, and simply habit play major roles. The fact that smoking rates are highest among divorced or separated individuals is a good example of the complex interaction between sociological and psychological factors in contributing to addictive behavior. Jerome Jaffe develops a very persuasive case for viewing dependence on tobacco in some individuals as being the same kind of phenomenon as dependence on other drugs. Tobacco use disorder will be a welcome and justified inclusion in the next edition of the American Psychiatric Association's *Diagnostic and Statistical Manual (DSM-III)*.

The treatment section includes a report by Jerome Schwartz on the various techniques used to help people to stop smoking and a discussion of the critically important issue of prevention by Ellen Gritz. As with the other sections the authors must be given the highest credit for excellent reviews of the current status of these complicated problems.

This book is a scholarly contribution to the understanding of a serious public health problem. It will be of value to a broad spectrum of readers.

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Child Effects on Adults, *by Richard Q. Bell and Lawrence V. Harper. Hillsdale, N.J., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 236 pp., $12.95.*

This book, by two research psychologists (with a single additional contribution by a historian, Valerie French), is a collection of essays united by the theme that research and theory in developmental psychology has overemphasized a unidirectional model of interaction in which children's behavior is determined by the activities of the parents. The authors believe that this misguided emphasis has resulted in a failure to appreciate all the ways in which the behavior of adults is shaped by the activities of their children. The authors set about to overcome this error by a systematic review and reinterpretation of the literature in human and animal developmental psychology. Their method is to single out the child's effects on the parent or teacher in these studies. The goal is evidently to convince the reader of a model of developmental psychology based on constantly advancing reciprocal interactions between child and adult.

The book appears to be intended for the academic psychologist rather than the pediatric or mental health clinician. To judge from the labored and polemical style in which the authors express themselves, they apparently believe their thesis will face formidable opposition. However, the child psychiatrist or developmentally oriented clinician will find the constant repetition irritating and unnecessary. For the clinician, the concept of a finely tuned, constantly advancing reciprocal pattern of interaction between parent and child in which parents as well as their offspring develop and change is hardly novel. Clinical research in this area and its application to clinical work with children and their families has undergone great advances in the past decade or two.

The individual essays vary widely in quality. The first two chapters, devoted to enumerating attitudes toward children in Western history from ancient civilization to modern times, seem simplistic and irrelevant. This is unfortunate because several later chapters are well worth reading.

Chapters four, five, and six by Richard Bell are of particular interest. I recommend them to any student or clinician working with children. Chapter four begins with a concise review of the major studies of socialization. Bell then proposes a general explanatory model of mutual control systems by which mothers and infants regulate the upper and lower limits of their interactive behavior. This model takes into account the significance of congenital differences between children and the effect of environment and social stress on the responsiveness of the mother to the child.

Chapter five is a lucid presentation of research strategies in the field and their various advantages and disadvantages. It should be read by all who are interested in child developmental research. In chapter six, Bell presents his own formulation of the important developmental issues in the first year of life. He delineates four principal lines of development: 1) from activity to reactivity, 2) from preformed organization to malleability, 3) from stimulus reduction to stimulus seeking, and 4) proximal-distal shift. These lines of development occur within the matrix of three principal systems of mother/infant interaction: the behavioral interaction system, the caregiving interaction, and the social interaction. This developmental scheme is integrated with the research findings of Emde and associates' *Emotional Expression in Infancy* (1). I found Bell's formulations here reminiscent in some to Anna Freud's concept of developmental lines although more behaviorally oriented.

Chapters seven through eleven are by Harper. They are offered as supportive data to the major formulations of Bell and are useful principally because they pull together research findings from both animal and human studies in a concise and readable fashion.

In summary, this book contains a few chapters by Richard Bell that are pearls and deserve wide attention. As a whole, however, it is curiously ungainly and overpolemical. It is further marred by two clumsy, unnecessary introductory chapters of historical review that are likely to dissuade many readers from continuing before reaching the truly substantive parts of the book.

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Dying and Death: A Clinical Guide for Caregivers *edited by David Barton, M.D. Baltimore, Md., Williams & Wilkins Co., 1977, 238 pp., $14.95 (paper).*

We see in our society unmistakable signs of a changing attitude toward death and dying. Widespread media cov-

erage of the topic and extensive course offerings at all levels bespeak a renewed interest in the subject and call our attention to the greater frankness with which matters relating to death are being discussed. Within the medical sphere these changes are perhaps most evident. In addition to a burgeoning literature there are signs of more open communication between dying patients and their physicians. The growing application of the hospice concept with its special programs for dying persons is an expression of this new attitude.

Yet, almost inevitably, a counter reaction has begun to appear. Some feel that little is being written about thanatology that is new or of substance. Others react negatively to the sentimental appeal or expression of romanticism of some writers and lecturers. A few physicians and nurses bristle at the urging of thanatologists to leave traditional roles in order to meet the needs of dying persons. Important shifts in attitude have occurred, but they are in danger of being undermined. One hopes that the passage of time will bring research efforts that support or refute present notions and increase the scientific respectability of the field.

In this climate new books continue to appear on the care of dying patients. *Dying and Death* is intended for all who have contact with such patients and will serve them well. The first half, written by Dr. Barton, covers the subject systematically. The second half includes chapters by different authors approaching it from a number of perspectives. Discussions by dying patients, an ethicist, a physician, and a clergyman reflect both personal experience and thoughtful consideration of the subject. Although somewhat obscure at points, Dr. Barton's writing also shows the influence of experience in dealing with individuals in the crisis of dying. He provides an in-depth review of major concepts and, despite little new material, an excellent statement of current thinking.

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Biological Bases of Psychiatric Disorders, *edited by Alan Frazer, Ph.D., and Andrew Winokur, M.D., Ph.D. New York, N.Y., SP Books (Spectrum Publications), 1977, 262 pp., $15.00.*

Recent developments in our understanding of the biological bases of mental illness have been primarily in the correlates of the functional psychoses. It has been suggested that schizophrenic patients have raised serum creatine phosphokinase, raised autoimmunity, lowered platelet monoamine oxidase, raised histamine tolerance, raised CNS dopamine, and increased transmethylation. In the affective psychoses, biological theories include abnormal levels of biogenic amines, biogenic amine biosynthesis blocking studies, administration of monoamine precursors, and neuroendocrine changes. In addition, the drugs used to treat these illnesses have also been examined for what light they can shed on the biological correlates of mental illness. It has been suggested that phenothiazines block dopamine, that tricyclic antidepressants block the reuptake of noradrenaline, and that lithium impairs central catecholamine transmission. The benzodiazepines were also theorized to decrease the turnover of both norepinephrine and serotonin and to be related to the γ-aminobutyric acid (GABA) functions.

Although the genetics of alcoholism have been explored in the psychiatric literature, other biological correlates of propensities to alcoholism have usually been published elsewhere. Some workers have suggested that alcoholics have a lowered production of adversive acetaldehyde. Other investigators theorize an opiate-like substance as a metabolite of alcohol.

The editors of *Biological Bases of Psychiatric Disorders* are to be congratulated on their ability to maintain their central theme in many of the contributed chapters in this book. The findings listed above are discussed in brief, and frequent reference is made to literature sources. These theories and the experimental work related to them are exciting developments for psychiatrists interested in biological theories of etiology of various mental illnesses. Most of the chapters are appropriately restrained in their exposition of etiological relationships. The title of the book is perhaps less restrained than these chapters.

Of particular interest are the chapters on "Biological Theories of Schizophrenia" by Lipinski and Matthysse, "Biological Aspects of Mania and Depression" by Alan Frazer, "Alcoholism" by James Stinnett, and "Therapeutic and Pharmacological Aspects of Psychotropic Drugs" by Frazer and Winokur. Lipinski and Matthysse cite their more extensive chapter in the 1975 *Annual Review of Medicine* (1), but they do not emphasize this excellent reference to the extent that it deserves. This book is aimed at the introductory neuroscientist. It is more current and informative than many books with this aim.

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The Placebo Effect in Healing, *by Michael Jospe. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 162 pp., $15.95.*

Long before Beecher (1) wrote about "the powerful placebo," healers had an awareness that not all patient response could be attributed to the direct actions of any particular potion, pill, or procedure. Indeed, the origin of the word "placebo," which means "I will please," indicates knowledge that a pleased patient may sometimes be equated with a responsive and recovering patient. For behavioral scientists the mechanism of placebo response remains a research challenge demanding theories that can be tested. Michael Jospe is a psychologist who has carefully reviewed the placebo literature and now presents an excellent and up-to-date overview that should be of value to all who are interested in the topic.

After offering a historical perspective, the author reviews laboratory experiments before proceeding to clinical situations. Of special interest are sections on the placebo effect in psychiatry and psychotherapy. A good deal is known about personality and habits of placebo reactors. For example, they are often churchgoers who depend on others and trust in the ability of others to help them.

An excellent chapter on methodological problems in placebo research leads to a final section in which the author draws a comparison between placebo response and Schachter's theory of emotion, which states, "When people are in a state of arousal, they may experience very disparate emo-

tional states or no emotional states at all, depending on the concurrent cognitions" (2). Jospe suggests that, according to such a view, the placebo effect can be interpreted as having three components: physiological arousal, emotionally relevant cognitive or situational factors, and a perceived causal link between these factors.

He concludes with a review of recent work in attribution research that has become an important part of social psychology. Jospe believes that further understanding of the placebo can come from conceiving factors in relation to attribution, from which a network of testable hypotheses could be produced.

Much work remains to be done in this area, but the present volume is a valuable contribution and I hope that the author will publish updating editions from time to time as knowledge advances.

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The Annual of Psychoanalysis, Vol. V, *edited by the Chicago Institute for Psychoanalysis. New York, N.Y., International Universities Press, 1977, 436 pp., $17.50.*

This is the fifth volume of what might be called the *Fortune* magazine of psychoanalysis. It maintains its usual high caliber of articles. In the first section, Psychoanalytic History, George Pollock's very interesting "The Chicago Institute for Psychoanalysis from 1932 to the Present" includes the mention of the leading analysts of that day and of both the Berlin and Viennese Institutes. W.W. Meissner's "The Wolf Man and the Paranoid Process" is a carefully detailed analysis but so technically written as to be easily read only by those analysts versed in metapsychological thought processes. For example, Meissner states,

> One stratum of the introjective organization thus derived can be described in terms of the correlative pairing of victim-introject and aggressor-introject. Both of these elements could be identified in the father's introjective organization, but were undoubtedly made more complex and complemented by the integration of introjective components derived from the Wolf Man's mother.

In section two, Clinical Theory, Sklansky summarizes his "The Alchemy of Love" as follows: "The two major forms of libidinal experience, erotic and narcissistic, are confluent and synergistic throughout development, and are transmuted from coarse separate strands into the finer coalescence of young adult love." Arnold Goldberg's "Some Countertransference Phenomena in the Analysis of Perversions" stresses narcissistic elements, and Ira Miller's "On Sadness" continues this occupation with narcissistic elements. Pollock summarizes succinctly the history of Alexander's psychosomatic specificity concept as it developed from specificity of a disease to specificity of a human being. Selma Fraiberg brings to psychoanalytic studies of ego development rich observations on children with congenital sensory and motor defects, supplemented by Freedman's "Studies in Sensory Deprivation" in children. Donald Silver finds that contact lenses have unconscious significance for separation and defloration and many of the anxieties of the adolescent female.

In section three, Developmental Psychology, Essman's excellent and clearly written "Developmental Psychology and Explanatory Theory in Psychoanalysis" shows how Freudian and Piagetian psychologies complement each other, thus advancing the unification of psychology.

Section four, Clinical Psychoanalysis, opens with an illuminating "Preliminary Report of a Case of Consummated Mother-Son Incest" by M. Margolis. It is followed by "Comments on the General Functioning of the Analyst in the Psychoanalytic Situation" by Lichtenberg and Slap, a timely discussion of the suggestion that the analyst be humane and natural instead of a blank screen.

Section five is composed of Gedo's "Essay on Psychoanalytic Education." Gedo looks beyond mere quantitative improvements in the existing psychoanalytic institutes to transforming them into mini-universities where interested participants at all levels of experience can join for corrective learning.

Section six, Psychoanalysis and Psychotherapy, consists of Paul and Anna Ornstein's reflections and predictions "On the Continuing Evolution of Psychoanalytic Psychotherapy," with an excellent discussion by Zaleznik and Schwaber. This article falsely sees psychoanalytically oriented psychotherapy as not recognizing the process nature of psychotherapy, thereby being technique-oriented without guidelines for its intervention, but the authors are trying to develop a systematic theory of the psychoanalytic therapies.

In section seven, A Tribute, it is good to see "Reflections on the Occasion of Jean Piaget's Eightieth Birthday," by Heinz Kohut because it is a step in the unification of the best in all the psychologies.

Section eight, Application, contains Weisblatt's "The Creativity of Sylvia Plath's *Ariel* Period: Toward Origins and Meanings." The author sees as most important "the ultimately failing attempt at mastery of the regressively resonated traumatic state." W.W. Meissner follows with "A Case in Point," which illustrates the "paranoid process" in the case of Golyadkin, hero of Dostoevsky's *The Double*.

Happily, this year the quotations and case material are in normal-sized rather than overly small print. This avoids the previously irritating interruptions of the reader's concentration and adds to the enjoyment of this varied fare.

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That Which Is: An Inquiry Into the Nature of Energy, Ethics, and Mental Health, *by John O. Beahrs, M.D. Palo Alto, Calif., Science and Behavior Books, 1977, 260 pp., $11.95.*

In the complexities and pressures of twentieth century living, while mankind is attempting to deal with a technology that threatens to overwhelm us all, the philosophical and religious concepts of past centuries are less satisfying to many. Ours is an age of skepticism on the one hand and a need for a philosophical home base that stands up under the penetrating scrutiny of today's scientific knowledge on the other.

The author, a young psychiatrist who practices in the northwest, addresses this problem in *That Which Is*. He believes that the philosophical answers he has formulated have practical applications for coping with much of the emotional and mental malaise seen by today's practicing psychiatrist.

Beahrs points to the philosophical dualism postulated by Descartes as the basic problem in reconciling dogmatic Western religious philosophy with scientific thinking. The error lies in viewing the material and spiritual aspects of existence as separate entities. Beahrs describes the thinking of Descartes as ontological dualism: "a belief in the existence of two entirely separate but equally real substances: 'Material,' constituting the material universe and our bodies, and 'Spiritual,' constituting God and our minds." The Christian church, according to Beahrs, is the bastion of ontological dualism in that it holds to the concept that God and man's spiritual nature are separate and apart from the material world—from the clay from which man was fashioned.

Beahrs declares philosophical war on religions based on ontological dualism. He terms these "religions of fantasy." In his quest for the true nature of reality, he prefers to turn to the monistic philosophy of Benedict de Spinoza and to bring up to date that seventeenth-century philosopher's pantheistic concepts in the light of modern physics and psychology. This book is dedicated to Spinoza.

The book is composed of four essays. The first discusses the irreconcilable contradictions of ontological dualism (viewing God, mind, thought, and moral concepts as spiritual and viewing objective reality as material). Through logic and theories of modern physics, the author presents his view of the true nature of existence, the basic principle of which is that all that has been experienced by human beings throughout history (physical matter, energy, life, feelings, intellect, divinity, and whatever else) is constituted of the same "stuff" or "substance" that is eternal, unbounded, and unchangeable in its basic nature. This substance is not "crude, inert, lifeless matter" as commonly conceived but is identical to pure energy as the term is used in physics.

Beahrs offers convincing scientific arguments that the molecular structures of all things in the universe have an ultimate common denominator—energy. Higher forms of life and brain function are simply more complex organizations of this omnipresent essence. Then, in a series of brilliantly expounded propositions, he makes a philosophical extension of his concept of energy essence to argue that energy is spiritual in the final analysis and that "spirituality is the essence of energy as it exists independently of an external observer." He terms his theory of energy "spiritual monism," summing it up by declaring, "All existence is constituted by one 'stuff'—energy—whose true essence is spiritual. It is regarded as 'material' only when viewed by an external observer."

The author proposes an interesting division of the polarities of consciousness, terming the conscious, reasoning level "Self 1," which is related to the left hemisphere of the brain, and the unconscious level "Self 2," related to the right hemisphere of the brain. Self 1 is the executive; it is voluntary, gives commands, and is linguistic, denotative, and digital. Self 2 is involuntary, spontaneous, symbolic, and analogic. Self 1 represents advanced or higher development. Self 2 represents primitive or lower development.

The author then goes a step farther and suggests a Self 3, a more primitive and basic level of human brain function, even more primitive than the Freudian Id. In a theological sense, this deep lower level of being is the system's support and power base, a level of biological function and a link with God and reality. Prayer, in the author's view, is perhaps a form of self-hypnosis that has a real and scientific application. In praying, one voluntarily surrenders his or her primary Self 1, surrenders himself or herself to "God's will," and thus reaches the deeper levels of his or her personality: "What is our link with God, with ultimate reality—That Which is—but the very deepest, most basic roots of our personality, first Self 2, then Self 3." This idea is not entirely unlike the thought of William James (1), who suggested that if man experiences God, it may be through subconscious levels of the mind.

In his personal preface to the book Beahrs recalls his own early adjustment difficulties in reconciling himself to the dualism of dogmatic religions. This personal factor no doubt strongly influenced both the writing and the direction of this book.

REFERENCE

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GEORGE A. CONSTANT, M.D.
Victoria, Tex.

Review of Inhalants: Euphoria to Dysfunction. NIDA Research Monograph 15, *edited by Charles Wm. Sharp, Ph.D., and Mary Lee Brehm, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1977, 345 pp., no price listed (paper).*

This monograph on inhalants and inhalant abuse, the outgrowth of a NIDA-sponsored conference held in June 1977, aims at providing "a thorough review of the literature and critical assessment of the state of our knowledge . . . and an important post-1970 selected bibliography on the topic." Its chapters outline the epidemiology of inhalants, psychological factors predisposing to use, mental status and personality studies of users, medical and neurological evaluation of inhalant abusers, and preclinical pharmacological, toxicological, and behavioral studies. In addition to the bibliography noted above, each of the chapters has its own excellent list of references. Although an excellent summary of current knowledge, the monograph highlights our lack of understanding. Unfortunately, it serves more to outline our confusion and to uncover poor methodology and ignorance than to illuminate.

The phenomenon of glue-sniffing commonly involves an unknown number of individuals from unknown populations who inhale a wide variety of volatile substances, often in unknown combinations. Exposure may in time (in some known cases, does) produce a series of undefined or ill-defined toxicities in addition to the subjective experience defined by the user as a high. The substances known to be used in this way, singly or in commercially available combinations (often with changing formulas), include aliphatic and aromatic hydrocarbons, halgenated hydrocarbons, freons, aliphatic nitrites, ketones, esters, alcohols, glycols, ethers, and gases. Many of these are easily available, conveniently packaged, and cheap (e.g., varnish, gasoline, and nail polish remover). Perhaps for these reasons such agents are often the first nonmedically used psychoactive agents, sometimes antedating alcohol and tobacco use. They appear to be used early and may serve as an introduction to other drug use. Usage is reported to decrease with age.

The youth of the prototype user, who is usually pre-

adolescent or in early adolescence, combined with possible subtle neurological deficits, may result in markedly dysfunctional adults. The search for obliteration of reality in place of adaptation often precludes maturation. Unfortunately, the data available do not permit firm conclusions. Despite this, a variety of controls have been suggested and in some areas instituted. These recommendations include the addition of noxious or unpleasant agents to substances of possible abuse and/or forbidding their sale to minors. The first may merely add to toxicity or drive users to more questionable compounds, and the second is unrealistic when one considers the number of preparations, their wide distribution, and their many uses in the home and commercially.

Remedies should be applied only when it is clear that there is a significant problem and when it is clear that the remedy is less dangerous than the disease. It is important that we not contribute to the harm people do to themselves. We need careful study, clinical as well as preclinical, behavioral as well as biological, to uncover the complex motivations of users and the physiology and pathophysiology of use. Intelligent intervention can only follow understanding. This volume clearly states the problem as well as charting the needed courses.

WILLIAM A. FROSCH, M.D.
New York, N.Y.

Transactional Analysis After Eric Berne: Teachings and Practices of Three TA Schools, *edited by Graham Barnes. New York, N.Y., Harper's College Press (Harper & Row), 1977, 531 pp., $15.95.*

The psychotherapeutic strategies known as transactional analysis (TA) began about 1955 when Eric Berne introduced the concepts of Parent, Adult, and Child ego states and enlarged on these ideas with transactions and games theories that were related to the communications theories of cybernetics (1). Both TA and Gestalt therapy, which overlap in theory and in practitioners, have roots in group therapy and often emphasize rapid change in symptomatic behavior. The International Transactional Analysis Association now has almost 10,000 members in 35 countries, according to the preface of this book.

Transactional Analysis After Eric Berne is a collection of 22 articles by leading TA practitioners and theorists. It is a very good sampling of the TA universe, showing how the concepts of the "classical school" (Parent, Adult, and Child life scripts) differ in emphasis from those of the "redecision school" (Robert and Mary Goulding's role playing and redeciding of self-defeating rules learned in childhood) and from those of the "cathexis school" (Jacqui Schiff's Ferenczi-like treatment, the reparenting of schizophrenic patients in a residential setting).

Therapists usually begin by teaching TA concepts to patients (chapter 22, "What Transactional Analysts Want Their Clients to Know" by Woollams, Brown, and Huige). The terminology of TA is apparently chosen to be meaningful to patients (e.g., "games," "rackets" and "racketeering," "sleepy, scary, and spunky forces," and "strokes"), Robert Goulding describes rapid treatment of phobias. Robert Drye describes "The Best of Two Worlds: A Psychoanalyst Looks at TA."

A number of the leaders in TA are psychiatrists and psychoanalysts, but there is little emphasis on precise diagnosis and few significant elements of an empirical or medical model in this book. Its major purpose is to give an overview of the complexity of theory and practice that has developed in the last decade and to be used as a textbook in teaching. It succeeds very well in these purposes. However, I can find no outcome data in this book other than case descriptions by an individual therapist and anecdotes, and there is no systematic attempt to evaluate the effectiveness of what I found many therapeutic strategies that make good sense. However, This is a stimulating book of ideas for the psychotherapist.

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ROSS F. GRUMET, M.D.
Atlanta, Ga.

Beyond the Male Myth: What Women Want to Know About Men's Sexuality. A Nationwide Survey, *by Anthony Pietropinto, M.D., and Jacqueline Simenauer. New York, N.Y., Times Books, 1978, 417 pp., $12.50.*

Not since the 1948 Kinsey report has there been such a revealing look at the sexual and emotional preoccupations of today's American male. *Beyond the Male Myth* is mandatory reading for women of all ages who are concerned about their relationships with men. The authors, one a psychiatrist and the other a psychiatric editor and writer, asked women what they wanted to know about men because their sexual response is affected by the many myths and changing attitudes about male sexuality.

The statistics come out of a random sample of more than 4,000 men carefully controlled for age, education, income, occupation, race, and marital status. Age, race, education, and occupation were found to produce substantial differences in the findings. For example, 70% of the men under age 40 said they would cheat on their women, compared with 36% of the men over 55 years old. Of the younger group, 31% said they liked oral sex best compared with 16% of men over 40. Thirty percent of the high school graduates reported that sex was their greatest pleasure, but 17% of the college graduates reported this attitude. The biggest discrepancy on the basis of race came with attitudes relating to the overall importance of sex: 64% of the white respondents and 41% of the black respondents reported that sex was "not the most but a very important pleasure." Sixty-five percent of the college graduates reported that sex was very important, compared with 48% of those with less than a high school education. The choice of marriage with outside sexual activity climbed steadily in popularity with earning power: 12% of the men earning less than $10,000 would choose this option compared with 27% of those earning $27,000 or more. A greater percent of professional men would choose this option.

The authors' survey also destroys a number of myths. For example, 87% of the married men reported that they still believed that marriage was their best option; 2 out of 3 men expressed positive views about women's liberation, men reported that they were not preoccupied with youthful sexual partners; 4 out of 5 men said they made a conscious effort to delay orgasm; and 98% said they felt that it was important for a woman to have an orgasm and that love, companionship, and home life were more important than sex.

By comparing studies done by Kinsey, Hunt, Kaplan

Hite, and the Rubins, the authors note the changing attitudes of men toward women and their own sexuality. They describe such notables as Cleopatra, Hera, Don Juan, Farah Fawcett-Majors, Lucille Ball, and Mary Magdalene; such movies as *A Clockwork Orange, Catch-22,* and *Divorce, Italian Style;* and such plays as *Hair* and *The Divine Comedy* to break up the collage of figures that may otherwise confuse the reader. Pietropinto's interpretations, opinions, and case histories add a reflective touch that stimulates the reader's own thoughts.

On completion of this book, I find I have three main questions. Why is it that women complain of male insensitivity in the light of the fact that men see themselves as being highly sensitive? If marriage is the most desired option of men, why do they jeopardize it by being unfaithful? Finally, what is it about the nature of the marital relationship that it is so abused yet so sought after by men and women?

MARY ANN BARTUSIS, M.D.
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Clinical and Experimental Hypnosis in Medicine, Dentistry, and Psychology, 2nd ed., *by William S. Kroger, M.D. Philadelphia, Pa., J.B. Lippincott Co., 1977, 391 pp., $21.00.*

The author has written a big book in which he attempts to cover the enormous field of clinical and experimental hypnosis in medicine, dentistry, and psychology. He is too ambitious in this venture. However, there are a few advantages for the reader.

The volume opens with basic themes that appear in most volumes on this subject. There are chapters covering the history of hypnosis, hypnotic phenomena, suggestion and suggestibility, theories, induction procedures, and misconceptions about hypnosis. There are data also on autohypnosis, group hypnosis, precautions and dangers, and dehypnotization. The author deals with additional subjects, not all of which are to be found in other books on hypnosis. He discusses antisocial aspects of hypnosis, legal and ethical issues, including criminal investigation, and religious concerns. He has material on magic and faith healing, placebo effects, biofeedback, meditation, semantics, learning theory, and behavior modification.

He then moves on to a vast territory, as a result of which one legitimately wonders how much ground an author is in a position to manage with his own measure of experience. Kroger deals with neurophysiological and psychophysiological mechanisms and presents material from an array of medical specialties and subspecialties. These encompass internal medicine, surgery and anesthesiology, obstetrics, gynecology, dermatology, physical rehabilitation, ophthalmology, otolaryngology and rhinology, genito-urinary problems, oncology, pediatrics, orthopedics, and dentistry (this contribution was prepared by a dentist colleague). He focuses, in addition, on acupuncture and a variety of miscellaneous problems and offers a comparative evaluation and criticism of hypnotherapy and psychoanalysis.

It was possible during the nineteenth century for one determined individual to be concerned with and to attempt to write about the entire field of medical and scientific hypnosis. There are several well-known classics of such literature. Both authors could still attempt this in the early decades of the twentieth century. Because of advances in medical specialization and growth of the field of psychology, however, no one person in the last few decades could possibly master all of these areas. It is for this reason that books have been written by several authors and edited with specialists in various areas contributing chapters based on their personal experience and knowledge.

However, Kroger's book can be helpful, especially to novices involved in clinical and experimental hypnosis who wish to search for some preliminary information in the areas of concern to them. They are in a position to seek out references supplied at the end of chapters for guidance toward additional reading in the works, books, and articles by investigators writing about their personal experience. In this book, some investigators are referred to by name as well as reference number in the text. There are numerous instances in which only reference numbers are supplied so that the reader does not know immediately which contributors are responsible for statements of fact, allegations, or opinions referred to in the text itself; it is necessary to turn to the reference list at the end of the appropriate chapter for such information. Consistent with this observation, one notes that the book has a subject index but not an author index.

Despite the shortcomings mentioned, the merits of the book permit recommending it to readers as an initial reference for preliminary information and as a guide to other writings for data based on direct experience of specialists in the many areas of clinical and experimental hypnosis.

JEROME M. SCHNECK, M.D.
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Should the Children Know? Encounters with Death in the Lives of Children, *by Marguerita Rudolph. New York, N.Y., Schocken Books, 1978, 110 pp., $8.95.*

Unfortunately, the title of this book promises more than can be found in its text. Initially I had a fantasy of reading an exhaustive clinical disquisition on the multifaceted aspects of death as it relates to children. I was unprepared, although not necessarily disappointed, to read a more limited, personalized, and anecdotal presentation on the subject.

The author provides "case" material gathered from her work as a teacher and director of preschool and day-care programs and from her personal experiences as a mother and grandmother. She displays the wisdom and sensitivity of a knowledgeable paraprofessional and offers some perceptive and absorbing glimpses of the older preschool child's reactions to the death of a classmate, a pet animal, or an aged great-grandmother. The children she describes are enmeshed in what Fraiberg (1) called the magic years. This is the time when fantasy, dreams, enchantment, and curiosity blend with and complement the dawning of an intellectual discrimination between magical thought and real experience. Thus, for the child, death is both a separation from a loved one and a nonpermanent but real state like sleep. This is reflected in the children's questions, which the author uses as chapter titles: "Does the World Stop? Who Deaded Him?" and "Can You Still Have a Birthday When You Die?"

There is an answer to the book's provocative title, *Should the Children Know?* By the process of psychologically working through the untimely death of a preschooler named Rachel, Ms. Rudulph answers affirmatively. She believes a child has the right to feel the sadness and grief inherent in working through feelings about death without the interference of a "protecting" adult who would say, "Tell them Rachel moved."

Aside from implying that Dr. Elisabeth Kübler-Ross is a

psychologist and that "in the Jewish tradition, children are named for dead relatives" (this is customary among Ashkenazim, but Sephardic Jews often share a child's name with that of a living relative [2]), this book is sensibly written, geared primarily for the lay reader or teacher, and includes a useful annotated bibliography for additional exploration of subjects relating to death-education for children.

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MICHAEL F. HEIMAN, M.D.
Stockton, Calif.

The Meaning of Anxiety, revised ed., *by Rollo May. New York, N.Y., W.W. Norton & Co., 1977, 425 pp., $12.95.*

Dr. May is a training and supervising analyst at the William Alanson White Institute. He wrote the first version of this book in 1950. In this revised edition, he adds two new chapters on the management of anxiety.

The general aim of this work is to summarize the various theories of anxiety, which Dr. May describes as "a pervasive and profound phenomenon of the time we live in," appropriately called by Auden the Age of Anxiety. The interpretations of Spinoza, Pascal, Kierkegaard, Freud, Rank, Adler, Jung, Horney, Sullivan, and others are examined in an attempt to synthesize the various viewpoints into a comprehensive theory. To summarize such complex theories is an ambitious project indeed, and the reader is left with the uneasy impression that some synopses might suffer in objectivity and in depth. For example, one is struck by the importance that May gives to Freud's early formulations of anxiety, the ones that are certainly of less significance to the psychotherapist.

Although a full chapter is devoted to the biological interpretation of anxiety, no useful reference is made to its pharmacological management, as if the therapeutic means available to the clinician had remained at the stage they were in 25 years ago. In a brief footnote (page 262) the author describes how a young man he treated in psychotherapy ended up being lobotomized, and continues, "If drugs had been available when he was in the hospital, this would have been one time they would have been useful to tide him over that episode." This is hardly a comprehensive view of what drugs have to offer. It seems as if the author ignores or underestimates the important role played by anxiolytic and antipsychotic drugs in controlling the symptoms of anxiety found in various psychotic and neurotic conditions. The exclusively psychotherapeutic approach he suggests will be of limited value to the practicing psychiatrist. He also makes no mention of the use of relaxation and of deconditioning in the treatment of anxiety.

Dr. May seems convinced that anxiety is essential to the human condition in that it creates a tension necessary to preserve human existence and creativity. As true as this hypothesis might be from a philosophical standpoint, the fact remains that physicians have more to offer therapeutically to their patients than what is presented in the last two chapters of this book. The weakness of the new section might be due to the fact that the author's diagnostic distinction between psychologically crippling anxiety states and "normal anxiety" seems very vague.

Richard Chessick (1) stressed that "the psychotherapist, in order to become relatively wise and mature, must understand something about history, philosophy, ethics, philosophy of science, psychology, anthropology and sociology." Although *The Meaning of Anxiety* does not provide new theoretical or therapeutic insights, it can be recommended for humanistically inclined readers who do not have the time to go back to original sources.

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PHILIPPE E. CHAIX, M.D.
Washington, D.C.

Psychotherapy and Its Financial Feasibility Within the National Health Care System. Publication 100, *by the Group for the Advancement of Psychiatry Committee on Therapy. New York, N.Y., GAP, 1978, 45 pp., $2.50 (paper).*

This GAP publication is a well-written and concise statement of the case for including psychotherapy under national health insurance (NHI). It is written for the informed layman and for key policy planners. The writers are to be commended for their lack of jargon as well as their direct style.

This pamphlet presents its strongest argument for the value of psychotherapy from a humanitarian perspective. The authors articulate well the human pain associated with mental illness, especially neurotic illness, and the need for care of suffering human beings. The clinical examples presented are well drawn and persuasive. However, the arguments presented in terms of more hard-headed financial cost benefit issues are simplistic and unconvincing. This is especially true in the case for long-term intensive psychotherapy as contrasted to short-term psychotherapy.

Most policy planners and even some budget analysts concede that short-term psychotherapy is cost effective and are willing to consider the cost offsets in general medical utilization with short-term psychotherapy. The higher cost inherent in high intensity treatments such as psychoanalysis is another issue altogether. In addition to the higher cost there is the issue of the appropriateness of such care and the socioeconomic class and educational biases of patients who receive such care. The criteria and indications for long-term intensive psychotherapy are not well understood at the present time. This pamphlet implies that we have already developed diagnostic and prognostic criteria that could clearly delineate the most effective treatment for a given individual. Since these treatments vary in cost by as much as ten times, this is not an academic exercise.

Other basic problems in the case for coverage of long-term intensive psychotherapy involve the extent to which the treatment can be conceptualized as "medicine." In physical medicine the diseased organ and to a lesser extent the diseased person are the prime foci of intervention and insurance coverage. The long-term goals of intensive psychotherapy and psychoanalysis imply considerable efforts at re-education and as such do not conform well to the medical model.

In addition, the credentials of various professionals to perform the treatment and the lack of clear role specialization

create other economic problems as more nonpsychiatrist professionals claim expertise in performing this treatment. As these professionals are reimbursed by health insurance carriers as independent practitioners the utilization and costs go up. The issues of who can perform and who should be paid for this treatment are not addressed at all in this publication.

From the perspective of long-term intensive psychotherapy, it is essential that the West German studies cited in this publication be replicated in this country and that there be increased understanding on the part of insurance companies and policy planners as to the value of long-term intensive psychotherapy. With this understanding will come an increased willingness to finance such treatment.

It would have also been better to elaborate on the key equity issue of the appropriateness of long-term intensive psychotherapy for patients from lower socioeconomic groups. At present, long-term intensive psychotherapy is utilized almost exclusively by college-educated white people from the middle and upper classes. Would NHI coverage of long-term intensive psychotherapy be a subsidy of the rich by the poor? The equity issue of the redistribution of health resources to the poor and minority groups in the United States through national health insurance precludes consideration of this form of treatment unless it is considered utilizable by those for whom such care has been traditionally denied. We need more data on this issue.

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Rockville, Md.

Emergency Psychotherapy and Brief Psychotherapy, 2nd ed., *by Leopold Bellak, M.D., and Leonard Small, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 274 pp., $18.50.*

This book, which was first published about 10 years ago, has been revised and expanded because of the growing interest in a subject whose importance has finally been recognized. It is composed of three parts. The first deals with basic principles and methods, the second lists several clinical syndromes illustrated with case examples, and the final one contains a discussion of a multiple-level study of brief psychotherapy in a clinic setting. Three case histories conclude the volume.

Valuable information is offered to the reader about various definitions, theories, and principles of brief psychotherapy, and a five-to-six-session therapy is described briefly. The discussions of diagnostic formulation, history taking, therapeutic contract and alliance, transference, interpretation, and termination issues are quite helpful.

One difficulty with the book, as far as I am concerned, has to do with the position taken by the authors in reference to planning psychotherapy "within the framework of what the patient is willing to engage in." This surrender to consumerism undermines the basic conclusion shared by several investigators in this field, that the patient must fit the treatment chosen for him by an expert. This conviction has resulted from extensive research involving a specification of criteria for selection of appropriate candidates, a description of specific technical interventions, and, most importantly, a systematic follow-up study of outcome. As a consequence of their investigations several distinct and successful kinds of brief psychotherapy have been developed. In contrast to this trend, the somewhat global approach to this subject advocated by the authors detracts from the potential value of their book to a large number of readers who look on brief psychotherapy with increasing interest and fascination.

P.E. SIFNEOS, M.D.
Boston, Mass.

Therapist Disclosure: The Use of Self in Psychotherapy, *by Myron F. Weiner, M.D. Boston, Mass., Butterworths, 1978, 172 pp., $11.95.*

This book is courageous, honest, and full of homely advice. It concerns an important area of psychotherapeutic technique about which too little has been written. The author strikes bravely against the hegemony of the emotionally remote, silent therapist.

One wants to like this book, but it is consistently disappointing. To begin with, the clinical examples are cursory, and in nearly half of them the reader is hard-put even to locate what it is the therapist has disclosed. The balance of examples contain mainly simple statements of positive and negative regard. An early anecdote illustrates several of my difficulties:

> I had worked intermittently with Mr. D., a 25-year-old man, for five years. During one session, in response to his request, I sat with him on my couch, held his hand, and allowed him to literally cry on my shoulder. Previously, he had worked through some homosexual fantasies about me and was beginning to separate me, in his mind, from his deceased mother, whom he had seen as cold, ungiving, and controlling. Several weeks later, when the distortion arose again, I cited this time of physical intimacy to correct his distorted perception of me. (p. 23)

The reader is left to ponder not only what the therapist has disclosed, but also whether the author is aware of the limitations he has imposed on the uses of the transference in this treatment effort of five years' duration. There are indications for such choices, but his failure to discuss them is not reassuring. Initially one is less disturbed by the author's assumption that the technical and theoretical significance of self-disclosing interventions is self-evident than by the level of discussion when it comes. It is disappointing to be brought in the final pages of the book no farther than the reminder that if the therapist elects to have physical contact with his patient (nonsexual touching, we are assured), he or she must keep in mind that such behavior might have magical and even sexual meanings to the patient. Thus, the larger failure of this book is that it does not deal substantively with the technical and theoretical issues of its topic.

In the jungle of jargon that characterizes so much of contemporary writing on psychotherapy the topic of transference management is a beacon in the night. Most varieties of therapy are usefully clarified in terms of how the therapeutic relationship is handled. We are grateful, for example, to Gill and Muslin (1) for their critique of the silent analyst and their thoughtful injunction to greater therapist activity within the framework of psychoanalytic theory and technique. However, outside of psychoanalytic circles the fall of the bastion of the silent therapist has too often become an invitation to chatsy informality. If we elect as therapists to become more active and more open, what are the rules of the game? More specifically, how is therapy shaped and to what extent con-

stricted by interventions that limit transference regression? Spontaneity of the therapist surely has a place in psychotherapy, but when it becomes uncoupled from the careful exercise of professional discipline, and in the absence of an organizing theory of psychotherapy, the results can be disquieting.

The therapist's use of self in conducting psychotherapy is a critically important issue in the advance of the science of our craft. We are, unfortunately, more indebted to Dr. Weiner for the attention his title calls to the topic than for the text of his volume.

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JUSTIN SIMON, M.D.
Berkeley, Calif.

Renewal in Psychiatry: A Critical Rational Perspective, *edited by Theo C. Manschreck and Arthur M. Kleinman. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 326 pp., $23.50.*

This is a very interesting book, the product of a monthly seminar that examined the role of critical reasoning in psychiatric theory, research, practice, and education. It includes 11 chapters divided into three parts. The first part concerns Issues That Require Clarification and Reconceptualization in the Fields of Psychiatry, the second concerns Issues That Determine the Frontiers and Limits of the Fields of Psychiatry, and the third concerns Issues That Provide Novel and Significant Perspectives in the Fields of Psychiatry.

Part one discusses acute psychosis, social and cultural aspects of psychopathology, and drug dependence. Each chapter is tightly written and well documented and contains an excellent bibliography. With *DSM-III* just around the corner, the authors' discussion of making psychiatry more scientific, measurable, and clear is very timely. I found the drug dependence chapter most interesting. It includes an explanation of Jasinski's structuring of 40 items from the MMPI into three scales: MBG for euphorogenic effects of a drug, the LSD for psychotomimetic effects, and the PCAG for sedative effects. Using this model, psychoactive concomitants of pharmacological agents can be clearly documented and measured, thus moving the subjective sensations into objective data.

Part two contains my favorite chapter, which discusses temporal lobe epilepsy and compares different brain-damaged areas in epilepsy to their associated behavior and diagnostic entities in psychiatry. Since psychiatry seems to be moving more and more toward the organic and away from the functional, this chapter lends credence to the former.

Part three covers patients' expressions about psychiatric hospitalization and the influence of fathers on children; it also includes a chapter titled "Middlehomes: An Introduction to Residence and Developmental Needs," and one titled "A Life History of the Civil Rights Movement." The chapter making a strong case for the importance of fathers for healthy child development is detailed and well documented.

Overall, this book is excellent in places, as mentioned above, but has some chapters that do not really seem to fit. However, I think most psychiatric house officers and practicing psychiatrists will find enough chapters of interest to warrant its perusal. This book has one of the most thorough group of chapter bibliographies I have ever seen.

JAMES A. DAVIS, M.D.
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Spatial Organization of Cerebral Processes, *by M. N. Livanov; translated by the Israel Program for Scientific Translations. Jerusalem, Israel, IPST (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 211 pp., $21.50.*

The Cyrillic alphabet and the complexities of grammar combine to make Russian publications inaccessible to most Western readers. This volume was originally published in Moscow in 1973. Essentially, the work concerns spatiotemporal mapping of cerebral potentials in animals and man and summarizes work carried out over a number of years in the author's laboratory. Underlying these efforts are the hope that such maps are analyzable and the postulate that such an analysis will yield explanations of electrogenesis, function and behavior. For many years a large number of investigators have pursued this goal with varying degrees of sophistication and varying, often uncorrelated, degrees of success. High-speed digital computers permit statistical processing of large amounts of data yielding functions such as power-density spectra and auto- and cross-correlations. But such instruments are two-edged swords. Ingenuous experimenters with flawed experimental designs or fallacious postulates can find themselves inundated by a flood of calculations that yield little more than trivial conclusions or epiphenomena.

Readers should approach this volume with some understanding of the scientific climate in the Soviet Union, where neurosciences move within the flume of Pavlovian physiology. In the study of "higher nervous activity" (behavior) conditioned reflexes occupy a central position. As a result, Russian scientists have expended great efforts in attempting to define the electrophysiological and morphological changes as a conditioned reflex emerges.

The volume begins with a consideration of "conditions which determine the spread of excitation in the cerebral cortex," on the assumption that this will clarify "the processes which underlie such major cortical functions as the establishment of a conditioned reflex." This recondite chapter considers the genesis of the activity in rabbit cortex and the significance of widespread synchronization. If the translation is accurate, the research methods are, at least, unusual: "Using a scalped rabbit, we determined the [electric] potentials for the hind limb by direct [sic] stimulation through the bone." Statistical analysis is casual or nonexistent: "Though not particularly large, the difference is based on a sufficient body of material and is therefore significant."

The second chapter attempts to study the synchronization of electrical activity in the cortex during the formation of "time link" and deal with an always stormy problem for Pavlovian physiology, the role of subcortical structures. The final three chapters of the book deal with human activities. The conclusions prove curiously elusive. It may be that the concepts have suffered in translation, but a sample translation may prove illuminating:

> Monitored experiments on man have to date fur-

nished too few data to permit any definitive conclusions. It appears, however, that the principles derived from the role of synchronization in the transmission of excitation in the cerebral cortex of rabbits apply at least in part to the human brain. (p. 109)

These seem to be minor conclusions, particularly since the author's research methods were supposed to rectify the situation in which "the meager achievements in this field have disappointed scientists and even aroused some doubts as to the value of electrophysiology as a tool for the analysis of cortical functions." Anyone familiar with contemporary neurophysiology will recognize the error of even this premise. For example, Mountcastle has shown that the psychophysical identification and discrimination functions for flutter-vibration are identical in man and monkey and that detection functions can be defined in terms of the increment in activity in a particular set of post-central cortical neurons, whereas discrimination functions can be defined in terms of the cyclic entrainment of activity in such cortical neurons. In visual physiology the work of Hubel and Wiesel and that of Blakemore and others has clarified the cortical microcircuitry that underlies feature-extraction and stimulus generalization and has shown the existence of such entities as cortical columns, which constitute the morphological correlates of these neuronal microcircuits.

Are Soviet scientists unaware of or indifferent to contemporary concepts? A review of the bibliography discloses virtually no citation of non-Russian publications for the past 10 years. From these observations one may conclude that constraints on directions of research, lack of access to contemporary Western neuroscience, and linguistic isolation have proved serious impediments in the development of neurosciences in the Soviet Union.

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Your Body Biofeedback at Its Best, *by Beata Jencks, Ph.D. Chicago, Ill., Nelson-Hall, 1977, 262 pp., $11.95; $6.95 (paper).*

Bringing together under one cover many terms, techniques, and related concepts is always helpful, but providing too great a variety of choices can be overwhelming. *Your Body Biofeedback at Its Best* alternates precariously between these two poles. The concept of using one's own mind/body processes to aid in the establishment of a more ideal psychophysiological state is a sound one. The intent of this book is to provide a variety of lesson plans to choose from, emphasizing the author's point that one does not need to use electronic biofeedback equipment.

As a long-time user of a variety of biofeedback instruments or tools, I find myself in disagreement with the idea that the mind does not need electronic equipment to get in touch with the body. My experience has been that even with electronic monitoring many minds still cannot make a meaningful contact with the body.

This is an age of expanding development of sophisticated electronic equipment, devices that explore the body in rewarding, painless ways. Electronic biofeedback equipment development is in its infancy. The art and science of the effective application of present-day electronic biofeedback in clinical medicine is only in the beginning stages of its establishment. For many minds (not all), the only route to the establishment of a meaningful sensory awareness of the body will be through this electronic type of biofeedback.

This book is structured around the positive willpower philosophy of "putting your mind to it" in a disciplined way; "success will follow." Without rewarding input via electronic equipment this system has failed in the past. More discipline and a firmer insistent approach is usually only self-defeating. The value of this book lies in its presentation of nonelectronic approaches to the development of sensory awareness with appropriate caution about altered states of consciousness. The book is a scholarly effort. The author is to be congratulated for pulling so much material relating to body awareness together so effectively.

The psychiatrist learns early in his or her training that effectively externalizing and objectifying thoughts and feelings is the beginning of meaningful insight. The techniques developed to provide this constitute the art of psychotherapy. Electronic biofeedback is to body awareness what insight techniques are to psychotherapeutic awareness. After the awareness comes the reliance on one's self for the continued therapeutic effect started in the therapy sessions. It is to these processes that Beata Jencks seems to be referring in her book, which provides ways to continue and build on the successes with electronic biofeedback but not ways to begin the process.

The reader looking for definitions, descriptions, self-reliant techniques, and practices to aid to developing inherent body biofeedback will find this book helpful. It seems to spell out many issues not dealt with in so detailed a manner by authors of books on electronic biofeedback. Yogis, Zen masters, and TM practitioners have lived much of this book's contents. We have learned from them, and we can learn from this book. The author's presentation is an Americanization of the practices of Eastern mystics, perhaps more acceptable and understandable to our Western minds. Read it and get it in perspective in your learning of the role of electronic biofeedback in clinical management of psychosomatic disorders.

PHILIP C. ROND, M.D.
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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Co-operation Between the Sexes: Writings on Women, Love and Marriage, Sexuality and Its Disorders, *by Alfred Adler; edited and translated by Heinz L. Ansbacher and Rowena R. Ansbacher. Garden City, N.Y., Anchor Books (Doubleday & Co.), 1978, 455 pp. $3.95 (paper).*

Psychiatric Diagnosis: Exploration of Biological Predictors, *edited by Hagop S. Akiskal, M.D., and William L. Webb, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1978, 490 pp., $25.00.*

The Mind in Sleep: Psychology and Psychophysiology, *edited by Arthur M. Arkin, John S. Antrobus, and Steven J. Ellman. New York, N.Y., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1978, 624 pp., $29.95.*

It's Scary Sometimes, *by Marcella Bacigalupi, Georgio Bini, Claudio Costantini, and Piero Fossati; drawings by the children themselves. New York, N.Y., Human Sciences Press, 1978, unnumbered pages, $6.95.*

Psychopharmacology of Thiothixene, *by Thomas A. Ban, M.D. New York, N.Y., Raven Press, 1978, 236 pp., $12.50.*

Psychiatry Observed, *by Geoff Baruch and Andrew Treacher. Boston, Mass., Routledge & Kegan Paul, 1978, 254 pp., $11.50 (paper).*

Motivation: Theories and Principles, *by Robert C. Beck. Englewood Cliffs, N.J., Prentice-Hall, 1978, 459 pp., $12.95.*

Neurochemical and Immunologic Components in Schizophrenia, *edited by Daniel Bergsma, M.D., and Allan L. Goldstein, Ph.D. New York, N.Y., Alan R. Liss, 1978, 425 pp., $46.00.*

A Bibliography in Dynamic, Familial and Social Psychiatry, *by John Birtchnell and Sheila Hafter Gray. Colchester, England, Society of Clinical Psychiatrists, 1978, 135 pp., 50 English pence (paper).*

Mechanisms of Nervous Disorder: An Introduction, *by David Bowsher, M.D., Ph.D. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1978, 102 pp., $9.75 (paper).*

Human Growth and Development: Wolfson College Lectures 1978, *edited by Jerome S. Bruner and Alison Garton. New York, N.Y., Clarendon Press (Oxford University Press), 1978, 167 pp., $10.50; $5.50 (paper).*

The Impact of Alcoholism, *by Steven F. Bucky, Ph.D., Richard Boyatzis, Ph.D., Jere Bunn, Barbara Czesnik, M.A., Anthony Del Nuovo, M.A., Gloria Cody, Ph.D., Douglas Harry, LL.B., David Jacobs, Ph.D., Rosalie Jesse, Ph.D., James Massman, M.D., and Adrienne Nesfadd, Ph.D. Center City, Minn., Hazelden, 1978, 120 pp. $3.50 (paper).*

Annual Review of the Schizophrenic Syndrome 1975–1977, *edited by Robert Cancro, M.D. New York, N.Y., Brunner/Mazel, 1978, 636 pp., $27.50.*

Principles and Practice of Child Psychiatry, *by Stella Chess, M.D., and Mahin Hassibi, M.D. New York, N.Y., Plenum Press, 1978, 486 pp., $20.00.*

Basic Principles and Techniques in Short-Term Dynamic Psychotherapy, *edited by Habib Davanloo, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1978, 549 pp., $30.00.*

Neurological Pathophysiology, 2nd ed., *by Sven G. Eriasson, M.D., Ph.D., Arthur L. Prensky, M.D., and William B. Hardin, Jr., M.D. New York, N.Y., Oxford University Press, 1978, 442 pp., $16.95; $9.95 (paper).*

Client Participation in Human Services: The Prometheus Principle, *edited by Constance T. Fischer and Stanley L. Brodsky. New Brunswick, N.J., Transaction Books, 1978, 241 pp., $12.95.*

The Scientific Evaluation of Freud's Theories and Therapy: A Book of Readings, *edited by Seymour Fisher and Roger P. Greenberg. New York, N.Y., Basic Books, 1978, 447 pp., $25.00.*

Primary Prevention of Psychopathology, Vol. II: Environmental Influences, *edited by Donald G. Forgays. Hanover, N.H. University Press of New England, 1978, 249 pp. $15.00.*

Effective Ingredients of Successful Psychotherapy, *by Jerome D. Frank, Rudolf Hoehn-Saric, Stanley D. Imber, Bernard L. Liberman, and Anthony R. Stone. New York, N.Y., Brunner/Mazel, 1978, 212 pp., no price listed.*

Directory of Unpublished Experimental Mental Measures Vol. 2, *by Bert Arthur Goldman, Ed.D., and John Christian Busch, Ed.D. New York, N.Y., Human Sciences Press 1978, 472 pp., no price listed.*

Neuroses: A Comprehensive and Critical View, *by Melvin Gray, M.D. New York, N.Y., Van Nostrand Reinhold Co., 1978, 314 pp., $15.95.*

Edward Jarvis and the Medical World of Nineteenth-Century America, *by Gerald N. Grob. Knoxville, Tenn., University of Tennessee Press, 1978, 287 pp., $14.00.*

Alcohol, Other Drugs and Violent Death, *by Paul W. Haberman and Michael M. Baden, M.D. New York, N.Y., Oxford University Press, 1978, 130 pp., $13.95.*

I've Done So Well—Why Do I Feel So Bad? *by Celia Halas, Ph.D., and Roberta Matteson, Ph.D. New York, N.Y., Macmillan Publishing Co., 1978, 304 pp., $10.95.*

Within Reach: Providing Family Planning Services to Physically Disabled Women, 2nd ed., *by Julia Hale-Harbaugh, Ann Duecy Norman, Jane Bogle, and Susan Shaul; illustrated by Katie Venables. New York, N.Y., Human Sciences Press, 1978, 48 pp., $2.95 (paper).*

Developments in Opiate Research, *edited by Albert Herz. New York, N.Y., Marcel Dekker, 1978, 414 pp., $35.00.*

Projective Assessment: Methods in Clinical Psychology, Vol. 1, *by Robert R. Holt. New York, N.Y., Plenum Press, 1978, 330 pp., $17.50.*

Prediction and Research: Methods in Clinical Psychology, Vol. 2, *by Robert R. Holt. New York, N.Y., Plenum Press, 1978, 324 pp., $17.50.*

Psychiatric Drugs: A Desk Reference, 2nd ed., *by Gilbert Honigfeld and Alfreda Howard. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 236 pp., no price listed (paper).*

Being and Loving, *by Althea Horner. New York, N.Y., Schocken Books, 1978, 142 pp., $9.95.*

Drugs of Abuse: Handbook of Psychopharmacology, Vol. 12, *edited by Leslie L. Iversen, Susan D. Iversen, and Solomon H. Snyder. New York, N.Y., Plenum Press, 1978, 412 pp., $32.50.*

Biology of Mood and Antianxiety Drugs: Handbook of Psychopharmacology, Vol. 13, *edited by Leslie L. Iversen, Susan D. Iversen, and Solomon H. Snyder. New York, N.Y., Plenum Press, 1978, 430 pp., $32.50.*

Inheritance of Creative Intelligence, *by Jon L. Karlsson, Ph.D., M.D. Chicago, Ill., Nelson-Hall, 1978, 200 pp., $14.95; $7.95 (paper).*

Handbook of Sex Therapy, *edited by Joseph LoPiccolo and Leslie LoPiccolo. New York, N.Y., Plenum Press, 1978, 526 pp., $29.50.*

Dimensions of Human Potentiality: The Collected Works of Eugene Pumpian-Mindlin, *edited by Michael T. McGuire, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications), 1978, 333 pp., $15.00.*

Quality of Life in Severely and Profoundly Mentally Retarded People: Research Foundations for Improvement, *edited by C. Edward Meyers. Washington, D.C., American Association on Mental Deficiency, 1978, 384 pp., $14.95.*

Cerebral Logic: Solving the Problem of Mind and Brain, *by Charles W. Needham, M.D., M.Sc. Springfield, Ill., Charles C Thomas, 1978, 205 pp., $12.75.*

Paradox and Counterparadox: A New Model in the Therapy of the Family in Schizophrenic Transaction, *by Mara Selvini Palazzoli, M.D., Luigi Boscolo, M.D., Gianfranco Cecchin, M.D., and Giuliana Prata, M.D.; translated by Elisabeth V. Burt. New York, N.Y., Jason Aronson, 1978, 186 pp., no price listed.*

Self-Starvation: From Individual to Family Therapy in the Treatment of Anorexia Nervosa, *by Mara Selvini Palazzoli; translated by Arnold Pomerans. New York, N.Y., Jason Aronson, 1978, 289 pp., no price listed.*

Les Voies Nouvelles de la Dépression, *edited by P. Pichot. Paris, France, Masson, 1978, 200 pp., no price listed (paper).*

BT: Behavior Therapy. Strategies for Solving Problems of Living, *by Spencer A. Rathus, Ph.D., and Jeffrey S. Nevid, Ph.D. New York, N.Y., Signet (New American Library), 1978, 312 pp., $2.25 (paper).*

Propranolol and Schizophrenia, *edited by Eugene Roberts, Ph.D., and Peter Amacher, Ph.D. New York, N.Y., Alan R. Liss, 1978, 150 pp., $15.00.*

Estudios Sobre el Suicidio, *by Enrique Rojas. Barcelona, Spain, Salvat Editores, 1978, 539 pp., no price listed (paper).*

Violence and Responsibility: The Individual, the Family and Society, *by Robert L. Sadoff, M.D. New York, N.Y., SP Medical & Scientific Books (Spectrum Publications) (Halsted Press, John Wiley & Sons, distributor), 1978, 133 pp., $14.95.*

Toward Intimacy: Family Planning and Sexuality Concerns of Physically Disabled Women, 2nd ed., *by Susan Shaul, Jane Bogle, Julia Hale-Harbaugh, and Ann Duecy Norman; illustrated by Katie Venables. New York, N.Y., Human Sciences Press, 1978, 61 pp., $2.50 (paper).*

The Trauma of Time: A Psychoanalytic Investigation, *by Irvine Schiffer. New York, N.Y., International Universities Press, 1978, 271 pp., $15.00.*

Consciousness and Self-Regulation: Advances in Research and Theory, Vol. 2, *edited by Gary E. Schwartz and David Shapiro. New York, N.Y., Plenum Press, 1978, 428 pp., $22.50.*

Methods of Group Psychotherapy and Encounter: A Tradition of Innovation, *by Jerrold Lee Shapiro. Itasca, Ill., F.E. Peacock, 1978, 354 pp., $12.50.*

Schizophrenia: Science and Practice, *edited by John C. Shershow. Cambridge, Mass., Harvard University Press, 1978, 239 pp., $15.00.*

Mind and Madness in Ancient Greece: The Classical Roots of Modern Psychiatry, *by Bennett Simon, M.D. Ithaca, N.Y., Cornell University Press, 1978, 325 pp., $17.50.*

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OFFICIAL ACTIONS

Report of the Secretary: Summary of Meetings of Board of Trustees and Executive Committee May 1977–May 1978

IT IS MY personal and constitutional privilege to report the actions of the Board of Trustees and its Executive Committee over the past year. These items do not include many matters that have been referred to appropriate components for further study and recommendation. The recommendations and related actions of the Assembly are included with the relevant items.

This report is arranged in alphabetical order according to topic except for the first item concerning the adoption of the consent calendar concept, which permits the Board and Executive Committee to vote en bloc on matters seen to be less controversial and often of an administrative nature.

The actions reflect the varied issues that have been considered by the Board over the year. There have been internal issues, such as continuing education, constitutional change, elections and related matters, fiscal and budget issues, problems of the chronic patient, as well as a need to remodel or relocate our Central Office facilities to meet the needs of our growing membership activities and staff support. The Board has also been involved in responding to external forces, such as legislation (proposed or enacted), establishment of the President's Commission on Mental Health, activities of related organizations, and so forth.

I would remind you that every member of APA is privileged to attend any session of components of the Association except for meetings of the Ethics Committee or when a component is in Executive Session. Recommendations for consideration by the Board or other components are most welcome. The Board (or its Executive Committee acting as its agent) took the following actions.

PROCEDURE

1. To provide more time for policy discussion at meetings, the Executive Committee (Sept. '77) and the Board (Dec. '77) voted to employ the consent calendar concept for use at all of their meetings.

ANNUAL MEETING

1. Authorized the Program Committee to review all scientific programs held at the annual meeting, including those beginning on Saturday and Sunday preceding the meeting that are not sponsored by another scientific organization, before they are included in the program schedule (Bd., Dec. '77).
2. Approved waiving annual meeting registration fees for foreign visitors, beginning in 1979, and changing the Policy and Procedural Manual for the annual meeting to reflect this (Bd., Dec. '77).
3. Endorsed the action of the Reference Committee to authorize staff to proceed with a direct advance registration mailing to the membership promoting the annual meeting and highlighting continuing medical education requirements (Bd., Dec. '77).
4. Revised the Policy and Procedural Manual for the annual meeting.
5. Accepted the recommendation of the Committee on Women that, where relevant, officials in cities being considered for APA annual meetings in the 1980s be reminded of the Association's resolution on the Equal Rights Amendment (Ex.C., June '77). Voted to approve recommendation of the Assembly as follows: APA will notify legislatures of states that have not ratified ERA of our theoretical policy favoring the ERA and that we shall attempt active implementation by changing convention plans as soon as possible (Bd., Dec. '77).

AWARDS

1. Voted to reduce the Isaac Ray Honorarium to $1.500 and authorized it to be awarded every other year (Bd., Dec. '77).
2. Voted to establish the honorarium for the Foundations Fund Prize for Research in Psychiatry at $1,500 (Bd., Dec. '77).
3. Voted to establish the Blanche F. Ittleson Award at $2,500 (Bd., Dec. '77).

CENTRAL OFFICE

1. Established an Ad Hoc Committee on the Central Office as recommended by the Council on Internal Organization and its House Committee that would include one member representing the Assembly. Its mandate is to study the options available for meeting the need for a Central Office building, report to the Board in December 1977, and begin to

This is an edited version of the report presented by the Secretary to the annual business meeting in Atlanta, Ga., May 9, 1978.

educate the membership immediately through usual channels (including *Psychiatric News*) and alert the Assembly to the problem so it may become actively involved in future planning (Ex.C., Sept. '77).

2. Ratified an emergency action of the chairperson of the House Committee and the Medical Director to install a new furnace at an estimated cost of $14,524, to be charged to the Reserve for Future Expansion and Development Account (Ex.C., Sept. '77).

3. Terminated the Ad Hoc Committee on the Central Office with thanks and authorized the House Committee to be the implementing body to review alternatives for obtaining new facilities for the Association, including remodeling the present facilities, leasing, constructing a building, or purchasing a building; authorized staff to a) sign a sales contract for real property located at Connecticut Avenue and Hillyer Place or other suitable site in the amount of $800,000 and to include in its major stipulations that APA be allowed at least 90 days from the date of the contract to make a final decision; b) obtain architectural and engineering studies and test borings, at an estimated cost of $17,500, to satisfy itself that a building permit can be obtained so as to determine the feasibility of purchasing the property for construction of an office building; and c) deposit $50,000 in an escrow account as earnest money—such earnest money to be returned to APA if the sale is not concluded. Authorized the President, Speaker, and Medical Director to use the emergency mechanism if a superior option was available to allow flexibility for rapid action if necessary; authorized a mail or telephone poll of the Board if there is a need to move quickly, and considered the possibility of holding a meeting of the full Board at the time of the presently scheduled meeting of the Executive Committee in February 1978; voted to request the Editor of *Psychiatric News* to advise the membership of the discussion of the Board around these issues; voted to request staff to send members of the Board full reports of the House Committee and other relevant material to assist them in their deliberations (Bd., Dec. '77).

4. Authorized staff to submit a sales contract for the purchase of the Connecticut and Hillyer property with the same provisions as the previous contract except a) the period allowed for the determination of the feasibility of purchasing the lot be reduced from 120 to 90 days and b) the addition of a clause to ensure that APA's rights are vested prior to closing; authorized discretionary use of earnest money (up to $10,000) to be nonrefundable if APA decides not to purchase the lot even if the feasibility study yields positive results (Ex.C., Feb. '78).

5. Ratified the emergency action of the President, Speaker, and Medical Director to authorize staff to submit a revised sales contract on the property at Connecticut Avenue and Hillyer Place, Washington, D.C., in the amount of $869,220, with $10,000 of the earnest money to be nonrefundable if the sale is not consummated (Bd., Mar./Apr. '77).

6. Approved selection of the architectural firm of Hartman Cox and authorized staff to enter into contractual arrangements with them (Bd., Mar./Apr. '77).

CONSTITUTION AND BY-LAWS

1. Chapter One, Section 8, of the By-Laws ("Members") and Chapter Eight, Section 6, of the By-Laws ("Privileges and Responsibilities") involve the question of Life Status and the "95" formula for dues exemption. Voted against separating the definition of Life Status from the "95" formula in the amendments for the ballot in 1979—the amendment to Chapter One, Section 8, will provide for Life Member and Life Fellow (and the new Life Associate Member) to be defined by the following formula: years of membership as an Associate Member, General Member and/or Fellow, plus the member's age to equal 95, and will also require dues-exempt members to pay a reduced subscription rate for the *American Journal of Psychiatry* and *Psychiatric News* (Bd., Mar./Apr. '77).

2. Chapter One, Section 12, of the By-Laws ("Members"). Approved the following amendment to go on the ballot in 1979: "Inactive members of any category shall be those whom the Board has, for sufficient reason, excused from paying dues" (Bd., Dec. '77).

3. Chapter Eight, Section 5, of the By-Laws ("Privileges and Responsibilities") and Chapter Nine, Section 1, of the By-Laws ("Voting"). Approved amendments for the ballot in 1979 that would require petitions for candidates and referenda to be in the office of the Secretary by December 1 rather than December 15 of the year preceding the next election (Bd., Mar. '78).

4. Chapter One ("Members"). Authorized amendments to allow for exemptions to CME requirements as "exempted by the Board" to maintain membership in good standing, and a requirement for 8 rather than 5 years as General Member to be eligible for Fellowship status for the 1979 ballot. Also authorized a change in the *Operations Manual* to require 10 years of experience in psychiatry to be eligible for Fellowship status (Ex.C., Sept. '77).

5. Approved the recommendations for constitutional change prepared by the Joint Conference Committee on the Key Conference Recommendations; the Committee on Constitution and By-Laws reviewed them to ensure that they were in order; the amendments were submitted to the Secretary 60 days before the annual meeting and were presented to the membership at the annual meeting in 1978, so they can be voted on in the 1979 ballot (Bd., Dec. '77).

COUNCILS, COMMITTEES, TASK FORCES, AND COMMISSIONS

1. Renewed for one more year the following ad hoc committees: on Interprofessional Services; on Election Procedures; to Plan a Conference on the Chronic Mental Patient; on Implementation of the 1976 Referenda on Psychiatric Certification; and to Review *Psychiatric News*. The Ad Hoc Committee on Biographical Data and the Ad Hoc Committee to Plan a Working Conference on Law and Psychiatry were not renewed (Bd., May '77).

2. On recommendation of the Council on International Affairs, approved the "Resolution on Behalf of Argentine Scientists" decrying the general pattern of violations of human rights by the military government of Argentina, and asked that the resolution be communicated to the Organization of American States' Inter-American Commission on Human Rights, the State Department of the United States and concerned congressional committees, and the President of Argentina (Bd., May '77).

3. Established three subcommittees of the Program Committee (Film, Video, and Spouses Scientific Program) (Bd., Dec. '77).

4. Approved the Position Statement on Active Treatment, pending approval by the Assembly (Bd., Dec. '77).

5. Established a Committee on Asian-American Psychia-

trists on recommendation of the previous task force (Ex.C., June '77).

6. Terminated the Committee on Psychiatric Nursing, with thanks (Bd., Dec. '77).

7. Endorsed the concept of providing consultative services to the Commission on Professional and Hospital Activities and recommended to the Reference Committee that travel expenses for a task force within the Council on Mental Health Services be authorized from the 1978 Reference Committee Contingency Fund (Bd., Dec. '77).

8. Established a Committee to Review *Psychiatric News* within the Council on Internal Organization with a mandate to make a formal report every two years to the Council, Reference Committee, and the Board and inclusion of the charge in the *Operations Manual* (Bd., Dec. '77).

9. Established a Committee on Psychiatry and Foreign Affairs within the Council on International Affairs (Bd., Dec. '77).

10. Authorized the Ad Hoc Committee on the President's Commission on Mental Health to prepare a position paper for the PCMH based on 10 points identified by the Ad Hoc Committee and the Assembly to meet the January 3, 1978, deadline for input to the PCMH (Bd., Dec. '77) and ratified the emergency action taken by the President, Speaker, and Medical Director to approve the APA position on the Interim Report of the PCMH (Ex.C., Feb. '78).

11. Endorsed the AMA standards for the education and training of advanced emergency medical technicians and approved in principle becoming a sponsoring organization for the accreditation program and to use the emergency mechanism to so sponsor if necessary (Bd., Dec. '77).

12. Endorsed efforts by the Council on Medical Education and Career Development with necessary agencies to bring about full voting representation of psychiatry on any certification board for emergency medicine (Bd., Dec. '77).

13. Asked a newly forming Task Force on Emergency Psychiatric Care to develop a grant proposal to develop standards for emergency services (Bd., Dec. '77).

14. Authorized the Commission on Judicial Action to prepare an amicus curiae brief in the case of *Ferraro & Kaviani v. the USA*, which has to do with whether a patient who had multiple hospitalizations in VA hospitals should have been retained until his treatment was finished (Bd., May '77).

15. Authorized the Commission on Judicial Action to file an amicus curiae brief on the side of the plaintiffs in *Parham v. J.L. and J.R.*, which involved long-term commitment of two minimally disabled children in the State of Georgia, provided that the Georgia District Branch approves (Ex.C., June '77).

16. Authorized the Commission on Judicial Action to find a witness to testify in the case of *Rone V. Fireman* on behalf of the Department of Mental Health at the request of the Ohio Psychiatric Society (Ex.C., Sept. '77); and approved the recommendation that Dr. Douglas Lenkoski testify on behalf of APA on the side of the Ohio Department of Mental Health in this case (Bd., Dec. '77).

17. Authorized $3,000 to assist in preparing an amicus curiae brief in the *Bellah v. Greenson* case from the special $25,000 fund for the Commission on Judicial Action to assist district branches at the local level (Bd., Dec. '77).

18. Authorized the Commission on Judicial Action to provide an amicus curiae brief for an appeal in Texas in the Fifth Circuit Court on issues of informed consent and prediction of dangerousness (Bd., Mar. '78).

19. Encouraged the Committee on Comprehensive Health Planning to collaborate with the Joint Commission on Government Relations to visit the area councils and facilitate collaboration between the district branches and Health System Agencies (Bd., Dec. '77).

20. Approved four recommendations of the Committee on Continuing Education regarding a) cooperation with the Association of American Medical Colleges to develop review procedures for audiovisual materials, b) the committee's responsibility for the technical and education review process, c) Category I credit for formal university programs that enhance practice, and d) disallowing personal psychoanalysis for CME credit (Ex.C., June '77).

21. Approved initial input to the regulatory process of PL 94-484, Health Professionals Educational Assistance Act (Ex.C., June '77); approved the response to the proposed regulations for the training of physician assistants in accordance with PL 94-484 (Bd., Dec. '77).

22. Endorsed a document, "Toward a National Policy for Psychiatric Manpower—Problems, Assets and Recommendations," which had been prepared conjointly by the Executive Committees of the American Association of Chairmen of Departments of Psychiatry and the American Association of Directors of Psychiatric Residency Training (Bd., Dec. '77).

23. Approved the recommendation of the Committee on Standards of Practice and Third-Party Payment that APA not make a national study of the Insurance Claim Project (field tested in California) and that ongoing work on confidentiality issues continue within the Committee on Confidentiality and the Committee on Peer Review (Bd., Dec. '77).

24. Endorsed an Area II Action Paper approved by the Assembly calling for retention of the Commission on Certification in Administrative Psychiatry until appropriate alternatives are found within or outside APA and rescinded previous action of the Board to phase out the Commission in 1989 (Bd., Dec. '77).

25. On recommendation of the Assembly, approved publicizing the fourth edition of the *Physicians' Current Procedural Terminology* to the membership and encouraged its use in insurance reporting (Bd., Dec. '77).

26. Approved continuing support to the Fellowship program and exploring other sources to fund the program subsequent to the termination of support by the National Medical Fund (Ex.C., June '77).

27. Approved a reduction in size of the fellowship program to 20 Fellows after 1979 (10 second-year Fellows and 10 to be chosen that year); requested the Budget Committee to plan for continuing support and authorized seeking outside funding to partially or, if possible, totally underwrite the expenses to continue the program, including support by the Fellows' residency training program, donations from Fellow alumni, and donations from APA members (Bd., Mar. '78).

28. Endorsed the NIMH Minority Fellowship Program and urged NIMH to continue its support (Ex.C., June '77).

29. Approved inviting an observer-consultant representative of the APA/NIMH Minority Fellows to the meetings—this individual to be elected by the current Fellows and alumni in the program (Bd., Mar. '78).

30. Approved the recommendations of the Committee on Liaison with the American Hospital Association and the Commission on Standards of Practice and Third-Party Payment that APA support the voluntary cost containment program in principle and encourage individuals to support the program in local institutions; that APA take an official pos-

tion that any voluntary cost containment effort must recognize continuing and existing psychiatric services, including research and education, and must give special attention to psychiatric services provided to children, and that the Assembly request district branches and members to inform APA staff of the adverse effects of cost containment as regards psychiatric services in their areas; authorized the Medical Director, in cooperation with appropriate APA components, to seek outside funding to support a study of cost containment (Bd., Mar. '78).

31. Approved the recommendation of the Assembly that APA cease referring to its members as "mental health professionals" and cease using the term "mental health benefits," wherever possible (Bd., Dec. '77).

32. Informally authorized the Joint Commission on Government Relations to coordinate APA's response to the "Legislative Guide" developed by NIMH and currently being published in sections in the *Mental Health Disabilities Law Reporter* of the American Bar Association. The first section concerning civil commitment was distributed to APA members for comment in December 1977. Responses were received, reviewed, and summarized in a special edition of the *Legislative Newsletter* (March 1978) and submitted to the editorial board of the ABA publication for publication in the MHDLR (Bd., Mar./Apr. '78).

33. In collaboration with the President's Commission on Mental Health, APA (Ad Hoc Committee to Plan a Conference on the Chronic Mental Patient) held a conference on this topic in January 1978. Dr. Talbott, chairperson of the ad hoc committee, presented a set of recommendations that are embodied in the preliminary report of the conference to a task panel of the PCMH (Ex.C., Feb. '78). The ad hoc committee is preparing a conference report and working papers of the conference for publication. The Board endorsed the recommendation of the Joint Commission on Public Affairs calling for APA to give high priority to the problems of this patient group and to discuss the composition and future work of a component concerning these issues (Ex.C., Feb. '78).

ELECTIONS AND TERMS

1. Voted to continue the present plurality system of balloting for one more year and then to conduct preferential balloting for three years when there are more than two candidates for one position (Ex.C., June '77).

2. Approved the date of receipt at the Central Office as the deadline for APA ballots, since postmarks are often illegible or nonexistent (Ex.C., May '77).

3. Authorized staff to print on the ballot, "no information submitted," if a candidate's biographical data are not submitted on the official form (Ex.C., June '77).

4. Approved the recommendation of the Ad Hoc Committee on Election Procedures that the President (rather than the Secretary) approve the APA ballot (Ex.C., June '77).

5. Approved printing biographical data in straight alphabetical order on the reverse of the APA ballot, without regard to the order of rotation of names on the front of the ballot (Ex.C., June '77).

6. Endorsed the recommendation of the Ad Hoc Committee on Election Procedures regarding clarification of presentation of biographical data for candidates for election to APA offices, particularly with regard to errors and number of items permitted, and authorized incorporation in the *Operations Manual* (Ex.C., Feb. '78).

7. Authorized the Committee of Tellers and staff to carry out a run-off election, if necessary (Ex.C., Feb. '78).

ETHICAL MATTERS

1. Approved a basic change in procedure that all ethical complaints (no matter where they arise) must be referred to the APA Secretary before there can be any hearing, evaluation of complaint, or other action—and approved the "Procedure for handling allegations of unethical conduct," as submitted by the Ethics Committee (Ex.C., June '77).

2. Reaffirmed its previous opinion that the Nuremberg Code and the Declaration of Helsinki are still guides to be used by physicians doing research on human subjects. (The Ethics Committee was asked to develop guidelines for the acceptance of research funding by psychiatrists and to establish a task force to assist.) (Bd., Dec. '77).

3. Approved the Ethics Committee recommendation that any charges of unethical conduct against Corresponding Fellows or Members or Distinguished or Honorary Fellows be referred to the appropriate component of the World Psychiatric Association (WPA) for advice and recommendations to assist the APA Ethics Committee in reviewing the complaint (Bd., Dec. '77).

4. Established a Committee on Abuse and Misuse of Psychiatry in the United States within the Council on Emerging Issues to have liaison with its counterpart WPA committee, and authorized the charge for the *Operations Manual* to include receiving complaints about ongoing abuses of psychiatry in the United States, referring those that are appropriate to the APA Ethics Committee and referring general issues that do not concern a particular practitioner to the WPA committee for discussion and recommendation (Bd., Dec. '77).

5. Approved sending a letter from the APA President to the Judicial Council of the AMA with comments and suggestions about proposed changes in the new *AMA Principles of Medical Ethics*. (The APA Constitution requires APA members to abide by the AMA principles.) (Bd., Mar. '78).

6. Approved the Assembly position statement on "Abuse of Psychiatry: Continuing Use of Psychiatry and Psychiatrists for Political Purposes," and agreed to empower APA's delegate to the World Psychiatric Association General Assembly (Dr. Weinberg, President) to decide what specific motions he will support and/or introduce at the meeting of the General Assembly; and approved the Assembly's recommendation that APA urge the World Psychiatric Association to establish a mechanism to study reports of political abuse of psychiatry and empowered APA's delegate to bring this to the attention of the General Assembly if he deems it appropriate to do so (Ex.C., June '77).

FISCAL MATTERS

1. Authorized attendance of an AMA delegate to report to the May and fall meetings of the Assembly, and $500 for travel expenses to the fall meeting (Bd., May '77).

2. Voted, on recommendation of the Assembly and the Committee on Constitution and By-Laws, to include the immediate past Speaker of the Assembly and the Treasurer as voting members of the Budget Committee and to revise the *Operations Manual* to include this charge (Bd., Dec. '77).

3. Approved termination of Suter Associates Retirement

Planning Company as administrator of the APA tax-deferred annuity plan (Prudential) (Ex.C., June '77).

4. Approved funding from the Board Contingency Fund to cover planning and development expenses for the 1977 fiscal year ($2,000 for the French meeting and $2,950 for the Pacific Congress of Psychiatry) and recommended that the budgets for fiscal years 1978 and 1979 include funding to support these costs for the Second Pacific Congress with the expectation that APA would be reimbursed from income from these meetings (Ex.C., June '77).

5. Approved APA's participation in a nationwide survey of psychiatrists, conducted by the Medical Activities and Manpower Projects Division of Research in Medical Education at the University of Southern California School of Medicine (Ex.C., June '77).

6. Approved in principle the preliminary proposal for APA's fourth Psychiatric Knowledge and Skills Self-Assessment Program and authorized the PKSAP-IV Steering Committee and staff to enter into further negotiations with the National Board of Medical Examiners (Ex.C., June '77).

7. Approved the program specifications and cost estimate in the amount of $147,305, as proposed by the National Board of Medical Examiners to assist APA in the development of PKSAP-IV (Bd., Dec. '77).

8. Authorized a voluntary fund-raising campaign to the APA membership as the first phase in financing a survey on public attitudes toward psychiatry, and authorized expenditure of $5,000 for the purpose, with the understanding that the money thus expended will be returned to APA from receipts of the drive (Ex.C., June '77).

9. Ratified the emergency action of the President, Speaker, and Acting Medical Director to seek outside funding by submitting a contract proposal to NIMH on "Psychopharmacologic Criteria Development" by the deadline of August 7, 1977; this contract will provide about $10,000 to cover preparation of a document containing such criteria (Ex.C., June '77).

10. Approved funding for the Commission on Psychiatric Therapies in the amount of $10,000 for the first year from the Board Contingency Fund to establish the Commission in May 1978 and authorized the Commission to seek outside funding to supplement APA's support (Bd., Dec. '77).

11. Approved a 7% cost-of-living increase for APA staff as of October 2, 1977 (Bd., Dec. '77).

12. Approved revising the travel regulations to allow reimbursement for the actual cost of rail travel (regardless of class) up to the first-class air travel cost, and increased reimbursement for automobile travel to 15 cents per mile (Bd., Dec. '77).

13. Ratified the mail ballot of the Board for the purchase of a minicomputer in the amount of $103,310 (Bd., Dec. '77).

14. Approved providing complimentary subscriptions to all member associations of the World Psychiatric Association via first class mail (Bd., Dec. '77).

15. Voted, on recommendation of the Assembly, to reimburse deputy district branch representatives for transportation expenses (excluding food and lodging) to the spring and fall meetings of the area councils to bring this level of funding to that of the district branch representative, and to fund these expenses from the Board Contingency Fund in 1978 and to instruct the Budget Committee to include this item in the 1979 budget (Ex.C., Feb. '78).

16. Approved the recommendations of the Treasurer for distribution of surplus funds from 1977 operations and excess receipts from the VI World Congress of Psychiatry (Ex.C., Feb. '78).

17. Authorized Dr. Reginald Lourie, in collaboration with the Medical Director, to seek outside funding in the name of APA to support the program of the Inter-American Council of Psychiatric Associations to use the communications satellite to exchange scientific information between components of the IACPF (Ex.C., Feb. '78).

18. Approved APA's being represented in the AMA House of Delegates by one delegate and one alternate delegate and authorized funding ($1,180) from the Board Contingency Fund to support their attendance at the annual and interim meetings of the AMA House of Delegates in 1978 and instructed the Budget Committee to include this in the 1979 budget (Ex.C., Feb. '78).

19. Voted to inform the AMA that APA wishes to have a Section Council on Psychiatry within the AMA as soon as APA has been notified that the Association is approved for a delegate and alternate delegate in the AMA House of Delegates and directed the Budget Committee to include at least $5,000 in the 1979 budget to fund the Section Council (Ex.C., Feb. '78).

20. On recommendation of the Assembly, voted to retain a Consultant in Parliamentary Procedure to assist in the conduct of meetings of the Assembly in 1978; to fund these expenses from the Trustees' Contingency Fund in 1978 (Ex.C., Feb. '78).

21. Authorized $1,000 in support of the National Association of Insurance Commissioners' Malpractice Closed Claims Study (Bd., Mar. '78).

INSURANCE

1. Approved rate increases (in the APA members' insurance plan) for hospital and nursing care by 50%, major medical insurance by 35%, and a moderate increase for disability insurance for those over 50, because of inflation (Bd., Dec. '77).

2. Approved new rates for professional liability insurance with the proviso that the Committee on Professional Liability Insurance continue to obtain additional data during the next year on which new rate structures can be based (Bd., Dec. '77).

3. Approved reducing all benefits in APA's Major Medical Plan as a temporary measure in keeping with APA's policy of equal coverage for mental and physical illnesses; charged the Committee on Member, Life, Accident and Health Insurance to seek ways to affiliate APA's plan with a larger group, and to explore alternatives if the present insurance plan cannot remain fiscally sound (Bd., Dec. '77). Voted to reconsider its action, but a motion to limit reductions in only psychiatric benefits did not pass and the action of December 1977 stands (Bd., Mar./Apr. '77).

LIAISON WITH OTHER ORGANIZATIONS

1. Authorized APA's becoming an affiliate member of the American Association for Comprehensive Health Planning for one year ($200) (Bd., May '77).

2. Favored ratification of the By-Laws of the Council of Medical Specialty Societies which would allow societies having conjoint examining boards membership within the CMSS (Bd., May '77).

3. Granted the request of the American College of Mental Health Administrators to use the American Psychiatric As-

sociation as a mail address and for purchase of services (Ex.C., June '77).

4. Approved the American Board of Psychiatry and Neurology (ABPN) policy concerning qualifications of applicants with non-U.S. training, and the criterion for approval of the first post-graduate year of training at a training center located outside the United States and Canada, as submitted by the ABPN (Ex.C., June '77).

5. Voted to suggest to the ABPN that the issue of the present system of tenure and a possible six-year term for directors be studied by the ABPN (Bd., Dec. '77).

6. Endorsed the concept of inservice training examinations and instructed the APA Office of Education to work with appropriate organizations to develop an external evaluation program, and authorized a winter 1978 meeting to address the issue of linking inservice training assessment with Board certification (Bd., Dec. '77).

7. Voted to actively explore tripartite composition (APA, ABPN, and AMA) of the Psychiatry and Neurology Residency Review Committee (Bd., Dec. '77).

8. Endorsed the recommendation of the Reference Committee that the full Board of Trustees should review the membership survey on Board certification, after response had been received from the ABPN and returned for later approval by the Board; approved a survey on Board certification to be circulated to a sample of the APA membership (Bd., Dec. '77).

9. Voted that APA instruct its councillors on the Accreditation Council for Psychiatric Facilities (ACPF) to urge the ACPF to review the present standards for community mental health programs and make necessary modifications to ensure that high quality psychiatric care will be a significant and integral part of all community mental health programs; that APA work with AHA to urge the ACPF to move forward toward development of core standards; and that APA's councillors to the ACPF be instructed concerning the APA's position in favor of a single inspection of psychiatric facilities, to avoid multiple surveys (Bd., Dec. '77).

10. Voted to establish liaison with the Association of Mental Health Clergy (Bd., Dec. '77).

11. Voted to establish formal liaison with the Office of Human Development in regard to the proposed White House Conference on the Family (Bd., Dec. '77).

12. Voted that APA urge the American Medical Association to effect continuous psychiatric representation on the National Joint Practice Commission (Bd., Dec. '77).

13. Approved APA's cosponsoring with the National Council on the Aging, Inc., the Annual Conference on Aging to be held April 16–19, 1978, in St. Louis, Mo., at no cost to the Association (Bd., Dec. '77).

14. Recommended that the National Society for Autistic Children, the National Association of Social Workers, and the Epilepsy Foundation of America be admitted for membership in the Accreditation Council for the Mentally Retarded and Other Disabled Individuals (Bd., Dec. '77).

15. Voted to establish liaison with the American Correctional Health Services Association for a period of two years at no cost to APA (Ex.C., Feb. '78).

16. Approved APA's becoming a cooperative sponsor of the Fourth AMA National Congress on Improving the Quality of Life with Focus on the Family at no cost to APA (Ex.C., Feb. '77).

17. Authorized APA's participation in the formation of a National Psychosurgery Board; funding will be sought and provided by the appropriate agency or agencies in the Department of HEW (Bd., Mar. '78).

MEMBERSHIP MATTERS

1. Adopted the policy recommended by the Membership Committee that the appropriate district branch be notified when any member elected to Fellowship has missed three Convocations at which the member was eligible to receive the medallion and certificate, so that there can be appropriate presentation and recognition by the district branch (Bd., May '77).

2. Accepted for inclusion in the *Operations Manual* the policy that a member is responsible for dues to the district branch in which he or she is officially enrolled on the first day of January, as recorded in the Central Office Membership Division; and that, in the event of transfer to another district branch, the above-mentioned payment of dues will keep the member in good standing for the balance of the new branch's year (Ex.C., June '77).

3. Accepted for inclusion in the *Operations Manual* the recommendation that all joint membership actions, other than waiver of dues, shall become effective simultaneously in both the national APA and the district branch as of a date defined by the Central Office for implementation of the action (Ex.C., June '77).

4. Voted to approve the reduction in dues to $110 per year for members who have reached age 70 and who have been dues-paying members for at least 15 years but who have not achieved life status (Bd., Dec. '77).

5. Voted to approve requiring Life Members and Life Fellows to pay for APA publications after January 1, 1979, at a cost of $12 per year for the *Journal* and $6 per year for *Psychiatric News* (Bd., Dec. '77).

6. Voted to authorize the Membership Committee to include Continuing Medical Education requirements for membership in its processing review recommendations regarding membership status of members to the Board (Bd., Dec. '77).

7. Voted to approve the policy that the Membership Committee utilize Inactive Member and Fellow status on other than a permanent basis, that such request be addressed on a case-by-case basis, and that recommendations for temporary inactive status be limited to one year (Bd., Dec. '77).

POSITION STATEMENTS AND RESOLUTIONS

1. Approved the position statement on active treatment, pending further comments and approval by the Assembly (Bd., Dec. '77).

2. Approved the Assembly revision of the resolution of the Committee of Black Psychiatrists on affirmative action (Bd., Dec. '77).

3. Approved the position statement on abortion as approved by the Assembly and the Reference Committee (Bd., Dec. '77).

4. Authorized publication of the Report of the Task Force on Arbitrary Discrimination Against Persons with Previous Psychiatric Treatment, including the position statement in the report that had been approved by the Assembly (Bd., Dec. '77).

5. Rescinded APA's 1972 position statement on involuntary hospitalization of the mentally ill because it does not reflect the current situation, and asked the Commission on Judicial Action to prepare an interim statement for use until the new position being developed by the Committee on Psychiatry and the Law has been finished and approved (Ex.C., June '77).

PUBLIC AFFAIRS

1. Formally requested the President of APA or his designee to communicate directly with the president of the Mental Health Association in the near future to discuss the issue of hard-line tactics regarding the quality of services and efficiency in community mental health centers and other matters of mutual interest (Ex.C., June '77).

2. Authorized the President, Speaker, and Medical Director to release the Joint Commission on Public Relations' press release regarding civil rights of homosexuals, as soon as the suggested modifications are made in the document (Ex.C., June '77).

3. Voted to file a formal "fairness doctrine" complaint with the Federal Communications Commission against the American Broadcasting Company for its news close-up documentary, "Madness and Medicine" (Ex.C. June '77).

4. Authorized the Deputy Director for Business Administration to negotiate a contract with the firm of Yankelovich, Skelly and White to conduct the first phase of the Attitude Research Project, using the monies in the Public Affairs Fund that were collected for this purpose (Ex.C., Sept. '77).

5. Ratified the emergency action taken by the President, the Speaker, and the Medical Director on the submission of recommendations to the President's Commission on Mental Health Task Panel on Public Attitudes and use of the media for promotion of mental health, as requested by the Joint Commission of Public Affairs (Ex.C., Sept. '77).

6. Ratified the emergency action taken by the Vice-President, the Speaker, and the acting Medical Director to send a letter regarding the TV program, "The Other Side of Hell," to the President of NBC-TV (Ex.C., Dec. '77).

7. Ratified the emergency action of the President, Speaker, and the Medical Director to send a letter to the Procurator General of the USSR, asking permission to send a physician-psychiatrist observer to Mr. Scharansky's trial if psychiatric conditions are a factor in this trial (Ex.C., Feb. '77).

8. Authorized the President and the Medical Director to explore the possibility of APA members visiting South Africa, and to consult with the World Psychiatric Association, the Committee of Black Psychiatrists, and the Black Psychiatrists of America concerning their views on such a visit; and asked the President to obtain in writing assurances that any APA members who visited South Africa would be free to go anywhere they wished, to stay as long as necessary, and to use their own interpreters (Bd., Dec. '77).

PUBLICATIONS

1. Authorized publication of "What Is a Psychiatrist?" as revised by the Council on Emerging Issues, with minor editorial revisions and with the addition of a qualifying preface differentiating psychiatrists' treatment qualifications from others' (Ex.C., June '77).

2. Authorized the Joint Commission on Government Relations, with appropriate committee assistance, to develop an operating definition of "what a psychiatrist does" for purposes of legislation and in discussions with legislators and legislative bodies (Ex.C., June '77).

3. Approved the "Model Law on Confidentiality of Health and Social Service Records" for publication (preferably in the *American Journal of Psychiatry*, with the consent of the Editor) (Ex.C., Sept. '77).

4. Approved publication and distribution of the report of the March 1977 Peer Review Conference (Ex.C., June '77).

5. Approved the Report of the Task Force on Professional Liability Insurance for Psychiatrists, "Professional Liability Insurance and Psychiatric Malpractice," for publication as a task force report (Bd., Dec. '77).

6. Approved publishing the proceedings of the APA Symposium on Culturally Relevant Training for Psychiatrists as an APA publication (Ex.C., Feb. '77).

STAFF

1. Received the report of the Ad Hoc Committee to Search for an Editor of the *American Journal of Psychiatry* and selected Dr. John C. Nemiah from a list of highly qualified candidates. Dr. Nemiah will begin his Editorship in July 1978, when Dr. Francis J. Braceland, who has served as Editor since 1965, retires (Bd., Dec. '77).

2. Established a new position, "Associate Director for Government Relations," in the Division of Government Relations (Ex.C., June '77).

3. Approved appointment of a Personnel Advisory Subcommittee (on recommendation of the Budget Committee) composed of three persons to serve as consultants for a period of one year; their function will not include personnel management (Bd., Mar. '78).

H. KEITH H. BRODIE, M.D.

Am J Psychiatry 135:10, October 1978

Report of the Treasurer

GENERAL: This report is prepared from audited figures for the fiscal period that ended December 31, 1977. Data presented also appear in the auditor's annual report.

Attached hereto is a statement of financial condition (table 1) and pie charts (figures 1 and 2) reflecting revenues earned and functional costs for the fiscal period. This report, together with the statement and charts, will provide the membership with the information needed to assess the operation and financial condition of the Association.

FISCAL STATUS: Execution of the fiscal year 1977 budget resulted in income of $6,684,311.40 (9.4% over the budget estimate) compared to expenses of $6,177,009.65 (about 1% over the budgeted figure). This experience resulted in a surplus of income over expenses amounting to $507,301.75 for 1977. Income was strengthened by additional overhead monies stemming from APA's expanding grant/contract program, increases in advertising sales, and attendance at continuing education courses offered during the annual meeting that outstripped projections. Despite the significant increase in activity, the Association was successful in maintaining a healthy expense picture. This stems in part from the ongoing cooperation among the policy-making bodies, officers, and staff in monitoring expenses during 1977. In addition, the growing grants/contracts program allowed program expansion without the increased expenses usually connected with progress. Finally, expenses were dampened by the fact that IRS authorization for the Association to retroactively change its fiscal year period from April 1–March 31 to January 1–December 31 was not received until mid-March 1977. Therefore, new programs contained in the January 1, 1977, budget were not implemented until almost April 1977.

The surplus from operations has been placed in reserve accounts. A portion of the $507,302 surplus ($279,650) was appropriated to the Reserve for Dues Stabilization. This reserve account, currently totalling $665,700, was established in 1976 as a source of funds to offset operational costs in future years, thus delaying the need for an increase in member dues. An amount of $225,371 was appropriated to the Reserve for Future Expansion and Development. This fund, amounting to $1,980,000, is scheduled to be drawn upon heavily to meet architectural and construction costs of a new Central Office building for APA (discussed in detail later in this report). The remainder of the surplus ($2,281) was appropriated to the reserve account entitled Unrestricted Surplus, which currently shows a balance of $260,009. Total reserves have been adjusted to reflect an unrealized decline in the market value of securities over a period of many years amounting to $69,089. APA reserves show a balance of $2,836,620 as of December 31, 1977.

PROGRAM DEVELOPMENT: In accordance with a mandate from the Board of Trustees, the Association has expanded its efforts in the areas of peer review, continuing education, government relations, and public affairs. The stimulation of income sources has enabled this growth to take place within the confines of a balanced budget.

This is an edited version of the report presented by the Treasurer to the annual business meeting in Atlanta, Ga., May 9, 1978.

During 1977, APA moved ahead in the area of peer review by obtaining a government contract in the amount of approximately $678,000 to establish a retrospective system of peer review of CHAMPUS claims for inpatient and outpatient psychiatric treatment. The system is to be utilized on a national scale. One major task of the project is development of review criteria for evaluating the appropriateness of treatment.

The Office of Education increased its effectiveness during 1977 and at the same time achieved a decrease in its 1977 budget as compared with the prior year. This was achieved by a shift in emphasis from operating expenses to personnel. Although an Office of Education has existed in APA for about 15 years, its initial function was related to continuing education projects in psychiatry for nonpsychiatric physicians. Effective January 1, 1977, the Office was given responsibility for all educational efforts within APA, including undergraduate, graduate, and continuing education.

The Public Affairs Division has moved positively toward meeting a mandate of the Board of Trustees to advance the public image of psychiatry. The Division has been strengthened in terms of both staffing and operating expenses. The result is a more positive public relations program for APA, especially in the areas of press relations, development of a public affairs network, and news releases on the positive aspects of psychiatry.

The Government Relations Division has been reorganized to strengthen APA's relations with the staff and members of the Congress and of the Executive Branch. The Association has significantly increased its capacity to develop and provide constructive legislative initiatives rather than to react to measures that may be counter to the interests of psychiatry and the mentally ill. In addition, the Division has initiated relationships with other health interest groups to stimulate broader consensus support on behalf of psychiatrists and their patients.

EMPLOYEE BENEFITS: The policy of the Association seeks to maintain employee salaries approximately commensurate with those paid by the federal government (the primary employer in the Washington, D.C., area). Thus, a cost-of-living increase closely matching that granted by the federal establishment (7%) was awarded to our employees in October 1977. Other benefit costs increased due to staff expansion and inflationary pressures; however, these costs were all within budgetary guidelines.

GRANTS AND CONTRACTS: In February 1977, based on the recommendations of the Treasurer, the Executive Committee of the Board of Trustees identified the obtaining of grants and contracts to support needed programs as a top priority and authorized an expenditure of $8,000 from its 1977 Contingency Emergency Fund for the procurement of such funds. At that time, the Association was receiving monies via grants and contracts at the rate of $294,842 per year. Efforts of management, at all levels within the Association, culminated in the attainment of five grants/contracts during 1977 amounting to additional income of $881,892, for a total annual income from grants/contracts of $1,176,734. Overhead income expected from grants and contracts has increased from $16,000 projected for 1977 (prior to the top pri-

TABLE 1
Comparison of Fiscal Years 1976 and 1977

Item	1976	1977
ASSETS		
Cash and savings accounts	$ 457,620	$ 15,000
Certificates of deposit	320,000	320,000
Accounts receivable	492,203	674,893
Accrued interest receivable	29,559	38,738
Deferred and prepaid expenses	99,711	147,545
Marketable securities		
Stocks and bonds (at quoted market value)	1,023,749	1,047,442
U.S. Treasury bills, etc., short-term investments at cost	594,130	1,165,863
Notes and advances receivable from the American Psychiatric Museum Association, Inc.	1,757,085	1,757,085
Less valuation reserve	(1,757,085)	(1,757,085)
Land and buildings (at nominal value)	1	1
Furniture and equipment (at nominal value)	1	1
Total assets	$3,016,974	$3,409,483
Deduct		
Cash and marketable securities allocated from general fund to restricted fund	302,223	400,964
Total general fund	$2,714,751	$3,008,519
LIABILITIES		
Accounts payable and accrued expenses	137,464	11,954
Due to district branches	3,639	36,641
Deferred income	20,573	23,304
Reserve for stabilization of future dues	579,075	665,700
Reserve for future expansion and development	1,754,629	1,980,000
Less valuation reserve, marketable securities	(38,357)	(69,089)
Total liabilities	$2,457,023	$2,748,510
Unrestricted surplus		
At beginning of year	257,728	257,728
Net change during year ended		2,281
At end of year	257,728	260,009
Total general fund	$2,714,751	$3,008,519

ority established for grants/contracts) to $120,000 expected during 1978 (an increase of $104,000). Federal funds to support fringe benefits for employees are projected to increase from $5,000 per year to $30,000 (an increase of $25,000). Efforts to expand revenue from grants and contracts will continue in 1978 and prospects are good for continued success.

CENTRAL OFFICE BUILDING: The Association staff has now exceeded the bounds of its existing office building, resulting in overcrowded conditions in the Central Office buildings and the leasing of space at two additional sites. Also, staff working conditions are inadequate, especially during warm and cold periods, and the trend for increased costs of building operations and maintenance noted during past years continues. These problems led to an extensive evaluation of the APA buildings by an architectural and engineering firm, an assessment of the property by a member of the American Institute of Real Estate Appraisers, and an analysis of real estate opportunities for the Association in the Washington metropolitan area. These studies revealed that renovation of the existing buildings is not feasible and point to the need for APA to obtain new office space to house its Central Office facilities. These studies indicate a current need for approximately 32,000 square feet of net usable office space initially, with expansion to about 50,000 square feet of space (based on industry averages) within 10 years. The Treasurer, in conjunction with the Investment Advisory Committee and staff, conducted a study of the fiscal implications of a decision for the Association to purchase or construct its own building, at an estimated cost of $4,500,000, to house its Central Office, as opposed to a decision to lease space for this purpose. The study revealed that a decision for APA to purchase or construct its own building would result in net *income* of more than $2,000,000 over a 30-year period. This compares to *costs* of $37,000,000 if a decision were made to lease. Therefore, APA would realize an advantage of about $39,000,000 over a period of 30 years if it elects to purchase or construct its own building rather than lease comparable office space. The APA House Committee and staff have reviewed approximately 20 buildings in the Washington metropolitan area and found none that meet the needs of the Association. After reviewing over 20 available lots, it has been determined that the lot located on the northern corner of Connecticut Avenue and Hillyer Place, Washington, D.C., is suitable in terms of location and size. A sales contract for the lot in the amount of $869,220 was executed on March 20, 1978, by both the seller and APA, with the understanding that the Association would have 90 days (until June 18, 1978) to conduct additional feasibility studies prior to closing.

INVESTMENTS: The stated investment policy of the Association is to "employ sound investment vehicles affording the maximum return consonant with safety of capital, i.e., the type of investment a prudent individual would seek." As a result, the majority of the Association's portfolio (86.4%) is invested in intermediate and short-term securities. The $320,000 held in savings and loan association certificates accrued interest at the rate of 7½%, for earnings of $24,000. Savings accounts in commercial banks (which were opened in late 1976 because of low yields on government obligations) had an opening balance of $450,000. Funds were held in these accounts until the yields on government obligations became more attractive. As the rates increased, large portions of these funds were transferred from these accounts into Treasury bills. Interest earned on these savings accounts amounted to $18,600. The U.S. Treasury bills constituted the remainder of investment funds with a balance of $1,165,863 at year's end. The amounts held during the year varied between $2,300,000 and $300,000. Earnings from these vehicles amounted to over $82,500 for the period. The year just ended was a year of decline in the stock market. It is interesting to note that in 1977 the percentage of decline for the Standard & Poor's "500," and Dow-Jones Industrial Average, and the New York Stock Exchange Composite were 10.7%, 11.5%, and 17.3%, respectively. Therefore, under the guidance of our Investment Advisory Committee, our portfolio decreased its holdings in equities and invested transferred funds in more secure fixed-income securities such as bonds and short-term notes. Equity holdings amount to only 13.6% of the total portfolio. The total decline in the equities section of the portfolio was 8.2% for the year, which compares favorably with the market in general. However, it should be noted that the average gain on equity investments since the establishment of the Investment Advisory Com-

FIGURE 1
Comparison of APA Revenues, in Percents

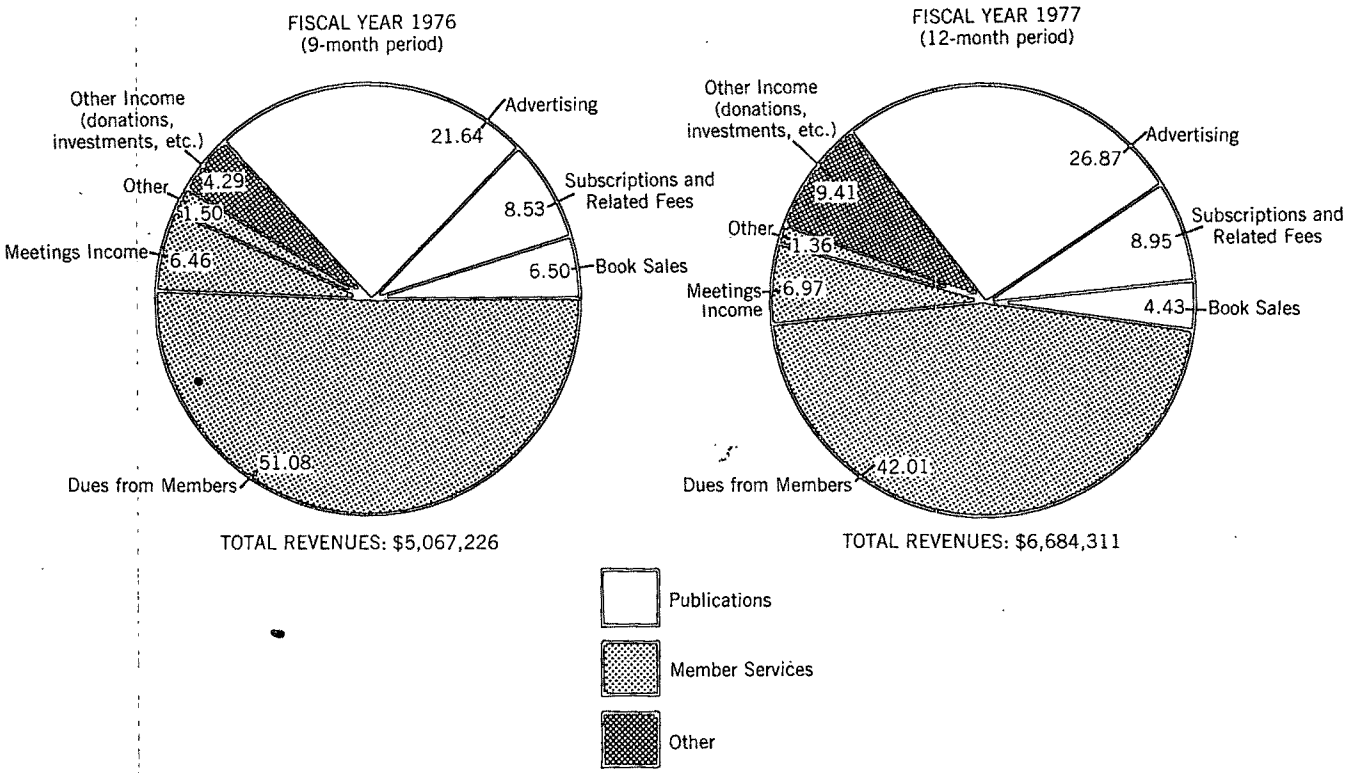
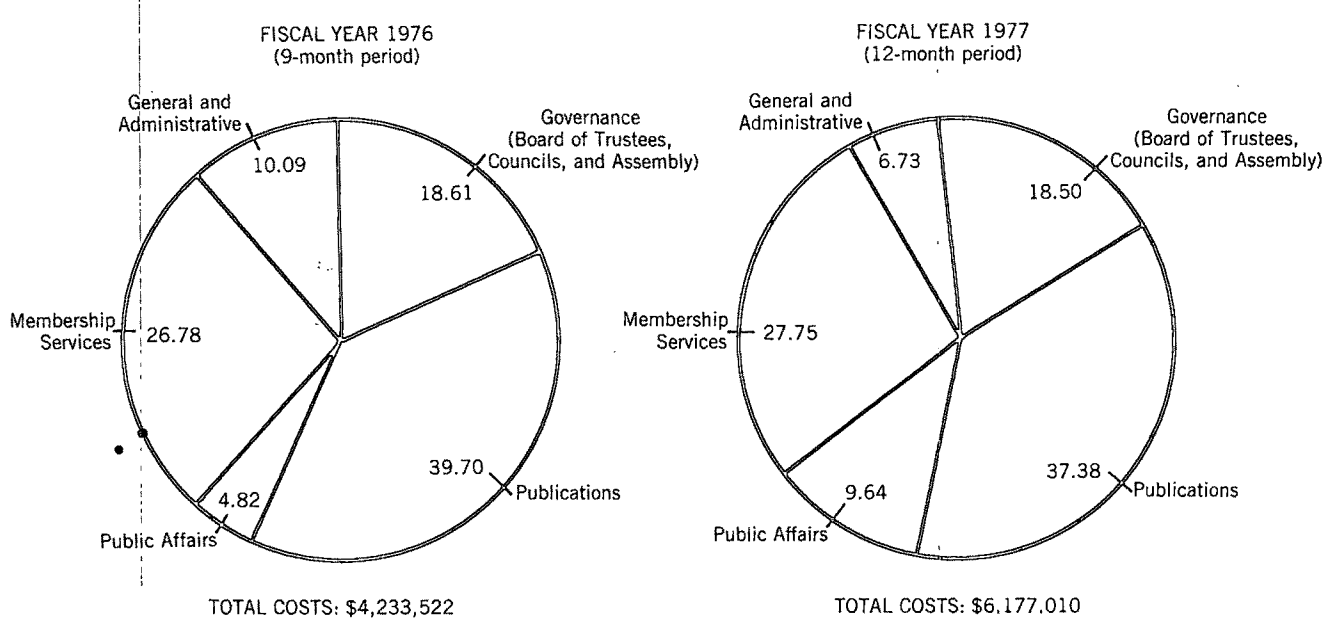


FIGURE 2
Comparison of APA Costs, in Percents



mittee (from October 1975 through December 1977) has been 15.2%. Dividends on stocks for the year totaled $17,050 and income from bonds amounted to $30,238.

PUBLICATIONS: *Psychiatric News* generated revenues of $998,804, contrasted with expenses of $841,006, for a surplus of $157,798. The revenue amount represents an increase of $407,658 over the 1976 amount. This gain stems primarily from a great increase in advertising revenues.

The *American Journal of Psychiatry* produced revenues of $874,553 and generated expenses of $948,569, for a deficit of $74,016. It should be noted that the Association levies an overhead charge to its three publications at the rate estab-

lished for indirect costs for federal grants and contracts. The overhead rate for the *American Journal of Psychiatry* during 1977 was 37.4% of direct costs. This publication showed a surplus of $137,483, excluding APA overhead charges.

The *Hospital & Community Psychiatry* journal realized revenues amounting to $295,806 and expenses of $394,357. The resulting deficit was $98,551. After adjusting for the APA overhead charge to the journal, the deficit for 1977 was reduced to $10,610.

The Association maintains approximately 90 book titles in inventory. The total revenues, net of discounts for 1977, were $266,457, which showed a modest gain over 1976 totals. The Association's publications are produced and marketed at prices which assure a return of the printing and distribution costs. The excess of revenues from sales over expenses keeps this activity on a self-sustaining basis.

VI WORLD CONGRESS OF PSYCHIATRY: APA served as host for the VI World Congress of Psychiatry, held in Honolulu from August 28 through September 3, 1977. In this capacity, the Association accepted full responsibility for the business and fiscal arrangements for the Congress. The event was successful in both professional and operational terms. Proceeds from the meeting were sufficient to allow APA to 1) donate $50,000 to the World Psychiatric Association and 2) reasonably cover the costs incurred in the planning and conduct of the Congress.

CONCLUSION: It is a pleasure to present what I believe is a very forthright and positive annual report on the fiscal activities of APA during 1977 and the financial condition of the Association at this time.

I wish to express my gratitude to the membership for entrusting me with the responsibility for the fiscal affairs of APA over the past two years and to the Assembly of District Branches, the Board of Trustees, the officers, and the Investment Advisory Committee for their support and their ongoing interest in the financial stability of the Association. Appreciation is also due to the Medical Director, the Deputy Director for Business Administration, the Comptroller, and the entire staff for their capable assistance whenever needed during the year.

CHARLES B. WILKINSON, M.D.

Am J Psychiatry 135:10, October 1978

Report of the Medical Director

IT IS A privilege to present my fourth annual report to you. I am particularly pleased to say that staff development has proceeded to a point where we can implement the goals and aspirations of our membership in a qualitatively improved manner. We are indeed a complex organization with multiple functions and perspectives. It is striking, however, that in recent years much greater consensus has been achieved regarding how we must be responsible and accountable in providing the best possible psychiatric treatment for our patients.

The Association continues to grow at the rate of approximately 1,000 new members per year, and there are distinct organizational implications of this level of growth. In my report this year, I focus on the consolidation of activities that members have requested, including continuing education, member benefits, government relations, and judicial action. In addition, APA is planning a new headquarters building which will provide an opportunity to consolidate structurally as well as functionally.

Internal consolidation of activities, to allow for management by objectives and line functioning, has led to the development of various staff committees to coordinate activities involving various staff. Examples of these committees are the Staff Committee to Coordinate the Board and Assembly and the Staff Committee on the Annual Meeting. These committees allow for a free-flowing exchange of ideas that we hope will improve the various activities that staff is involved in implementing for the membership.

This year has also brought a new coping phenomenon dealing with the multiple areas of accountability. Since September, we have been concerned with appropriate input into the preliminary report of the President's Commission on Mental Health. Now, the vital concern will be providing comments on the final report before legislation and regulation-writing.

National health insurance developments must be constantly monitored while we continue to develop data that will support the best possible psychiatric coverage for our patients. Related to this, one of the major issues on the current horizon is government pressures for medicine to be involved with voluntary cost containment.

During the past year, APA has assumed more of a proactive leadership role in developing a consortium of psychiatric education groups, in meeting and collaborating with the Mental Health Association in areas of mutual agreement, and with the American Psychological Association in attempting to define areas of agreement as well as delineating adversarial positions. Staff has spent a great deal of time working with the executive and legislative branches of government in attempting to ensure that the NIMH budget is adequate, that APA has some input into the changes being contemplated at ADAMHA, and suggesting persons to be considered for the directorship of NIMH.

APA has also assumed a growing and important role in the international arena; the outcome of the resolution presented by the Association to the World Psychiatric Association at the General Assembly in August 1978 in Hawaii will be reported to you. The APA Committee on Psychiatry and Foreign Affairs has been active in a new study, "The Emotional Aspects of the Present Egyptian/Israeli Interaction," and a group of APA members traveled to Cairo and to Jerusalem this year.

This is a capsule overview of the many activities that your staff has been engaged in; I will speak to some of these issues in a little more depth.

For historical purposes, I have asked each department and division head, and each deputy director, to prepare a detailed report on activities. This is available to any member who might wish a copy.

CHAMPUS PSYCHIATRIC PEER REVIEW PROJECT

One of our most innovative projects, perhaps with the most far-reaching implications for the field of psychiatry, began in the summer of 1977 when a contract was signed between APA and the Civilian Health and Medical Program of the Uniformed Services (CHAMPUS) to provide a peer review system that would cover the entire country. The contract provided $678,054 for operation of the project for one year, with a renewal clause. A project office was immediately established, headed by Mr. Norman Penner, and staff was assembled.

There are five specific tasks to be carried out by the APA: 1) provide a National Advisory Panel of psychiatrists to oversee the development and operation of the peer review system; 2) develop criteria (inpatient and outpatient) to be used by CHAMPUS claims processors to determine which claims go to a peer review panel; 3) identify psychiatrists willing to conduct CHAMPUS claims peer review; 4) training CHAMPUS claims processor personnel to handle psychiatric claims and case review; and 5) provide peer review. The contract provides that each case going to peer review is to be reviewed by three psychiatrists. APA will monitor reviewers' performance and reimburse them at the rate of $50 per hour.

Each district branch was asked to provide the names of at least four psychiatrists who might serve as peer reviewers. Some 300 names were received and a letter of appointment/agreement was sent to 295 psychiatrists.

In summary, the guidelines have been completed and presented to the CHAMPUS staff, the CHAMPUS fiscal intermediary staffs have been trained in the system, and some 290 psychiatrists have been assembled and are ready to begin the peer review for CHAMPUS.

Not only is there a high degree of interest in the project as evidenced by the large number of letters from members who have volunteered to serve as reviewers, but the federal employees' Blue Cross/Blue Shield plan and Aetna have asked to be kept informed about the progress of the program. There is no doubt that the project will result in a more uniform claim adjudication process nationally than is now the case, and may indeed prove to be a model for use by others.

This is an edited version of the report presented by the Medical Director to the annual business meeting in Atlanta, Ga., May 9, 1978.

GOVERNMENT RELATIONS

The division provides the interface for APA policy with the legislative and executive branches of the government, and, in addition, seeks to provide insights into and analysis of such legislative and executive activities to assist in the development of APA policy. As a result of APA's renewed commitment to improve and expand government relations activities as requested by various members and the Assembly, Jay B. Cutler, J.D., formerly Minority Counsel to the U.S. Senate Health Subcommittee, was appointed Special Counsel and Director of the Division in May 1977. During the past year the division has actively engaged the Congress on each issue that has an impact on psychiatry, ranging from national health insurance and health planning to drug regulation reform, NIMH appropriations, reimbursement for nonphysicians, and others.

One of the most important activities, undertaken in conjunction with Mr. Sam Muszynski, was ensuring that the Health Planning Law (P.L. 93-641) included mental health planning. The law has been successfully amended to explicitly include mental health planning.

In conjunction with the coalition of psychiatric education organizations, the division is actively lobbying to increase the budget for mental health manpower training in the Labor/HEW Appropriations Bill, fiscal year 1979, from $86.6 million to $108.25 million.

In all of the above, and in many more instances, APA has demonstrated proactive legislative responsibilities and is responsible for the inclusion of genetic conditions leading to mental illness as a matter for research and training under Title XI of the Public Health Service Act.

PUBLIC AFFAIRS

In August 1977, all APA members received a mailing asking for donations to support a survey of public attitudes to be under the direction of the Joint Commission on Public Affairs. Obviously this was an activity that greatly interested the membership, and more than $50,000 was donated by APA members to support the study. The survey was begun in November and preliminary results were shared with the members at a special session during the annual meeting. The firm of Yankelovich, Skelly and White, a nationally prominent research organization, conducted the survey; it is anticipated that there may be additional research or public relations programs developed on the basis of results of the research.

The division produced the first two issues of the *Public Affairs Network Newsletter*; in its first year, this network has grown to include 33 district branches and will be extremely useful in working with the news media.

The division hopes to provide accurate information about the psychiatric profession, refer reporters to experts in the field, establish more ongoing relationships between members and the media, and encourage reporting of events favorable to psychiatry in order to enhance its public image. In relation to these efforts, the division issued 10 press releases involving: APA's position on homosexuality, the Joint Information Service study on nursing homes, passage of the ERA, a protest of the ABC broadcast "Madness and Medicine," and comments on the preliminary report of the President's Commission on Mental Health.

In March, APA was informed that the National Science Foundation had awarded a grant of $37,600 to support a Symposium for Media Writers, "Research Briefings on Mental Health," to be held this summer. Mr. Robert L. Robinson and the Division of Public Affairs will work with the Joint Commission on Public Affairs in arranging for this meeting.

GENERAL COUNSEL

On January 1, 1978, Rogovin, Stern and Huge were designated General Counsel for the American Psychiatric Association. Before that, Mr. Joel Klein of that firm had served as Special Counsel. The major litigation effort of APA (other than judicial action matters) was to challenge the ABC program "Madness and Medicine." A "Fairness Doctrine" complaint was filed with the Federal Communications Commission in October, attacking the bias of—and the harm to the public created by—"Madness and Medicine." FCC staff has been actively investigating the complaint; ABC replied to the issues raised by APA in the complaint and APA also responded to the ABC document and filed additional materials, including an affidavit from Dr. Russel L. Guiss, Superintendent of Dammasch State Hospital in Wilsonville, Ore., expressing concern about the biased approach used by ABC in filming and reporting on conditions at that hospital. As yet, the FCC has not ruled on the complaint.

Mr. Klein and the firm have been quite active in advising the district branches on various legal matters, particularly those involving ethics and tax status, matters of confidentiality, and state commitment laws.

Another major activity was a legal analysis of the issues involved in setting up a body within the World Psychiatric Association to investigate and monitor abuses of psychiatry throughout the world. This analysis was used by Dr. Jack Weinberg and me at the meeting of the WPA Executive Committee in New Delhi in January. I plan to report further on the follow-up of issues relating to the WPA later in this document.

OFFICE OF EDUCATION

There has been rapid growth and development of the Office of Education under the guidance of Dr. Carolyn Robinowitz. The structure and function of the office continue to undergo major change. The office, in collaboration with the Council on Medical Education and Career Development, has been able to make great progress in the area of psychiatric education and has allowed APA to play a leadership role in the academic community as convenor, overseer, and stimulator for consortial projects from all sectors of the academic community. As part of its responsibility for all education efforts, the office has expanded its activities in the areas of undergraduate, graduate, and continuing education, and has become increasingly involved in issues related to manpower development.

The Office of Education has cosponsored approximately 335 programs for an estimated 4,606 hours of Category I CME credit. These programs have been developed primarily by district branches; the office has worked most closely with branches in New York, Massachusetts, Colorado, Washington, and the District of Columbia. Staff is also available for program consultation to district branch continuing education committees, and Dr. Robinowitz and Assistant Director Dr. Sue Quick have visited a number of branches and Area Councils during the past year.

APA continuing education will be mandatory to retain

membership, and the office will begin to inform individual members of the need to comply with the CME requirements on or before July 1, 1979. An information and recording booklet has been prepared and mailed to members; there is also a fact sheet on continuing medical education which was mailed to the entire membership. Reminders and information have also been prepared for district branch newsletters and for *Psychiatric News*.

The Office of Education was awarded a grant to hold a series of national and regional conference workshops on Educational Development for Psychiatric Educators (EDPE). These meetings are designed to stimulate development of educational planning, implementation, and evaluation skills, and have been planned through the joint efforts of APA and a consortium of psychiatric educational organizations. A national conference was held in January 1978 focusing on the topic Teaching the Resident How to Teach. There were 200 participants, and initial feedback and evaluation have been quite positive. Future planned activities include two or three regional conferences in 1978–79 and publication of proceedings and continued development of resource materials for teaching.

As the continuing education functions of the annual meeting increase, there has been greater liaison between the Office of Education, the Division of Public Affairs, and the Office of Professional Affairs in planning and implementing these activities.

Problems are related to change, increased demands, and further development of functions. During the coming year the office hopes to determine and implement policy on basic and continuing education through work with the Council on Medical Education and Career Development, develop a stronger research base in all aspects of psychiatric education, develop educational materials for the field, develop and maintain closer liaison with other staff and with outside organizations regarding government activities to ensure APA's viability, develop a closer working relationship with ABPN to develop a research base to determine the reliability and validity of the evaluation process, and to extend the close working relationships with psychiatric service and education organizations, with nonpsychiatric medical specialty societies, educational organizations, and other mental health disciplines in areas of common interest and concern.

DEPUTY MEDICAL DIRECTOR

During this past year, Dr. Donald W. Hammersley was instrumental in negotiating the contract with CHAMPUS and the Department of Defense which has resulted in the APA project alluded to earlier in this report. Negotiated primarily because of his efforts, this innovative collaboration between APA and CHAMPUS is one of the first national peer review projects undertaken by a medical specialty organization.

Working with an ad hoc committee, Dr. Hammersley and his staff planned and carried out the logistics for the Conference on the Chronic Mental Patient, held January 11–14, 1978. The conference, held in collaboration with the President's Commission on Mental Health, produced important recommendations which were presented to the President's commission and will be circulated to APA components, with a view to developing an APA policy and program for the chronic mental patient.

Under a contract with NIMH, APA has submitted a document on "Psychopharmacologic Screening Criteria," and we hope NIMH will grant permission to publish these criteria in the next revision of the *APA Manual for Psychiatric Peer Review*. Dr. Hammersley, working closely with Dr. Richard Dorsey, provided support to the panel of experts who developed these criteria as a model for a review process to identify what prescribing practices of psychopharmacologic medication should be examined in peer review.

OFFICE OF PROFESSIONAL AFFAIRS

During 1977 this office developed a brochure titled "Careers in Psychiatry" to answer the increased number of inquiries (more than 900 in 1977) concerning the practice of psychiatry which come in from high school and college students. This is a valuable information booklet that is available to anyone interested.

The Hospital & Community Psychiatry Service continues at the approximate level of last year, with slightly over 800 institutional members. The H&CP Institute, held in San Francisco in 1977, was a success and attracted more than 1,135 registrants. The focus of the meeting, "The Chronic Patient: Rights, Responsibility, Resources," provided valuable information to the Conference on the Chronic Mental Patient and to the President's Commission on Mental Health.

There has been an increase in activities in this office, as Dr. Henry Work has assumed staff responsibility for the Staff Committee on the Annual Meeting, the Staff Committee to Coordinate Board/Assembly activities, the Program Committee, the Assembly, and the Council on Research and Development. In addition, Dr. Work serves as a member of the Steering Committee for the Key Conference Recommendations.

Many liaison activities are carried out through this office; Dr. Work was recently elected secretary of the Group for the Advancement of Psychiatry and secretary-general of the American College of Psychiatrists.

OFFICE OF MINORITY AFFAIRS

Mental health issues relating to underrepresented groups continue to be the main focus of activity and responsibility of the office. In addition to providing staff support for the Council on Children, Adolescents and Their Families, and to the Council on National Affairs, the office has directed time and effort toward keeping channels of communication open between the councils and the various components in liaison activities, both within and without the APA structure. Liaison has been maintained with the American Academy of Child Psychiatry, the American Association of Psychiatric Services for Children, State Mental Health Representatives for Children, and the American Academy of Family Practice.

In addition, Dr. Jeanne Spurlock serves as the APA official liaison to the American Medical Women's Association and is a member of the NIMH Epidemiological Review Committee and the Graduate Medical Educational National Review Committee (GMENRC).

This year, a second Research Symposium was held for the APA/NIMH Fellows. There were 11 new fellowships awarded and 16 renewed; 6 Fellows completed their training in 1977. A committee of APA/NIMH Fellows volunteered to assist in the development and evaluation of a questionnaire,

to be distributed to all American medical schools, to assess mental health services for medical students.

This office has also provided support during the planning stage of the proposed Survey of Psychiatric Services for Black and Colored peoples in South Africa. This study developed from charges of alleged psychiatric abuse, the refutation of the charges during the VI World Congress of Psychiatry, and the subsequent invitation for APA to send two psychiatrists to visit South Africa, at the expense of the South African government, to look at psychiatric facilities. The visit is still in the planning stages but we are hopeful that it will occur during the coming year.

BUSINESS ADMINISTRATION

This office has now been in operation for three years. During 1977, emphasis was placed on provision of support services and fiscal resources necessary for expansion and refinement of priority programs, preparation of a balanced budget for fiscal year 1978, continued reorganization of the business activities, and planning for the development of new office space for APA.

The introduction of a new accounting machine and remittance control machine has minimized errors, reduced the time required to complete transactions, and provided for more detailed analysis and reporting of costs.

The Publications Services Division has assumed responsibility for the Publications Sales Office, providing coordination and control. In addition, this division has been active in monitoring and stimulating sales of advertising space. Sales increased by over $200,000 during 1977. A comprehensive project to evaluate the performance of the present advertising sales representatives has been initiated.

The Administrative Services Division has moved ahead significantly in the area of member benefits with the establishment, in May 1977, of the Office of Member Benefits. This office allows APA to respond to the needs of members more effectively and efficiently, and has enhanced coordination of APA components involved in insurance benefits for members.

During 1977, APA staff personnel increased from 107 to 122; 118 full-time and 4 part-time. In addition there were 13 terminations, for a turnover rate of 15%.

The Administrative Services Division also undertook the major task of revising the Personnel Policy Manual, a task which has not been done since 1963.

ASSEMBLY/BOARD

This year staff has attempted to help achieve a more viable communication system not only between the Board and the Assembly but with the district branches and area councils. In September the position of Coordinator for the Board and Assembly was established and Ms. Jeanne Robb was appointed to this post. In addition, during this past year we have attempted to provide additional staff support to the Area Councils, and various members of the staff have attended these meetings to discuss issues of concern, e.g., continuing education, government relations, and public affairs, just to mention a few.

To achieve better staff coordination, a committee was appointed with Dr. Work as chair; members include all those who provide support to the Assembly, the Board of Trustees and the Reference Committee.

In June, immediately following the 1977 annual meeting, the district branch executive secretaries came to the APA Central Office for an orientation session. This meeting was essentially one in which we attempted to provide information about how the Association operates and information about staff structure. It was a very productive meeting and will probably be repeated in 1979.

This year, steps were taken to close the communication gap between the Assembly and the other APA components, namely, the councils, commissions, committees, and task forces. During the Fall Assembly Meeting period, the Assembly liaison members to APA councils and other components met before and after the meeting to discuss how best to communicate information to the Assembly and to provide for Assembly input into issues under discussion.

AMERICAN JOURNAL OF PSYCHIATRY

After 13 years of distinguished service during which the *Journal* achieved preeminence among psychiatric periodicals, Dr. Francis J. Braceland announced his retirement as of July 1978, and the Board of Trustees selected Dr. John C. Nemiah as his successor.

In his final report to the Board of Trustees Dr. Braceland noted that in 1977 the *Journal* had experienced a larger increase in the number of papers received than in any year since he became Editor; the overall increase was 19%—1,220 papers received in 1977 compared with 1,028 in 1976. An ad hoc committee, headed by the then Editor Designate, Dr. Nemiah, met and discussed various solutions to problems occasioned by the large backlog of accepted manuscripts resulting in part from the increased number of submissions. Among the suggestions were printing one or more special issues or supplements, preferably grouped around a given topic and carrying some advertising (which would help defray costs), and adding more pages to the text section of each issue for at least a six-month period. The staff is moving toward implementing these recommendations.

PSYCHIATRIC NEWS

This is the 13th year of publication of this newspaper, which continues to provide the APA membership with broad coverage of various subjects of interest to psychiatrists. Independent studies indicate that the newspaper is probably the most widely read publication in the field.

During 1978, a new standing Committee to Review *Psychiatric News* will be appointed, reporting to the Council on Internal Organization. Staff welcomes this direct member input in determining whether *Psychiatric News* is continuing to meet APA member needs.

HOSPITAL & COMMUNITY PSYCHIATRY

During 1977, this journal continued to make progress in closing the gap between income and expenditures and steps are being taken to strengthen its financial base. There is now under way a review of *H&CP's* advertising sales to determine if the journal is currently being represented to the best advantage and if not, what steps should be taken to ensure that it is.

The magazine has been promoted primarily to psychiatric units of general hospitals, but promotional material is also

being mailed to private psychiatric hospitals, community mental health centers, partial hospitalization programs, and other groups.

H&CP is unique in that it is the only monthly journal specifically directed to professionals who work in organized treatment settings.

DATA PROCESSING DEPARTMENT

The Data Processing Department, under the Membership Division, was officially established in late April 1977. A long-range plan had been developed and APA contracted with Oceanic Enterprises, Inc. for a cost-effective turnkey system; OEI was to develop APA-owned software in a language compatible with the mini-computer that APA would eventually purchase at the end of a three-year timesharing period.

However, major internal problems developed at OEI and it was finally concluded that they could not complete the work. Increased staff technical expertise and recognition of the problems associated with dependence on an outside company to produce necessary work resulted in staff recommending the purchase of a mini-computer and custom software, which was approved by the Board of Trustees in December. During this period the department has produced and mailed 5 dues billings, processed dues payments, billed for 40 of the 71 district branches, completed specifications for all data processing requirements, and redesigned dues notice and membership card forms.

This has been a transition year; all work completed was due to heroic efforts by the staff of the Data Processing Department. It is our hope that all phases of the system will be fully operational by 1979.

LIBRARY

The librarian serves as liaison to the newly formed History, Library and Museum Committee. This group (formerly the History Commission) has prepared a "Bibliographic Guide to the History of American Psychiatry," which will be published in 1978.

A staff survey was conducted to determine the library's present information functions and future directions. As a result of this survey, a Staff Library Committee has been formed to discuss future directions of the library.

The archives special fund was terminated in 1977. However, many collections of interest to the history of APA and psychiatry in general were acquired.

In addition, archives staff have developed an overall records management program for the Association and a records management person is now a part of the library staff. During 1978, there will be an inventory and scheduling of records in order that accurate and necessary records are properly stored.

VI WORLD CONGRESS

This staff office was terminated on April 31, 1978, with the final reports on file. The meeting, held in Honolulu in August 1977, was a financial success; after all bills had been paid, APA was able to forward $50,000 to the World Psychiatric Association for use in operating the secretariat until the next World Congress. A total of 4,506 persons were registered at the congress.

MEETINGS MANAGEMENT DEPARTMENT

This department was responsible for arranging more than 200 meetings during 1977, including the fall meetings of components, Board of Trustees, Assembly, the Institute on Hospital & Community Psychiatry, and the annual meeting. In addition, the department was responsible for logistics for the VI World Congress of Psychiatry.

Also, for the first time in APA history, Meetings Management was to be involved in the logistics and staff support for the Joint Meeting of the Association with the Société Medico-Psychologique in Paris, scheduled immediately following the 1978 annual meeting. It is our feeling that staff should be involved in these joint meetings in order that the very best possible planning for our members can be accomplished.

MEMBERSHIP SERVICES AND STUDIES

The major events of 1977 were the return to the division of responsibility for dues billing other than production and mailing, problems with dues billing associated with the breakdown of the computer service, the signing of a contract with NIMH for a census of psychiatric manpower, and production of the *Biographical Directory*.

As of January 1, 1978, APA membership stood at 24,210, compared to 23,301 in 1977. APA continues to grow at a rate of approximately 1,000 new members per year.

During 1978, the division will begin the manpower study, with the hope that valuable data will be developed.

The workload of the division continues to increase. Apart from the increase in sheer numbers of members and membership transactions, a number of functions have been added to or returned to the department, and existing functions such as balloting have become more complex and time-consuming. In addition, this division will now provide staff support to the Committee on Constitution and By-Laws, and continue to do so for the Committee of Tellers, the Membership Committee, and the Ad Hoc Committee on Election Procedures.

JOINT INFORMATION SERVICE

This continues to be a very productive and important activity, jointly sponsored by APA and the Mental Health Association. One of its most important studies was published this year, *Mental Health Services for the Elderly*. Also, a report of a study of prevention services provided by community mental health centers will be published shortly. During the coming year, JIS will seek additional outside funding to support four extremely important studies: "The Effect of National Health Insurance on Psychiatric Services in Canada," "The Role of the States in Mental Health Research," and two studies intended to encourage the proliferation of psychiatric services in general hospitals.

OTHER ACTIVITIES

This past year, at the request of the Assembly and the Board of Trustees, Dr. Jack Weinberg, as the official APA

delegate to the World Psychiatric Association General Assembly, introduced a resolution that asked that WPA establish a Committee to Review the Abuse of Psychiatry, which would receive and review allegations of violation of the WPA policy regarding abuse of psychiatry and to recommend such corrective actions which seem appropriate to the WPA Executive Committee. This motion was passed by the General Assembly.

Drs. Jack Weinberg and Melvin Sabshin attended the WPA Executive Committee Meeting in New Delhi, India, in January to discuss the formation of this committee and presented a document, prepared by the APA General Counsel, dealing with the legal issues involved in setting up such a committee within the WPA. The WPA president has appointed an ad hoc committee to consider the form and function of the committee.

Also, Mr. A.B. McNulty, former Secretary of the European Commission on Human Rights, will work with a committee of three jurists who will be asked to advise the WPA on procedures to be adopted. In addition, Mr. McNulty will meet with APA and the All-Union Society of Psychiatrists (of the U.S.S.R.) to discuss issues involved in the establishment of such a committee.

APA has assumed a very proactive role in the field of international psychiatry. The APA Committee on Psychiatry and Foreign Affairs has travelled to Cairo and to Jerusalem to discuss the emotional aspects of the present Egyptian/Israeli interaction, meeting with various government and health officials.

At the request of the district branches and the Assembly, the Commission on Judicial Action has laid out a plan to assist in district branch litigation, including monies to support legal cases in the states. Guidelines have been drawn up and APA will support only those cases that have important public policy implications. For example, APA will support cases involving standards for civil commitment, the so-called right to treatment and the right to refuse treatment, and those involving important issues of patient confidentiality.

The Commission on Judicial Action hopes that this program will be of assistance to the district branches; we will report further to you next year on the outcome of the first year of experience.

APA has continued and accelerated its efforts to secure psychiatric input into the national health planning structure established by P.L. 93-641. Efforts have been focused toward seeking substantive changes in the number of federal regulations required by the act, sustaining district branch involvement with state and local health planning agencies, and recommending various legislative changes to congressional bills recently introduced to revise and extend the planning act.

Mr. Sam Muszynski, who has directed the Office of Comprehensive Health Planning, also serves as Liaison Officer for Federal Agencies. The office functions have expanded to include various additional activities with federal agencies responsible for the administration of programs that affect psychiatry including health manpower regulations required to nated APA responses to a number of issues of import to psychiatry including health manpower regulations required to implement P.L. 94-484, the Health Professions Education Assistance Act of 1976, reports and recommendations of the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research, and regulatory procedures reform in the Department of Health, Education and Welfare.

As we become more active in the legislative field, it is of equal importance for APA to have input into the regulation writing process that implements the laws affecting psychiatry.

CLOSING REMARKS

We continue to make progress in our attempts to coordinate staff functions in order to provide maximum service to members.

1978 will be an active year as we respond to the Report of the President's Commission on Mental Health, providing input into any legislation that is proposed as a result of the recommendations; as we make plans to implement the new Constitution and By-Laws, if the changes are approved by the membership; as we work with the many agencies of government that impinge on the practice of psychiatry: HEW, ADAMHA, and NIMH, to name just a few. In all of these activities, we will carry out the mandates and policies of the Assembly, the Board of Trustees, and the members.

I would like to thank each of the officers, members of the Board of Trustees, officers and members of the Assembly, and all of the many members who have worked so hard to improve our organizational effectiveness.

As Dr. Jack Weinberg's presidential year comes to a close, I want to express to him our appreciation of his strong leadership and good working relationships with staff. His support has indeed made this complex, changing year a most pleasurable one.

MELVIN SABSHIN, M.D.

Am J Psychiatry 135:10, October 1978

Report of the Speaker

THE ASSEMBLY of the American Psychiatric Association celebrates its Quarticentennial at this 1978 Annual Meeting. As the twenty-fifth Speaker of the Assembly, it gives me pride and satisfaction to look upon the accomplishments of the past quarter century of "grassroots" involvement in the deliberations of our professional organization. This past year particularly has seen some milestones. I would like to enumerate some of them for you.

Perhaps our most significant organizational event is bringing to closure the deliberations about the governance of APA, and developing for presentation at this meeting and for vote of the membership in 1979 changes in the APA Constitution and By-Laws. The product developed is the distillate of extensive input from the district branches and the consequence of what was called the "Key Conference" three years ago. I look forward to the reception our membership will give this proposal when it comes to them for vote in 1979.

The Assembly has accomplished many things over the past year. We have realized a revision and updating of the Procedural Code that governs the conduct of the Assembly. We have accomplished revision and updating of the Guidelines for District Branch Officers, providing for the district branch leadership a "how to do it" handbook to ease the often burdensome administrative and organizational chores of our leaders. For the first time, district branch executive secretaries participated in a productive orientation and training session conducted by central office staff. We have seen the Assembly take the initiative in developing guidelines for caring for and serving impaired physicians, which augment and complement the position of the American Medical Association. We have seen a new relationship between the Assembly liaison persons and the APA council structure, with more forceful leadership and direction on the part of the Assembly in the councils. A precedent was established by virtue of the President-Elect consulting with the Speaker-Elect of the Assembly on APA Presidential appointments, utilizing the extensive and very satisfactory suggestions and recommendations solicited from the district branches. The Assembly Executive Committee supported the action of the Board of Trustees in obtaining a site for a new headquarters building. Engineering surveys and soil testing are in progress and an architect has been selected. A new headquarters building promises long-range cost savings and functional improvement of our Central Office activities. The position of Recorder of the Assembly has evolved into an extremely important post: summarizing, keeping track, and reporting to the Assembly action items completed; items referred to district branches for study, comment, and recommendation; or items referred to other components of APA. I am indebted to John Houck for his excellent stewardship of this difficult and extremely important task. Recognizing that this is our Silver Anniversary year, and acknowledging the importance of a sense of history in the organizational decision-making process, a historical update of the Assembly and the district branches was developed.

I would like to share with you some of my perceptions of trends within the practice of psychiatry. I see a continuing transition from theory to technology. Technology in the practice of psychiatry has differed from technology in the rest of medicine. In the general medical field, the hospital was perceived as the only location in which to apply technology, and the heightened expectations were related to magical cures. Technology did not create the cure-all that had been anticipated. The disillusionment, disappointment, frustration, anger, and further demands for a response—from government, and from the technology of today's society—were the inevitable result. Technology in the practice of psychiatry operates in a direction different from that of general medicine in that psychiatry has decreased rather than increased its dependence on the hospital. The psychodynamics of the 1940s were overrun by the psychopharmacology of the 1950s and the heightened expectations of the 1960s, and the community mental health center movement expanded psychiatry into a "quick fix" for broad social problems. I hope we are at the tail end of the rebound antimedical and antipsychiatric attitude of the 1970s.

There is a seductiveness in nostalgia. We wish that we could return to the physician-humanist model of the early part of this century. It is my hope, and it will be my effort, to see the new psychiatrist modeled as a physician-humanist-scientist, who feels comfortable in the arts *and* the sciences and can provide clinical and program guidance for patients who are unable to cope with life crises or whose structure of character and personality cause them to abrade the social fabric of our communities and who may need highly technical intervention, as well as those with major mental disorders, for whom compassion, understanding, and human care must serve as we strive for greater scientific knowledge of these illnesses so painful to our patients and their families.

This past year saw the United States as the host country for the meeting of the World Psychiatric Association. We have concerned ourselves with the "rights issues" that resulted from this meeting. There was also much scientific and technological information and knowledge presented in Honolulu that can be helpful to us in program designs. We find that retrospective studies about the causes and results of mental disorder are still in process and are telling us the same things cross-culturally. The expectation that "controlled cultures" would enable follow-up and provide information not revealed in the United States has not borne fruit. Prospective studies are still in the "maybe" stage. The Third World countries appear to place a great emphasis on the use of social policy as a means of influencing health and controlling disease. Thus far what we have seen is that death-preventing technology has been successful; however, disability-preventing technology has not. We also have seen that, cross-culturally, primary prevention as a relevant and successful technology is not in view.

Observation on a lighter side about our activities leads me to make a few irreverent comments about our annual meeting. Having participated in some 15 of these annual meetings in my 20-plus years as an APA member, I must say that the

This is an edited version of the report presented by the Speaker of the Assembly of District Branches to the annual business meeting in Atlanta, Ga., May 9, 1978.

APA annual meeting seems to be the intellectual equivalent of a fundamentalist revival. We attend, purge ourselves of guilt over neglect and inaction, and return to our clinics and practice doing the same things we have been doing. We see an ecumenical embrace of indulgences in the form of CME credits, in addition to the traditional tithing by a dues payment and fees. One thing CME has done is to teach doctors to be "creative" when completing their CME forms. Whether the annual meeting will survive as an APA institution remains to be seen. The American Medical Association, the nation's largest professional group, is abandoning its huge annual meeting and replacing it with a series of smaller regional meetings directed primarily toward meeting continuing medical education requirements. The bell you hear tolling may be blown by the winds of the Equal Rights Amendment.

I could not end without some commentary about the most important public mental health event in the past two decades—the establishment of the President's Commission on Mental Health. Although modest in its service recommendations, the report recognized and addresses great needs in research, and puts focus on the oft-neglected group of patients with major mental disorders. Let us be ready for a real surge of progress as the seeds of this report take root in the capital of this country and its recommendations are implemented in our programs throughout the land.

Participation in the affairs of APA as one of its elected officers, although exhausting, is a very rewarding personal and professional experience. I thank you for the opportunity to serve.

DANIEL A. GRABSKI, M.D.

Am J Psychiatry 135:10, October 1978

Report of the Speaker-Elect

THE ASSEMBLY has been fortunate in its leadership during the 25 years of its history, and it is an awesome honor to have been chosen as its Speaker in these troubled times. The past several years have witnessed many changes in the organization of the American Psychiatric Association, and much of what had been thought to require constitutional revision has already been effected through less drastic modifications of procedures. The net result has been an amicable balance between the powers of the Assembly and the Board of Trustees, whose continuing attentiveness to the spirit of the Key Biscayne Conference can only be admired by those who view structure as a means to an end rather than an end in itself. Whether the current alliance between those two bodies will be enough to secure the goals of APA remains for the membership to decide. I hope that whatever decision is made will be on the basis of an informed membership, and that every effort will be made—at every level of APA—to look at the implications of the proposed changes in the Constitution.

It would be unfortunate, however, if we were to allow ourselves to focus exclusively on matters of internal organization. Now, more than ever before, we must grapple with external pressures and demands, and we need to come to some consensus on the host of policy issues that confront us. Any report on how psychiatry is faring at the moment in the arena of public policy might best be entitled "Notes from the Firing Line." Although it seems that only yesterday we were riding a strong wave of acceptance and enthusiastic support, the wave appears to have broken over our failure to fulfull the expectation we had generated in our admirers. Many of yesteryear's allies seem to have become this year's adversaries, now claiming not only that they do the same things we have always thought of as our functions, but even that they do them better.

The trail that we blazed through the mental health forest has become an overused expressway. We no longer enjoy exclusive rights of passage; quite the contrary, we are often challenged when we try to pass through the entrance turnstile and are asked to prove why a psychiatrist is necessary to do any of the things that have been traditional parts of our daily practice.

In the face of these countless skirmishes at the periphery, it is easy to lose sight of the central battle plan: to ensure the availability of a full range of appropriate psychiatric treatments for those who need them.

Several major obstacles to the attainment of that end can readily be identified. Often overlooked is one that is endemic to American society, where competence and achievement are revered and anything less is disavowed. As Norman Dain points out (1), this has meant that mental health reformers have generally deserted the objects of their original munificence when the "saved" patients are found to remain silent and inactive, refusing to assume the power wrested for them and seemingly unable to group themselves into a dependable and identifiable constituency for their rescuers.

We have our own brands of reformers today, and they appear in two main guises. One is the antiscience theorist, who argues that since most illness is socially induced, there is at best an expensive "window-dressing" role for medical science in the prevention or treatment of disease; that medical intervention only upsets the natural balance, which is more suitably maintained by naturopaths or other health cultists; that medicine fosters survival of the *un*fit and thereby endangers the very society it would treat; and that twentieth century treatments harm more often than they help.

Not radically different in their conclusions, and sometimes even more abrasive in their methods, are the second brand of reformers, the consumer advocates. They distrust the establishment, bureaucracy, and professionalism (which they view as the cornerstone of the health "industry"). In psychiatry, consumerism has focused on the issue of civil rights—the right to treatment, the right to refuse treatment, informed consent, commitment procedures, etc. In APA's Legislative Newsletter of March 6, 1978, I commented at length on the first chapter to emerge from the Mental Health Legislative Guide, which was published in the July-August 1977 issue of the *Mental Disability Law Reporter* as "Proposals for Change—Civil Commitment." At this point, I would only remind you of some of the more distressing proposals as an illustration of the dangers that threaten not only the profession, but even more lethally, our patients. Throughout the document, the major thrust is that the psychiatrist and his clinically based opinions are not to be trusted, and that the patient must be protected at every turn of the road by a lawyer, no matter how costly, cumbersome, traumatic, superfluous, or irrelevant his solicitous ministrations might be. The proposals ignore the patient's psychiatric condition to shepherd him through a maze of adversary proceedings, court hearings, and treatment review committees that would build a system of mandated malignant neglect. While the psychiatrist will not be allowed to testify at the time of commitment about possible dangerousness to self or others (because psychiatry has not demonstrated its competence in this area), the psychiatrist nonetheless will be asked to make such a prediction at the time of release. The proposals would allow all sorts of nonmedical personnel to be involved in treatment decisions; they advocate such intimate and restrictive involvement in clearly clinical (and not legal) issues to raise the specter of nonmedical committees ultimately deciding, by simple majority vote, what dosage of what drug may be given to what patient, at what frequency, and for what period of time. Beginning with the stage of preliminary examination and extending through the period of hospitalization and recurrent treatment reviews, the proposals require a massive dispersal of professional and other personal information to those who have neither a legitimate interest in some of the data nor a capacity to utilize them. The proposals obstruct access to treatment and destroy all possibility of continuity of care. They would transform the doctor-patient relationship into an adversary process that strips the physician of all power to make clinical decisions. They invite nonphysicians to define the specifics of treatment while they relegate the medical professional to an adjunctive role where his major function is to prepare reports for the

This is an edited version of the report presented by the Speaker-Elect of the Assembly of District Branches to the annual business meeting in Atlanta, Ga., May 9, 1978.

scrutiny of the judiciary or other reviewing authorities. The proposals' preoccupation with the mechanics of monitoring the medical profession bespeaks a reckless disregard for the welfare of the patients they claim to protect.

Distressing as such reforms might be, we must recognize that the biggest reformer of them all is the government. We know only too well that the federal government pays for an ever-increasing proportion of medical care in the United States, and it is demanding more and more control over what it pays for. What at first was a bothersome overemphasis on cost and productivity has become an intrusion by the government into professional decision-making. Costs can always be cut by shrinking the system, and that the government is clearly trying to do. But must we stand idly by if it goes on to destroy the system?

In order to deal with a society that may have good reason to distrust the establishment, in order to gain access to the architects of public policy, in order to send our message to the molders of public opinion, psychiatry must be able to articulate some concrete positions and policies that will have meaning for those we want to reach—the government, the legislatures, the media, and the citizenry.

I have already indicated how I would phrase our overall goal, viz, ensuring the availability of a full range of appropriate psychiatric treatments for those who need them. That goal can, I believe, be broken down into four major subdivisions, within each of which occur the specific, concrete questions we must be prepared to answer. Those subdivisions are as follows: 1) the kind of treatment given to those who need it, 2) who is to give the treatment, 3) where will the treatment be delivered, and 4) how will treatment be paid for?

This is hardly the time to discuss all the concrete issues contained within those subdivisions. Their very multiplicity constitutes one major problem we face; an equally important one is that any answer we might agree to give to one question will significantly affect the answers we can legitimately give to others. Let me show you what I mean.

As many of you know, it has long been my contention that we in APA have failed to define our role as physicians. In attempting to define what psychiatry is, we have often listed all the skills that the aggregate of individual psychiatrists might have. Having labeled those skills as psychiatric, we have argued that whatever is done by a psychiatrist is medical (and therefore compensable under medical insurance, to cite one application of the argument). Perhaps because we have been reared in a tradition of circular reasoning, we have also argued that since those skills are medical by definition, they cannot be practiced by anyone else.

Let us pretend that somehow we manage to break out of the circle, that as a group we agree to define what it is that the physician part of us contributes to the mental health field, and to recognize that some of the things a psychiatrist does by tradition are not, per se, medical.

By agreeing among ourselves on what a psychiatrist is, we would have answered one of the specific questions under the heading, Who is to give the treatment? In so doing, we might find that we had also answered one of the questions about how it will be paid for, in that we might find that third party payers would drop their discriminatory approach against psychiatric patients once we defined their discipline in more clearly medical terms. But at the same time we would soon learn that those among us who espouse a social systems, or ecological model of mental illness would feel betrayed. We would also have to recognize that such a step would put most of us in a position of practicing only part-time as physicians who treat the mentally ill, and the rest of the time as psychotherapists or counselors who deal with a variety of headaches in living in much the same way as other professionals and paraprofessionals within the broad field of mental health. We can also predict that by narrowing the definition of what is medically oriented psychiatry, we will have freed up the medical potential of currently practicing psychiatrists. Their medical skills can now be channeled into the void that would otherwise have required an influx of new psychiatrists.

If that were true, it might be possible to say in addition that training funds could be deployed more profitably in other areas, such as research or direct patient care. What would happen then to training programs whose ability to self-destruct seems no more remarkable or dependable than that of our own committees and task forces? And if some programs were to be eliminated, who would decide—the government, which funds them; the training directors, who run them; the residents, who use them; or the patients, who will buy their products?

As you can see, each answer is but a prelude to more difficult questions, and now you will also understand why I characterized my election to this position as an awesome honor. I can only trust that I will have your support as I continue to raise these issues before the Assembly and the Board of Trustees, and I hope that I will have your help in doing battle with the forces that would deny our patients the treatments they need.

REFERENCE

1. Dain N: From colonial America to bicentennial America: two centuries of vicissitudes in the institutional care of mental patients. Bull NY Acad Med 52:1179–1196, 1976

ROBERT J. CAMPBELL, M.D.

Am J Psychiatry 135:10, October 1978

Report of the Membership Committee

THE APA MEMBERSHIP COMMITTEE met for two and a half work-filled days, October 26–28. The first two days were spent with the processing of the various membership and fellowship actions and policies as required by the Constitution and procedures established by the Board. A joint meeting was held with the Assembly Membership Committee to consider policy matters of concern to both committees. I would like to discuss some of the items requiring Board action as part of my report.

The most difficult and sensitive task of the committee is the review of and recommendations concerning Fellowship nominations. This year the committee reviewed 224 nominations and has recommended 124 to the Board to be made Fellows. The percentage of committee approvals was down this year (55% versus 66% in February 1976 and 73% in October 1976). I am at a loss to explain this phenomenon. This year's committee was perhaps more deliberate than previous committees; however, I do not believe the standards changed—some members reviewed by previous committees were again nominated for Fellowship. The committee did feel that district branch screening was generally more lax this year and certainly more erratic. A number of district branches presented large numbers of nominees who had been very poorly screened. Several of these district branches used to do better and thus contributed greatly to the decline in the number of nominees recommended. A breakdown by district branch of nominations versus passed for 1976 and 1977 shows that if those district branches which passed 25% or less but nominated two or more candidates were removed from the totals, there would then be a much more "normal" pass rate of 62%.[1] Perhaps the fact that there were two Fellowship reviews last year introduced an additional aberration, i.e., a relatively depleted pool of eligible nominees. These matters were discussed in the joint meeting with the Assembly Membership Committee.[2]

The committee deferred 18 requests for advancement of at-large members. This was due only to the lack of necessary information regarding the candidates. Since the Membership Committee has the sole responsibility for assuring that these members meet the requirements for APA membership, the committee decided that it should have more complete information, including three references, certification of satisfactory completion of residency; and proof of licensure if required by the Constitution. While recognizing that gathering more information on persons requesting advancement to Membership-at-Large status will require additional staff, the committee felt that approving these advances with less information would be abrogating its responsibility.

In joint session with the Assembly Membership Committee, there was an extensive discussion with Dr. Robinowitz of the implementation of continuing medical education requirements. We were impressed with the outstanding work being carried out by Dr. Robinowitz and the staff of the Office of Education. In the course of the discussion, the question of the role of the Membership Committee in enforcing continuing medical education requirements was raised. There was a general consensus that the Membership Committee would be involved under its mandate to review and

TABLE 1
Membership Transactions

Status	Number in 1977
New	1300
Provisional Members	6*
Members-in-Training	584
General Members	688
Associate Members	14
Corresponding Fellows	1
Corresponding Members	4
General Members-at-Large	2
Advancements	802
Member-in-Training to General Member	662
Associate Member to General Member	136
Member-in-Training to Associate Member	4
Resignations and drops	340
Total resignations from APA	102
Total drops for nonpayment of APA dues	153
Total drops/resignations from district branches and consequently dropped from APA	85
Reinstatements	45
Member-in-Training	1
General Members	40
Associate Member	1
Fellows	3
Transfers in member status	150
General Member to Fellow	124
Fellow to Corresponding Fellow	1
General Member/Member-in-Training to Corresponding Member	14
General Member/Associate Member to Member-in-Training	8
Corresponding Member to General Member	3
Life Fellows, Life Members, and 50-Year	
Life Fellows/Life Members	269
Life Fellows, 1977	195
Life Members, 1977	57
50-Year Life Fellows/Life Members	17
Inactive status	26
Inactive Fellows	4
Inactive Members	22
Honorary and Distinguished Fellows	6
New Distinguished Fellows	4
New Honorary Fellows	2
Membership Committee deferral actions	123
Transfer General Member to Fellow	98
Applications for Corresponding Member	2
Advancement from Associate Member/ Member-in-Training-at-Large to General Member-at-Large	19
Requests for waiver denied, transfer from General Member to Fellow	2
Requests for advancement denied, transfer from Associate Member to General Member	2
Deceased	213

*Plus 1, effective January 1, 1978.

This is an edited version of the report presented by the Membership Committee to the annual business meeting in Atlanta, Ga., May 9, 1978.

[1]A table of fellowship nominations versus recommendations is available from Dr. Elpers on request.

[2]A copy of the report of the Assembly Membership Committee is available from Frederick Gottlieb, M.D., Chairperson, on request.

TABLE 2
APA Membership

Status	Number as of January 1, 1977	Number as of January 1, 1978
Provisional Members	—	7
General Members	13,548	14,653
Fellows	4,387	4,273
Members-in-Training	1,495	1,380
Associate Members	844	692
Life Members	422	449
Life Fellows	1,535	1,671
Corresponding Members	156	175
Corresponding Fellows	207	204
Inactive Members	648	504
Inactive Fellows		139
Distinguished Fellows	26	29
Honorary Fellows	33	34
Total	23,301	24,210

make recommendations to the Board of Trustees on issues of membership; however, we felt clarification is needed. It seems appropriate that the Membership Committee should review and make recommendations in those cases where membership status is to be affected by failure to meet continuing medical education requirements as it does now when a member fails to pay dues or meet other requirements of membership. Although this issue will not be pressing until 1979 or 1980, the committee would appreciate the Board's clarification of policy now so the necessary procedures can be established.

A final item brought to the Board of Trustees for policy consideration was the issue of reduced/excused dues for persons temporarily unemployed or employed only part-time. This problem seems to pertain primarily to women members who take time off from their practice for purposes of pregnancy and/or child-rearing; however, it also applies to a wide array of other special situations, one or two of which come to the attention of the Membership Committee each year.

The constitutional membership category of Inactive Member or Fellow has, in the past, been used only for those Members or Fellows with severe, permanent disabilities. Concern was expressed that should it be used otherwise, many frivolous requests for inactive status would arise and it would be difficult to assure that the member return to full dues-paying status when the reason for inactive status was no longer operative. However, it was also generally agreed that situations do arise wherein there is a legitimate reason for a member to become temporarily inactive and he/she should be able to do so without resigning membership completely.

The Board of Trustees approved two policy items at their meeting on December 10, 1977: 1) the Board of Trustees vests in the Membership Committee the responsibility to include continuing medical education requirements for membership in its processing, review, and recommendations regarding membership status of members to the Board; and 2) the Board of Trustees approves the policy that the Membership Committee utilize Inactive Member or Fellow status on other than a permanent basis, and that recommendations for temporarily inactive status be limited to one year.

JOHN R. EVERS, M.D.

Am J Psychiatry 135:10, October 1978

Report of the Committee of Tellers

THE COMMITTEE OF TELLERS met on March 9, 1978, at APA headquarters to certify the results of the 1978 election. Ballots had been mailed on January 30, 1978, at the direction of Dr. Melvin Sabshin to 22,421 eligible voting members. Sixty-eight undeliverable ballots were deducted from that number; the final number of eligible voting members was 22,353; 13,704 ballots were returned and included in the final vote tally (61.3% of the eligible voting members).

The Committee of Tellers acted on questionable ballots that had been held for their decision. The tellers also verified that all candidates for the 1978 election had certified the accuracy of their submitted biographical information and that all candidates had submitted the required statements of compliance with election guidelines.

The Committee of Tellers certified that the following individuals were elected to office: Alan A. Stone, M.D., President-Elect; Lewis L. Robbins, M.D., Vice-President; Charles B. Wilkinson, M.D., Treasurer; and Henry B. Brackin, M.D., Area V Trustee.

The Committee of Tellers further certified that in two offices, Trustee-at-Large and Area II Trustee, no candidate had obtained the mandated 40% plurality of the votes cast, as required in the *Operations Manual* of the Board. A run-off election between the top two candidates was held; Dr. Gene L. Usdin is now Trustee-at-Large, and Dr. John A. Talbott is Area II Trustee.

The Committee of Tellers certified that the two referenda (petition to demand a moratorium on the ABPN oral examinations and petition on convention sites and the Equal Rights Amendment) were defeated; the percentage of those in favor of each petition did not meet the requirements contained in the Constitution and By-Laws for adoption of referenda.

LOUIS F. RITTELMEYER, M.D.

This is an edited version of the report presented by the Committee of Tellers to the annual business meeting in Atlanta, Ga., May 9, 1978.

Report of the Committee on Constitution and By-Laws

THE COMMITTEE ON CONSTITUTION AND BY-LAWS (CCBL) was very busy this year. Numerous recommendations and referrals were received from several components of the Association. These were reviewed and studied, and then recommendations were made to the Assembly and to the Board of Trustees. The recommended amendments to the Constitution and By-Laws were approved by the Assembly and the Board of Trustees for reading to the membership at the annual meeting in May 1978 and placement on the ballot in 1979 (see appendix 1). In keeping with Article IX, Paragraph 3, of the Constitution, and Chapter Fourteen, Paragraph 1, of the By-Laws, they will be disseminated to the membership prior to January 1, 1979.

The CCBL, the Assembly, and the Board recommended the passage of these amendments. It is understood that if the membership approves the proposed Joint Conference Commission Constitution and By-Laws (JCC), these amendments, if approved by the membership, will be incorporated in the new Constitution and By-Laws. If the JCC document is not approved by the membership, these amendments, if approved, will be incorporated in the present Constitution and By-Laws.

The CCBL reviewed the JCC document[1] during its stages of development over the past year. This was in keeping with the responsibilities of the CCBL as outlined in the Constitution and By-Laws and the *Operations Manual*. During the Fall Committee Meetings the CCBL spent much time going over the document and making numerous recommendations to the JCC. These were taken into consideration by the JCC. In March 1978 a final review was made prior to the JCC presentation to the Board of Trustees in April 1978 and to the Assembly at the annual meeting in Atlanta and placing on the ballot in 1979.

The full minutes of the meetings of this committee are available to the membership of the Association, and any member is welcome to read them. The CCBL was unanimous in its recommendations of the proposed amendments and the recommendations to the JCC. There is a minority report in the minutes of the fall meeting, which expresses opposition to the recommendation that the committee recommend the petition document to the Assembly and the Board. The petition document was later withdrawn. The minority report also recommends that the functions and rules of the Assembly be more clearly spelled out in the Constitution and By-Laws rather than in the *Procedural Code* or *Operations Manual* and expresses opposition to the change in governance as proposed in the JCC document.

The proposed amendments are summarized as follows:

1. Constitution, Article II, and By-Laws, Chapter One, Section 9. This is proposed to create a new category of "Life Associate Member." The APA Membership Committee has found situations in which the absence of this category has been a problem and has recommended this amendment.

2. By-Laws, Chapter One, Section 3. After approving the requirement for CME it was realized that there would be situations where there should be an exemption from this requirement. An example would be a member who has retired or dropped out of active work for some other reason. This proposed amendment would provide for the Board to have discretion in this area.

3. By-Laws, Chapter One, Section 7. The Membership Committee studied the time requirement for advancement from General Member to Fellow and felt the limit should be raised from five to eight years.

4. By-Laws, Chapter One, Section 8, and Chapter Eight Section 6. For some time studies of the fiscal matters of the Association have shown that a problem is developing because of the increasing number of members reaching "Life" status. At the same time there seems to be a leveling off of the increase in new members each year. With the previous rapid rise in membership there will be more new Life status members; also, many members now reaching this level are still actively working. After much study by several components of the Association, it was felt wise to extend the time for a member to reach Life status, and with final approval of the Board, it was decided that a member will reach Life status in his or her category when the sum of his or her years of active membership in the Association and his or her age adds up to 95 (the "95" formula). By interpretation of the Constitution and by authorization of the Board this will not affect the present status of any member who has already reached Life status.

5. By-Laws, Chapter One, Section 12. The original section on inactive status was not clear; the proposed amendments on the "95" formula raised the question of a possible difference between an Inactive Member whose years in that status would not count toward the "95" formula and a "waiver of dues" member whose years in that status would count. The specific situations for these two categories would be spelled out in the *Operations Manual*.

6. By-Laws, Chapter Eight, Section 5, and Chapter Nine, Section 1. With the changes in the election procedures over the past few years, the Central Office staff has found an increasing problem in submitting the required material before the publishing deadlines for *Psychiatric News* on the ballots. This includes biographical data, position statements, photos, etc. Moving the deadline back from December 15 to December 1 will allow two months for the gathering of petition signatures, data, etc.

The committee appreciates the cooperation, requests, and recommendations of the various components of the Association and the review, study, critiques, and approval of these amendments by the Assembly and the Board of Trustees.

I wish to thank the members of the committee for their hard work, understanding, time, and willingness to work for a solution of the problems. The minority report outline above was well written and will be valuable to the membership in its deliberations.

W. PAYTON SCOTT, M.D.

This is an edited version of the report presented by the Committee on Constitution and By-Laws to the annual business meeting in Atlanta, Ga., May 9, 1978.

[1]The JCC document will appear in full in the October 2 issue of *Psychiatric News*.

APPENDIX 1
Proposed Amendments to the APA Constitution and By-Laws

The proposals that follow were received within the 60-day deadline before the annual meeting and were presented at the annual business session. They will appear on the ballot in March 1979.

Note: The following changes in type style indicate the kind of change proposed in the Constitution and By-Laws: 1) italic = proposed additions to the Constitution and By-Laws (e.g., "by resolution of the Assembly"*), and 2) brackets = proposed deletions from the Constitution and By-Laws (e.g., "may originate [either] . . .").* *Articles and sections of the Constitution and chapters and sections of the By-Laws that are not listed here have not been subject to any proposed changes.*

THE CONSTITUTION

ARTICLE III. MEMBERS

There shall be the following categories of membership: Life Fellows; Fellows; Life Members; General Members; *Life Associate Members*; Associate Members, Members-in-Training; Provisional Members; Distinguished Fellows; Honorary Fellows; Corresponding Fellows; Corresponding Members; *Inactive Fellows*; and Inactive Members, as defined in the By-Laws.

THE BY-LAWS

CHAPTER ONE. MEMBERS

3. To maintain membership in good standing all members, *except as exempted by the Board*, are required to participate in continuing education according to the standards of the Association.

7. Fellows shall have been General Members for at least [five] *eight* years and shall have made a significant contribution to the field of psychiatry. At its discretion the Board, upon recommendation to the Membership Committee, may waive the requirement for [five] *eight* years as a General Member. The criteria and procedures for selection and nomination of General Members for Fellowship shall be established by the Board and the Membership Committee and shall apply uniformly for all District Branches.

8. Life Fellows and Life Members shall be those in their respective categories [who have had 30 years of active membership in the Association.] *whose years of active membership in the Association plus age at the start of the fiscal year shall equal 95.*

9. Life Associate Members shall be those Associate Members whose years of active membership in the Association plus age at the start of the fiscal year shall equal 95.[1]

[12] *13.* Inactive [Members or Fellows] *members of any category* shall be those whom the Board has, for sufficient reason, excused from paying dues.

Members of any category who are placed in inactive status by the Board shall be excused from paying dues and shall not receive credit toward the "95" formula while that member is in inactive status. Active members may be granted waiver of dues by the Board for sufficient reason, and such members shall receive credit toward the "95" dues exemption formula for those years that the member is in the dues waiver status.

CHAPTER EIGHT. PRIVILEGES AND RESPONSIBILITIES

5. The voting membership may initiate referenda or change an action of the Board by the following procedure: A petition signed by at least 200 voting members shall be submitted to the Secretary no later than December [15] *1* to be voted on in the mail ballot in the following year. A statement from the petitioners setting forth the reasons for the action and a statement from the Board shall accompany the ballot. A simple majority of 40 percent of the eligible voting members shall be required for approval.

6. Every Fellow, General Member, Associate Member, and Member-in-Training shall pay both dues and assessments as determined by the Board and the District Branches. All other categories of membership shall be exempt from paying dues to both the Association and its District Branches. *A dues paying member shall be exempt from paying such dues when the sum of that member's years of active membership in the Association plus the member's age at the start of the fiscal year shall equal 95.*

Such dues will include amounts allocated to subscriptions for the *American Journal of Psychiatry* and for *Psychiatric News*. The membership application shall contain a provision for allocation of a specific portion of the dues to pay the cost of periodical subscriptions to these publications. *Dues exempt members shall pay a reduced subscription rate for these publications if such members desire to receive them.*

CHAPTER NINE. VOTING

1. Nominations for national office except that of Area Trustee shall be made by: (a) the Nominating Committee; or (b) by a petition signed by 200 or more members eligible to vote. Nominations for Area Trustee be made by: (a) procedures established by the Assembly; or (b) by a petition signed by 50 or more members of that Area who are eligible to vote. Nominating petitions must be filed with the Secretary before December [15] *1* of the year immediately previous to the one in which the nominee would be elected.

[1]If approved, this will become new Section 9; sections now numbered 9–12 would be renumbered 10–13, respectively.

Report of the Joint Conference Committee on the Key Conference Recommendations

ONE OF THE fundamental propositions emanating from the Key Conference held in March 1975 was that the Assembly become the policy-making body of the Association. Following the Key Conference, the Assembly and the Board established a series of components that have worked in concert to study a variety of organizational models and to prepare constitutional revisions that would implement the transfer of this responsibility. Early models presented bicameral systems that posed both legal and efficiency problems.

The Joint Conference Committee on the Key Conference Recommendations (JCC) has prepared an organizational model that is incorporated in the proposed Constitutional revisions. The JCC believes this model to be the best practical way to achieve the transfer of the management of the Association to the Assembly. Although this model is unicameral, checks and balances are provided for in a variety of ways, including the establishment of an Executive Board that is nationally elected by the membership. The JCC neither recommends nor opposes this change in governance but endorses this model should the membership wish to achieve the transfer of policy-making responsibility to the Assembly.

In October 1977, the Assembly voted to approve the JCC model for reorganization and to recommend it to the Board of Trustees for consideration and further action. In November 1977, the Reference Committee endorsed the recommendation of the Assembly. In December 1977, the Board of Trustees voted to present the Constitutional revisions prepared by the JCC to the membership at the 1978 annual meeting so that the amendments to the Constitution could be included on the ballot in 1979. The Committee on Constitution and By-Laws reviewed these amendments to ensure that they are in proper order. The membership will decide in the 1979 election whether the Assembly should assume the management of the Association.

COMPARISON OF PRESENT AND PROPOSED GOVERNANCE SYSTEMS

Referral and Reporting Processes

APA's annual cycle of activities is essentially from May through April. At the annual meeting in May, new officers and members of the Board and the Assembly assume their duties; changes occur in the composition of the Board, Reference Committee, and the councils and their components, as well as in the composition of the Assembly, its Executive Committee, the area councils and the district branches.

Recommendations for action or study come from both within and outside APA and may come to the Board and Assembly throughout the year. Internally, proposals for action or study may come from individual members, components of the Board (committees, commissions, councils, task forces, etc.) or components of the Assembly (district branches, area councils, Assembly or local committees and task forces, etc.). Externally, there may be pressures from legislation in process or enacted, requests from other organizations, and national or international trends and activities.

Proposals for action or study that are processed through the present governance system normally follow an established referral and reporting back process; if emergency action is needed, there are ways to expedite the process, including the emergency mechanism that involves the President, Speaker, and the Medical Director, together with appropriate consultants.

Present System

Board structure. Referrals to and reports from councils and their components are handled via the Reference Committee. Constitutional committees, ad hoc committees, and other special components have direct referral from and reporting to the Board.

Assembly structure. Referrals to and reporting from Assembly components (area councils, district branches, committees and task forces of the Assembly and counterpart committees and task forces of the area councils and district branches) are handled directly by the Assembly and its Executive Committee.

Interfaces. The Speaker is a member of the Board and its Executive Committee; the Speaker-Elect is a member of the Reference Committee. They report the recommendations of the Assembly to these bodies and report their actions to the Assembly. Each council has an Assembly liaison assigned who reports the activities of the council to the Assembly. There are some Assembly liaisons to components of councils. There are also joint commissions or conference committees that report to both the Board and Assembly. A recommendation coming to the attention of the Assembly or the Board in May can be immediately referred to each other since both bodies meet during the annual meeting. Issues may be studied at the same time by Assembly and Board components, or reports and recommendations may be studied when going through the approval process, i.e., by councils and components as well as area councils and district branches or subordinate components thereof.

Thus, in the present system, there are two organizational structures that serve for referral and reporting back with some interface and concomitant study throughout the annual cycle of APA activities.

Proposed Governance System

The Assembly would meet in May and December; the Executive Board (with all of the powers of the Assembly between meetings of the Assembly) would meet approximately four times during the course of the year.

The Executive Board would receive reports from the Reference Committee (which would also collate the reports of the nine councils and their components) and from the Constitutional committees, commissions, and special components. The Executive Board in turn would serve a triage function for the Assembly. It would act on some reports within policy previously established by the Assembly; other reports would

This is an edited version of the report of the Joint Conference Committee on Key Conference Recommendations to the annual business meeting in Atlanta, Ga., May 9, 1978.

be referred to other components for further study, recommendations, or action. In those instances when the Executive Board required policy elaboration, modification, or clarification, it would seek it from the Assembly. All actions of the Executive Board would be reported to the Assembly for ratification.

The Assembly would receive reports from two sources. The Executive Board would report to the Assembly as outlined above. The other source would be the area councils (which would also collate district branch reports). The Assembly Agenda Committee would receive all reports to the Assembly in order to organize them for consideration by the Assembly and generally facilitate the agenda process.

Thus, there would be one governance system for referral and the implementation of all Association component reports, namely the Assembly and its Executive Board. An important check and balance would be the ability of the Executive Board to delay the implementation of a policy decision by the Assembly until the next meeting of the Assembly. The Executive Board would not have the power to further delay or obviate a decision by the Assembly. The President, Speaker, and Medical Director could take emergency action when necessary.

Policy and Management

Assembly and Executive Board. The proposed governance system assigns ultimate responsibility for priority and policy decisions to the Assembly, a broadly based body with representatives who are close to their constituents, both personally and geographically, and who are accountable to them through a recall process. An Executive Board, including geographic representatives derived independently of the Assembly system, would exercise all of the powers of the Assembly in the interim between meetings of the Assembly but would report its actions to the Assembly for ratification. There would not be a Board of Trustees or an Assembly Executive Committee.

Board of Trustees and Assembly. In the present system, the Board of Trustees has the right and responsibility for policy-making decisions and management, with its Executive Committee assigned this power between meetings of the Board. Increasingly, the Assembly has played a strong role in policy-making decisions, since all position statements of the Association must be approved by both the Board and Assembly. In addition, at the annual business meeting the Assembly may by two-thirds majority vote reverse an action of the Board.

Components

Under the proposed system, components of the Association would primarily serve to aid the Assembly in its policy-making function and the Board in carrying out the policies of the Association. Under the present system, components of the Association primarily serve the policy-making and implementing function of the Board and its Executive Committee.

Assembly Agenda Committee. In the proposed system, the AAC would serve to facilitate the functioning of meetings of the Assembly. It would not have any of the powers of the Assembly in the interim between meetings of the Assembly and would probably meet the day before the first plenary session at any meeting of the Assembly to rank in order of importance the action and information items referred to the Assembly. The *Operations Manual* of the Association would specify the procedures to be followed by the AAC.

Constitutional committees. In the proposed system, the constitutional committees (Ethics, Membership, Nominating, Constitution and By-Laws, Budget, Tellers, and Reference) would report to the Executive Board, which would assume responsibility for referring matters requiring policy decision to the Assembly. The Assembly would have no need for separate committees of its own. In the present system, the constitutional committees report to the Board of Trustees for action on their recommendations. Some constitutional committees have had liaison and joint meetings with their Assembly counterparts (such as the Membership Committee), so that a broader consensus is reached before recommendations are sent to the Board of Trustees. There has occasionally, however, been duplication of effort in their work and the work of the Assembly committees.

Councils and Their Components

In the proposed system, the council chairpersons (and perhaps chairpersons of other components such as commissions) would be members of the Reference Committee, where the recommendations of components administratively housed within each council would be brought for recommended action or referral. The Reference Committee would report its recommendations for action to the Executive Board, which would have the responsibility for taking action or for referring the matter to the Assembly for policy clarification. If an interim action is taken, it would be presented for ratification by the Assembly. The responsibility for formulating position statements of the Association would rest with the Assembly. In the present system, council chairpersons also serve on the Reference Committee and present the recommendations of their subordinate components for action or referral; the Reference Committee reports to the Board of Trustees for action on their recommendations. The Assembly must be consulted and its approval obtained only if the Board of Trustees is establishing a position statement for the Association.

Reference Committee. In the present governance system, there is increased reporting to the Reference Committee by components that interact or overlap with the components within the council structure. The proposed system provides for additional members of the Reference Committee to be specified in the *Operations Manual* of the Association.

Commissions, Boards, and other special components outside the council structure. In the proposed system, these components would report to the Executive Board, which would have responsibility for interim action and reporting to the Assembly. There would be no need for joint commissions with dual representation. In the present system, there are joint commissions that report to both the Board and Assembly. All others report to the Board for action, although informational reports and requests to comment on their recommendations are often made to the Assembly for discussion by its components.

Area councils. Both the present and the proposed governance systems provide for geographic areas. The present *Procedural Code* of the Assembly defines the purpose, composition, and rules of operation of area councils. The *Operations Manual* of the Association would spell these out in the new system.

In the proposed system, the area councils would report to the Assembly through the area representative and to the Executive Board through the Vice-President from their respective areas. Area representatives would have a vote in the Assembly and area Vice-Presidents would have a vote both on the Executive Board and in the area council. The Assembly alone would make the position statements of the Association. In the present system, the area councils report only to

the Assembly and its Executive Committee; area representatives vote in the Assembly and Assembly Executive Committee; area Trustees have a vote only on the Board of Trustees and its Executive Committee.

District branches. In the proposed system, district branches "shall be established, continued, or dissolved according to the *Operations Manual* of the Association." In the present system, district branches "shall be established, continued, or dissolved according to the *Procedural Code* of the Assembly."

RATIONALE FOR DEVELOPING THE PROPOSED SYSTEM

In the original planning for the Key Conference and in the deliberations during the conference, a number of objectives were identified. Many of these, subsumed under the general heading of improvements in governance, have occupied most of the attention of the JCC during the past two years. Initially some of these, such as democratization and efficiency, seemed to be conflicting or even had mutually exclusive objectives. It became apparent that much could be accomplished by rearranging the sequence of some operations and organizing components into a unified system with elimination of the present bipartite, fragmented, and at times oppositional system. The JCC sought ways to accomplish unification of the system with minimal disturbance of the operations of the Association and with minimal, if any, increase in cost.

The following are some of the more significant objectives for improving the governmental process: 1) development of a cohesive governmental structure allowing for orderly process that could be more easily understood by the members and be more accessible to them; 2) improvement in the democratic process—giving the individual members an opportunity for greater voice and influence in the direction of the Association; 3) provision for more effective accountability of the members of the governing bodies, particularly those ultimately responsible for the direction of the Association; 4) development of an effective means for establishing priorities among the many concerns and activities of the Association; 5) improvement in efficiency, with particular reference to cost effectiveness; and 6) provision for timely and effective action by the Association, including a mechanism for emergency action.

The JCC recognizes that major improvements have been made in the democratic process in the Assembly through the requirements for 1) election by mail ballot; 2) petition nomination; 3) mandatory reporting to constituents; 4) increased representation for minorities; and 5) the possibility for recall election of Representatives and Deputy Representatives.

In addition, the JCC suggests the following improvements: 1) more proportionate representation in very large district branches by providing additional representatives with no increase in voting strength (such representation would be based on a numerical factor, for example, a Representative and Deputy Representative for each 500 members); and 2) automated voting procedures for voting by voting strength to make the process less time-consuming and cumbersome.

ADVANTAGES OF THE PROPOSED GOVERNANCE SYSTEM

The JCC has prepared a set of recommended changes in the Constitution reflecting these stated goals, which it believes may accomplish the following: 1) a unified governance system, with all subordinate components reporting to one policy-making body, the Assembly; 2) an orderly policy process, with accountability and responsibility clearly identified; 3) a small, efficient executive body (Executive Board), composed of popularly elected officers derived independently from the Assembly election system, which would handle the affairs of the Association in the interim between meetings of the Assembly and would be required to have its actions ratified by the Assembly; and 4) the assignment of ultimate responsibility for priority and policy decisions to the Assembly, a broadly based body with representatives who are close to their constituents and who are accountable to them through a recall process.

SUMMARY

The JCC has prepared recommendations for change in the governance system of the Association that would transfer policy-making responsibility to the Assembly. These have been translated into constitutional amendments by the Committee on Constitution and By-Laws and were presented to the membership at the 1978 annual meeting so that they can be voted on in 1979. These constitutional amendments will be presented for vote en bloc, since it would not be possible to implement portions of them.

The JCC neither recommends nor opposes this change in governance but endorses this model should the membership wish to achieve the transfer of policy-making responsibility to the Assembly. The JCC hopes that the Association will continue to evolve and change in response to need. To aid in the process, the JCC strongly recommends that a committee or commission be established with responsibility for assessing the operations of the Association and making recommendations for change in the future.

The JCC further emphatically recommends the establishment of a long-range planning committee for our Association. Long-range planning is a way to determine both our long-term objectives and our priorities in achieving them. It can provide us with a sense of direction, helping us to focus our energies and resources on those areas which have the most long-term significance, and at the same time provide us with a perspective that will allow us to make active, not reactive, decisions in the context of the total situation.

BRUCE ALSPACH, M.D.

Am J Psychiatry 135:10, October 1978

1977 Annual Report of the American Board of Psychiatry and Neurology, Inc.

OFFICERS AND MEMBERS OF THE BOARD

At its business meeting on October 16, 1977, the Board elected the following officers: Dr. Chester M. Pierce, President; Dr. Robert J. Joynt, Vice-President; Dr. Richard M. Steinhilber, Secretary; Dr. Melvin D. Yahr, Treasurer; and Dr. James N. Sussex, Executive Committee Member at Large.

Dr. Leonard Berg was elected to succeed Dr. Thomas W. Farmer, who had completed his second term of four years in December 1977. Drs. Marc Hollender, Richard Steinhilber, and Robert Joynt were elected to serve second terms of four years.

The 1978 Directors of the American Board of Psychiatry and Neurology, Inc., are as follows:

Nominated by the American Medical Association: Dr. Robert J. Joynt* (term expires December 1980), Dr. Richard M. Steinhilber* (term expires December 1981), Dr. Thomas R. Johns (term expires December 1978), Dr. James N. Sussex (term expires December 1978), and Dr. S. Mouchly Small (term expires December 1979).

Nominated by the American Neurological Association: Dr. Leonard Berg (term expires December 1981), Dr. Melvin D. Yahr (term expires December 1978), Dr. Patrick F. Bray (term expires December 1979), and Dr. John R. Calverley (term expires December 1980).

Nominated by the American Psychiatric Association: Dr. Marc H. Hollender* (term expires December 1981), Dr. Chester M. Pierce* (term expires December 1978), Dr. Alfred M. Freedman (term expires December 1978), Dr. Donald G. Langsley (term expires December 1979), and Dr. Richard I. Shader (term expires December 1980).

Nominated by the American Academy of Neurology: Dr. Audrey S. Penn (term expires December 1978) and Dr. Dewey K. Ziegler (term expires December 1978).

EXAMINATIONS

Part I

At the Part I (written) examination on April 11, 1977, 1,469 psychiatrists were tested; 1,215 (82.7%) passed, 254 (17.3%) failed. A total of 391 neurologists were examined; 312 (79.9%) passed, 79 (20.1%) failed.

For the 1978 Part I examination, 2,091 psychiatrists and 543 neurologists have accepted assignment. (See table 1 for the number of candidates declared eligible and notified for Part I in 1977 and table 2 for a comparison of these figures with other years).

TABLE 1
Number Declared Eligible and Notified for Part I Written Examination April 11, 1977

Item	Psychiatry	Neurology	Total
Number declared eligible and notified	1,995	424	2,419
Number who declined or withdrew	325	7	332
Number remaining for written examination as of March 1977	1,670	417	2,087

TABLE 2
Five-Year Comparison of Number Declared Eligible and Notified for Part I Examination

Item	1973	1974	1975	1976	1977
Total eligible	1,999	2,098	3,146	3,113	2,419
Total who accepted examination	1,381	1,821	2,408	2,391	2,087
Total who actually appeared	1,205	1,627	2,240	2,191	1,860

Part II

There were 333 candidates—258 psychiatrists and 75 neurologists—present at the Part II examination given in Seattle, Wash., February 7–8, 1977 (see table 3). A total of 617 candidates—474 psychiatrists and 143 neurologists—were tested in Washington, D.C., April 18–19, 1977 (see table 4). A total of 444 candidates—353 psychiatrists and 91 neurologists—were tested in Kansas City, Mo., June 6–7, 1977 (see table 5). One of the Board's largest Part II examinations to date was held in Philadelphia, Pa., October 16–17, 1977. A total of 1,095 candidates were tested—942 psychiatrists and 153 neurologists (see table 6).

Future examination sites for 1978 include Los Angeles, Calif. (April 17–18), Cincinnati, Ohio (June 5–6), and Boston, Mass. (November 6–7).

CHILD PSYCHIATRY

Members of the 1978 Committee on Certification in Child Psychiatry are Dr. Richard S. Ward, chairperson, Dr. Jeanne Spurlock, vice-chairperson, Dr. Joseph Green, secretary, Dr. Norman Bernstein, Dr. Norbert Enzer, Dr. Irving Philips, and Dr. Michael I. Cohen, liaison from the American Board of Pediatrics.

At its March 14–15, 1977, examinations in Chicago, the committee examined a total of 136 candidates. A total of 96 candidates (70.6%) passed. Of the remaining 40 candidates, 26 (19.1%) conditioned the examination, and 14 (10.3%) failed.

This is an edited version of the report of the American Board of Psychiatry and Neurology, Inc., to the American Psychiatric Association in Washington, D.C., April 1, 1978.

*These Directors are serving their second term of four years and are not eligible for reappointment.

TABLE 3
Analysis of Performance of Group Who Took Part II Examination in Seattle, Wash., February 7–8, 1977, by Specialty and Place of Graduation

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	333	100.0	258	77.5	64	19.2	11	3.3	269	80.8	64	19.2
Results												
Pass	193	58.0	148	57.4	39	60.9	6	54.5	161	59.5	32	12.5
Fail	40	12.0	29	11.2	7	10.9	4	36.4	32	11.9	8	12.5
Fail (must reapply)	11	3.3	10	3.9	1	1.6	0	0.0	3	1.1	8	12.5
Condition	89	26.7	71	27.5	17	26.6	1	9.1	73	27.1	16	25.0

TABLE 4
Analysis of Performance of Group Who Took Part II Examination in Washington, D.C., April 18–19, 1977, by Specialty and Place of Graduation

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	617	100.0	474	76.8	121	19.6	22	3.6	440	71.3	177	28.7
Results												
Pass	376	60.9	288	60.8	71	58.7	17	77.3	299	67.9	77	43.5
Fail	209	33.9	163	34.4	44	36.4	2	9.1	123	28.0	86	48.6
Fail (must reapply)	32	5.2	23	4.8	6	4.9	3	13.6	18	4.1	14	7.9

TABLE 5
Analysis of Performance of Group Who Took Part II Examination in Kansas City, Mo., June 6–7, 1977, by Specialty and Place of Graduation

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	444	100.0	353	79.5	80	18.0	11	2.5	254	57.2	190	42.8
Results												
Pass	279	62.8	209	59.2	61	76.3	9	81.8	184	72.5	95	50.0
Fail	32	7.2	29	8.2	3	3.7	0	0.0	13	5.1	19	10.0
Fail (must reapply)	133	30.0	115	32.6	16	20.0	2	18.2	57	22.4	76	40.0

TABLE 6
Analysis of Performance of Group Who Took Part II Examination in Philadelphia, Pa., October 16–18, 1977, by Specialty and Place of Graduation

	Total		Psychiatry		Neurology		Child Neurology		U.S. Graduates		Foreign Graduates	
Item	N	%	N	%	N	%	N	%	N	%	N	%
Candidates	1,095	100.0	942	86.0	115	10.5	38	8.5	673	61.5	422	38.5
Results												
Pass	651	59.4	555	58.9	73	63.5	23	60.5	464	68.9	187	44.3
Fail	361	33.0	319	33.9	28	24.3	14	36.8	172	25.6	189	44.8
Fail (must reapply)	83	7.6	68	7.2	14	12.2	1	8.7	37	5.5	46	10.9

The committee's 1978 examination was held February 27–28, 1978, in New Orleans for approximately 170 candidates. The committee continued to offer its "retake option" in the 1978 examination. As in 1977, this option was made available to reexaminees as well as those candidates taking the examination for the first time. Under this option, candidates who feel they have performed badly in an oral section have the opportunity to repeat one oral section during the examination. This option does not apply to the committee's written examination.

The committee agreed at its September 1977 business meeting that a pass on the child psychiatry written examination would be valid for a period of seven years beginning with the February 1978 examination.

The committee also decided to experiment with allowing those candidates who fail only the written examination to retake that examination in their own geographic region. The proctor for such an examination would have to be a known, reliable child psychiatrist in a center near the candidate's residence but not at the candidate's own institution. Such

an examination would be given on the same day as the oral examinations for all other child psychiatrists.

Under the leadership of Dr. Irving Philips, the committee is currently engaged in a project designed to involve child psychiatry training directors in the certification process. In the proposed model for the project, one aspect involves utilization of a questionnaire that asks the training director to grade the trainee as to how the training director feels the trainee will perform on the child psychiatry certification examination. Training directors would also be asked to grade the trainee's performance during the training program. Plans call for gathering data that could be transmitted to child psychiatry training directors pointing out similarities or differences between the training director's ratings and the actual performance. This would also give the committee some basic data on how reliable the training directors are in evaluating residents.•

BOARD DECISIONS AND ITEMS OF INTEREST

The following major policy decisions were enacted by the Board in 1977:

1. Effective November 1, 1977, the following criteria for admission to examination are applicable to those applicants with foreign training qualifications: the candidate must have had full-time specialty training in a manner equivalent in length of time and content to that required for U.S. training; the candidate must successfully complete a certification examination in the country of training in the specialty in which the physician is seeking admission to examination; the candidate must possess an unlimited license to practice medicine in a state or commonwealth of the United States or a province of Canada; and the candidate must have achieved certification within the past seven years, and if certification was achieved more than 7 years ago, then the candidate must have had one year of clinical practice in a setting in which his/her professional work has been observed by at least two ABPN diplomates in that specialty and documented by two letters of sponsorship from such diplomates.

2. Effective November 1, 1977, for those candidates who have completed the first postgraduate year of training in a training center located outside the United States or Canada the following criteria will be acceptable for admission to examination: the candidate must have completed a full year of supervised postgraduate training equivalent to that required by the Board for United States and Canadian graduates as evidenced by appropriate documentation. (This documentation should consist of a letter of verification from the institution at which the training was taken and from the person who was in charge of the training.) The burden for verification for this year rests with the candidate.

3. Beginning at the October 1977 Part II examination, the previously administered one hour of oral examination in Neurology for Psychiatry candidates will be replaced by a written examination. The written examination is based on a series of audiovisual vignettes of neurologic patients. Each vignette will last for approximately three to four minutes and will be followed by written multiple-choice examination questions pertinent to the brief patient presentation.

4. The Board approved the use of an audiovisual examination in child neurology to substitute for one hour of live patient examination in child neurology for candidates for Certification in Neurology with Special Competence in Child Neurology. The substitution went into effect at the Board's October 1977 Part II examination.

5. The Board agreed to accept the proposal of the Credentials Committee that three years of training in general psychiatry could include a pattern of two years in general psychiatry and one year in child psychiatry provided that two years of this training is spent in a single approved program.

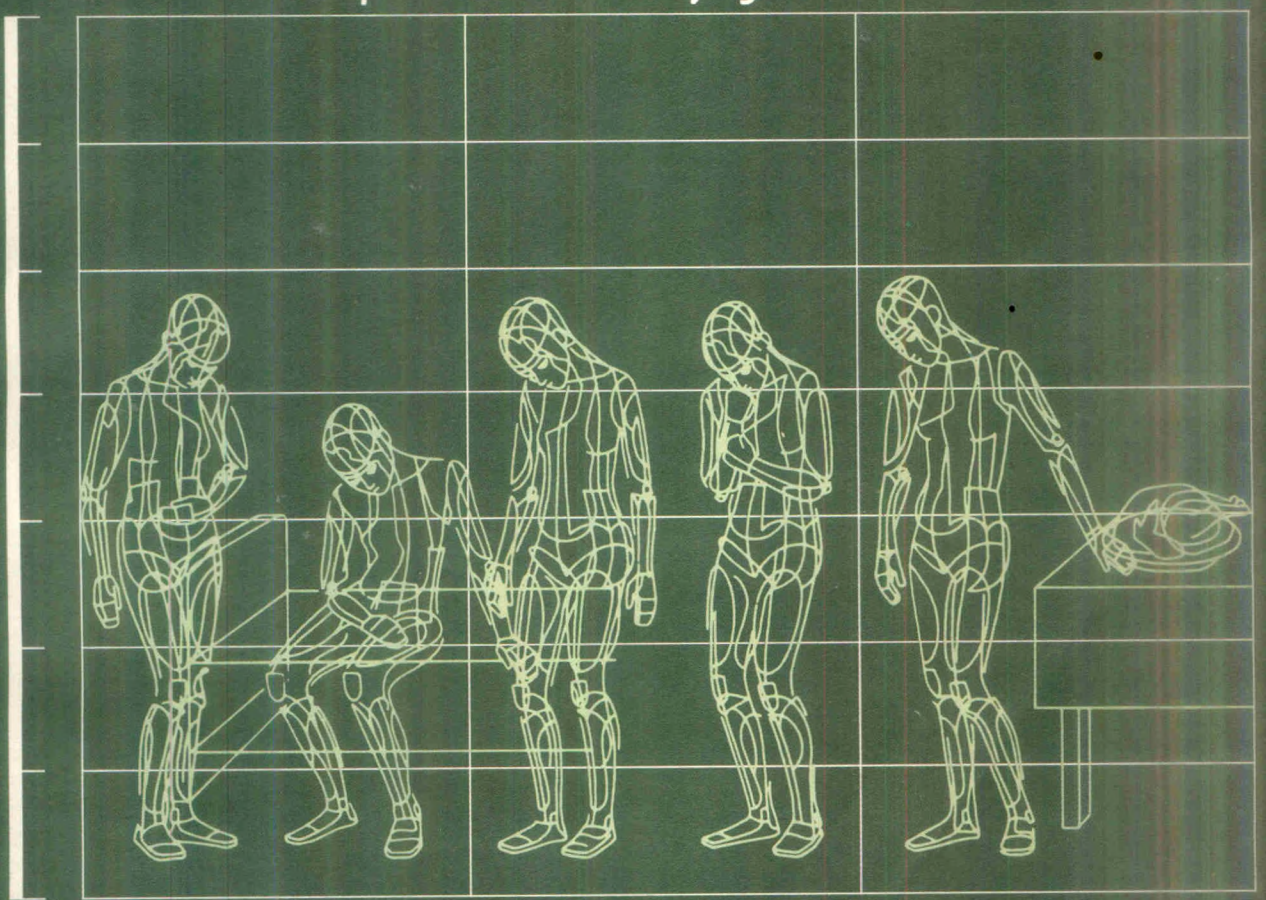
6. The Board agreed that as of January 1, 1978, the per diem for Part II examiners be raised from $50 to $65.

At its November 1977 policy meeting the Board took the following significant actions: 1) reaffirmed its position against a primary Board of Emergency Medicine; 2) agreed to establish a task force to initiate an audiovisual written examination in psychiatry for neurologists; 3) agreed to confer the title "Member of Test Committee of ABPN" on consultant members of ABPN examinations committees and to issue a certificate as a form of recognition to those consultants who have served on the Part I Examination Committee and on the Neurology for Psychiatrists Audiovisual Written Examination Committee; 4) agreed that the grading system for the Part II examination be revised beginning with the November 1978 examination. The new grading system to be used would be high pass, pass, conditional, and fail; 5) decided to include a release form on the Part I application, which would ask the candidates to authorize release of examination results to training programs; 6) authorized the Executive Committee and Executive Director to seek out a research associate; 7) agreed to distribute the following policy statement to appropriate individuals. "Individuals who contribute questions, videotapes, or other subject material for ABPN examinations are reminded that they must avoid explicit discussion of that material in seminars, review courses, or other instructional procedures used by candidates to prepare themselves for the examination, lest the principles of conflict of interest and unfair advantage to some candidates be applicable"; 8) assumed sponsorship of a proposal suggested by Dr. Donald Langsley in conjunction with the Board's Research and Development Committee. The proposal would compare evaluation of residents by training directors during the training period with subsequent performance on Board examinations; and 9) agreed to draw up a statement regarding an ethical code for ABPN Directors.

LESTER H. RUDY, M.D.
Executive Director

Within one week
A better response
For twice as many patients

compared to amitriptyline alone



Limbitrol®

For rapid relief of symptoms
in moderate to severe
depression and anxiety

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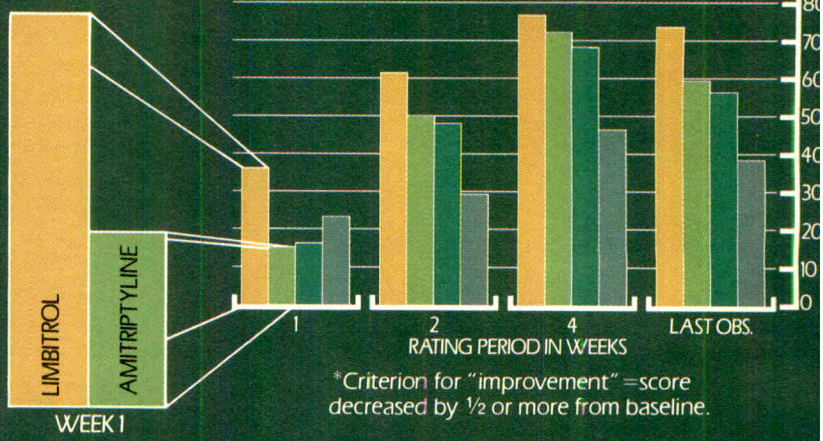
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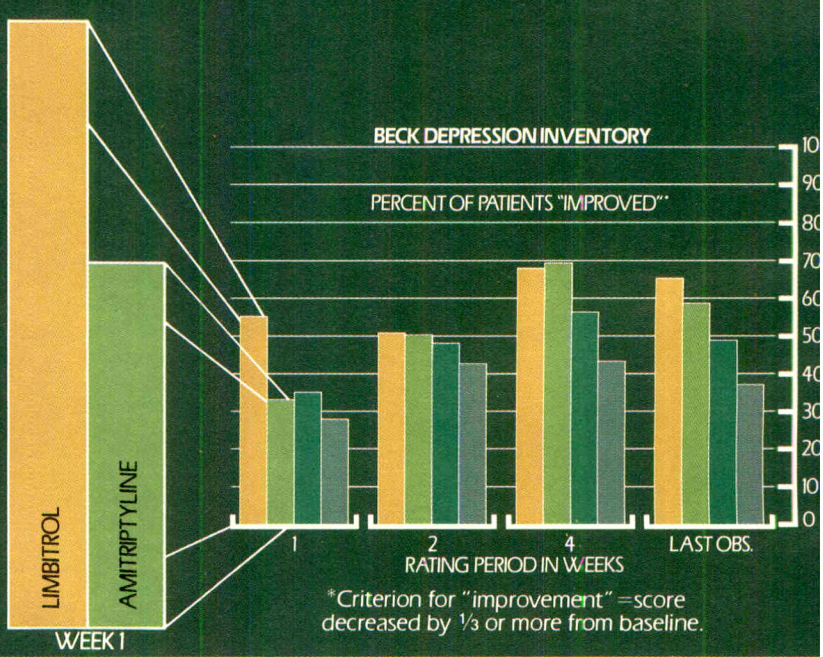
Within one week Twice as many patients improved–soone

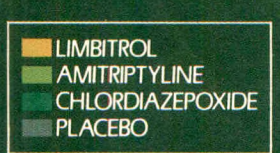
In a rigidly controlled nationwide multicenter study

The rating scales agree

- ☐ Patients respond faster
  - ☐ Limbitrol—in the first week approximately 2/3 of those who improved had already done so
  - ☐ Amitriptyline—only about 1/3 of those who eventually responded had done so by week one
- ☐ High correlation among all major indices employed including Physician's and Patient's Global Evaluations



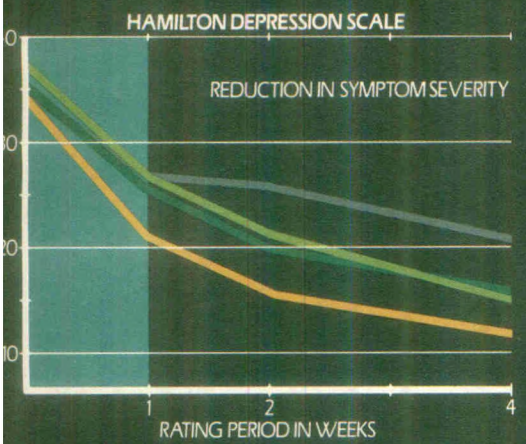




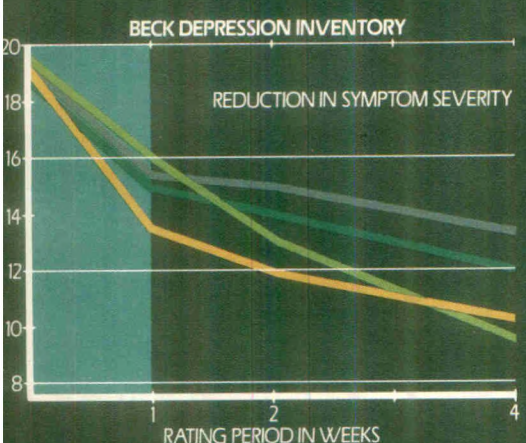
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nd better than with amitriptyline alone



Reduction in severity of symptoms based on mean observed value at each rating period.



Reduction in severity of symptoms based on mean observed value at each rating period.

The symptoms subside

Hamilton Depression Scale Items

- ☐ insomnia
- ☐ psychic anxiety
- ☐ somatic anxiety
- ☐ agitation
- ☐ suicidal ideation
- ☐ worthlessness

Beck Depression Inventory Items

- ☐ pessimism
- ☐ dissatisfaction
- ☐ guilt
- ☐ social withdrawal
- ☐ anorexia

By week one, Limbitrol patients' symptoms had, on average, improved to a point not reached by amitriptyline patients until week two.

The data presented are based on a rigidly controlled nationwide multicenter study of 279 patients with a diagnosis of primary depression with symptoms of depression and anxiety of moderate to severe degree.

Provides the well established antidepressant effect of amitriptyline with the proven antianxiety effect of Librium® (chlordiazepoxide HCl)

Limbitrol®

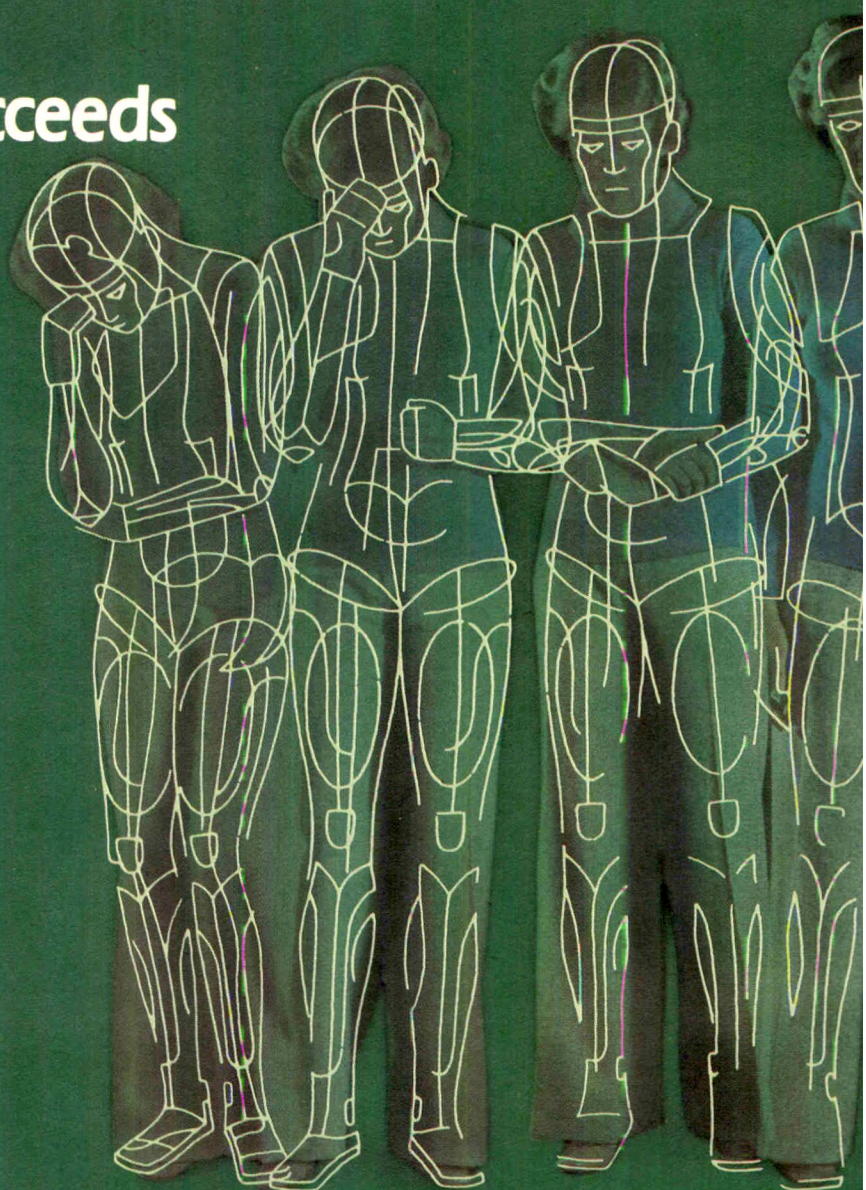
For rapid relief of moderate to severe depression and anxiety

A change for the better comes sooner–within one week

In a rigidly controlled nationwide multicenter study

The therapy succeeds

- ☐ Better patient compliance
- ☐ Lower dropout rate
  - ☐ Despite a higher incidence of drowsiness, the dropout rate due to side effects was lower with Limbitrol than with amitriptyline alone (Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)
  - ☐ The dropout rate due to therapeutic failure was lower with Limbitrol (Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)



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Please see last page of this advertisement for summary of product information.



How to initiate and maintain therapy

Select dosage strength appropriate for each patient

☐ Limbitrol 10-25 is recommended for most patients

☐ Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

☐ An initial dosage of three tablets is recommended

☐ Dosage may be increased to six tablets or decreased to two tablets daily as necessary

☐ Once a satisfactory response is obtained, evaluate response, and, if necessary, reduce dosage to smallest amount needed to maintain remission

Utilize dosage options to best accommodate individual patient needs

☐ T.I.D. or Q.I.D., familiar regimens most suited for patients who tolerate medication without undue drowsiness

☐ Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia

☐ Entire dosage *h.s.* to take maximum advantage of the sedative effect

Tablets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)

Tablets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

Limbitrol®

For rapid relief of moderate to severe depression and anxiety

Your guide to patient management

1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances:

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.

Limbitrol®

For rapid relief of moderate depression and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.

Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.

Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

***Usage in Pregnancy:* Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).

Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12. In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.

Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.

This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:

Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.

Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.

Anticholinergics: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.

Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.

Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.

Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.

Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.

Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly.

Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.

Supplied: Limbitrol 10-25 tablets each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and Limbitrol 5-12.5 tablets each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 50.

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

2. THE WORKING PAPERS OF THE 1975 CONFERENCE ON EDUCATION OF PSYCHIATRISTS Paper bound, 432 pages, $12.50.

Sixty two leading psychiatric educators served on seven preparatory commissions, one for each of seven topic areas, and prepared advance working papers as a basis for Conference deliberations. The topics and Commission Chairpersons were: Social Issues and Needs (James N. Sussex, M.D.); Goals and Objectives (Morton F. Reiser, M.D.); The Resident (David R. Hawkins, M.D.); Content, Methodology, Faculty and Environment (Milton Greenblatt, M.D.); Research (Daniel X. Freedman, M.D.); Economics (Herzl Spiro, M.D.); Evaluation and Quality Control (Eric Pfeiffer, M.D.). The Chairpersons, together with the Conference Chairperson, served as the Editorial Board for this volume.

Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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Must the MBD child take a controlled drug in school?



Not if he's taking Cylert® (pemoline) C IV

Children can be cruel. Their teasing about taking noontime medication can cause serious problems for the child with MBD.

Here's how Cylert eliminates this problem:

- Once a day dosage at home
- Eliminates mid-day school dose

In addition, Cylert offers these benefits:

- Avoids ups and downs of drug action brought about by multiple daily dosage
- Control of medication by the parent
- A chewable dosage form
- Less physician paper work (Cylert is in schedule IV)
- Safety and efficacy proven in extensive clinical studies*

*Copy of the Cylert Monograph available to Physicians on written request.

When not to use medication

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school-age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

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Please see next page for Prescribing Information.

Cylert and Cylert Chewable Tablets

(pemoline)

Prescribing Information

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannibalization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

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37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)
75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored)
in bottles of 100 (**NDC** 0074-6088-13)

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A Psychiatric Glossary

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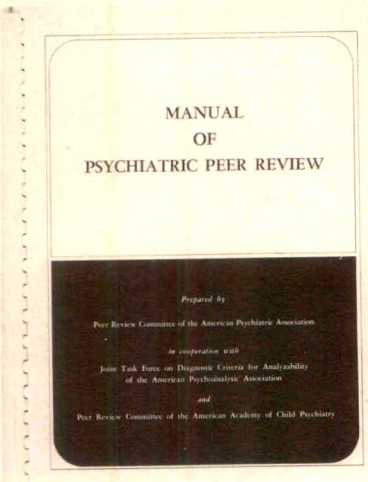
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MANUAL OF PSYCHIATRIC PEER REVIEW

by the Peer Review Committee of the American Psychiatric Association, Donald G. Langsley, M.D., Chairperson in cooperation with the Joint Task Force on Diagnostic Criteria for Analyzability of the American Psychoanalytic Association, William Offenkrantz, M.D., Chairperson, and the Peer Review Committee of the American Academy of Child Psychiatry. Larry B. Silver, M.D., Chairperson.



"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

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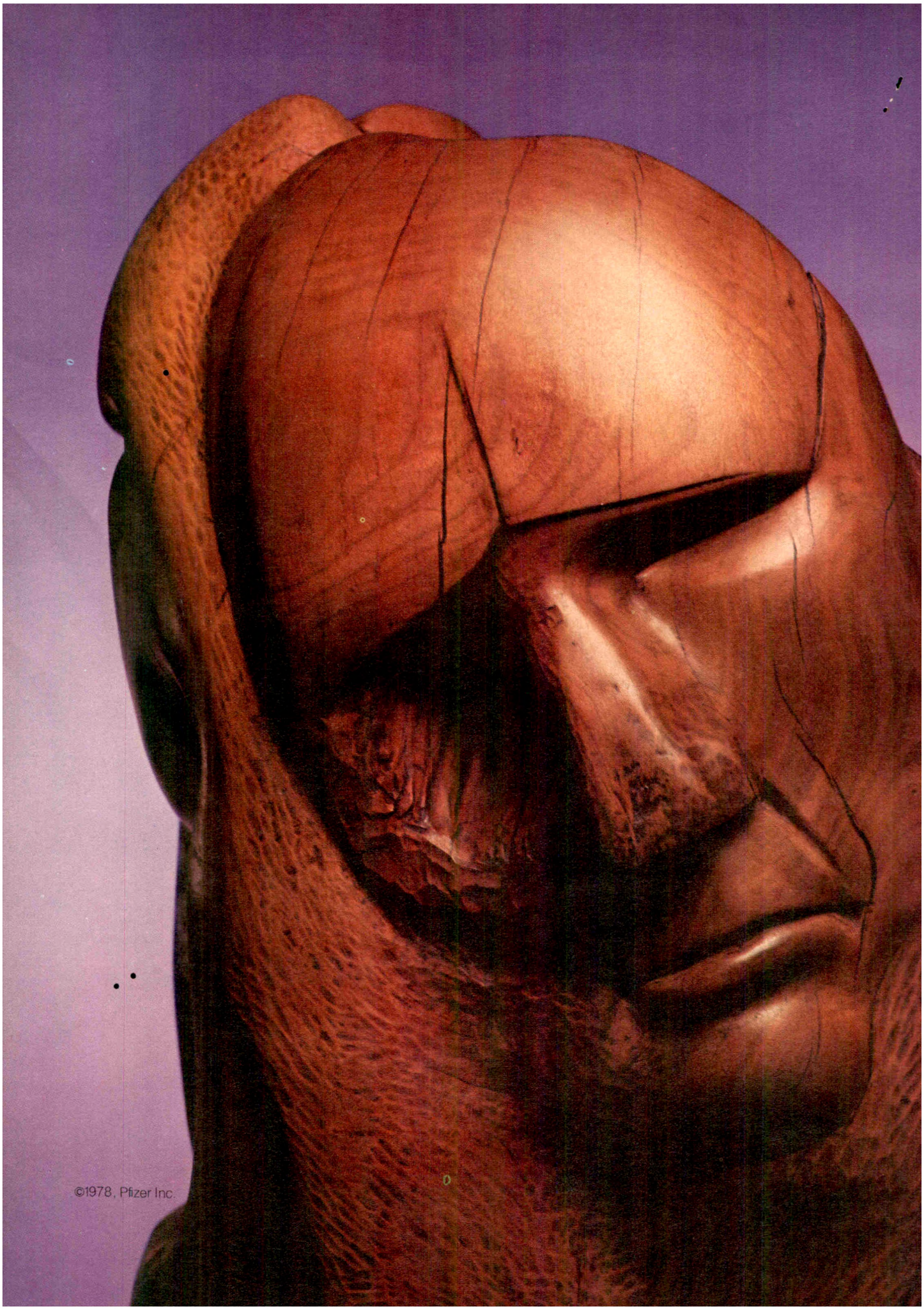
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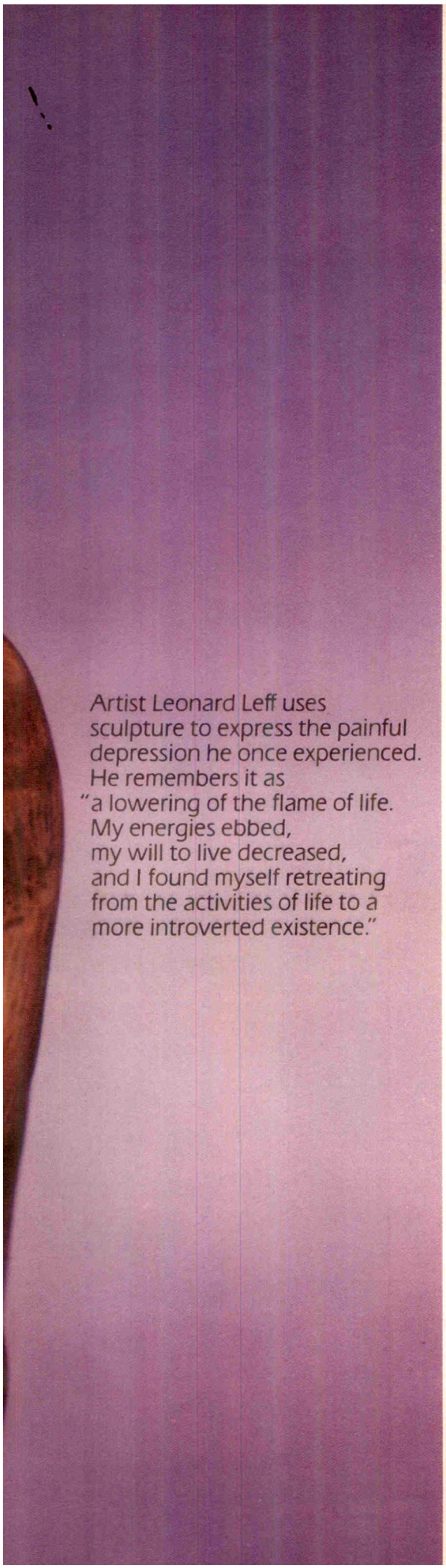
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

WHEN DEPRESSION EXPRESSES ITSELF

SINEQUAN®
(DOXEPIN HCl)

ANTIDEPRESSANT EFFECTIVENESS with convenient once-a-day *h.s.* dosage*

NEW 150-MG CAPSULE†

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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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NEW 150-MG CAPSULE*

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which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, starting daily dose of 75 mg is recommended. Dosage may subsequently be increased decreased at appropriate intervals and according to individual response. The usual optimu dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organ disease, lower doses may suffice. Some of these patients have been controlled on doses as lo as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-d dosage schedule. If the once-a-day schedule is employed the maximum recommended dose 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended f maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effe may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias ar tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressi supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbe The use of activated charcoal has been recommended, as has been continuous gastric lavag with saline for 24 hours or more. An adequate airway should be established in comatose patien and assisted ventilation used if necessary. EKG monitoring may be required for several day since relapse after apparent recovery has been reported. Arrhythmias should be treated with th appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CN symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intr venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine rapidly metabolized, the dosage should be repeated as required. Convulsions may respond standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depre sion. Dialysis and forced diuresis generally are not of value in the management of overdosag due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 m and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg ar 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 m doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Or Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrate at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 m doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted wit approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number carbonated beverages. For those patients requiring antidepressant therapy who are o methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixe together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not wit grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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Enrollment in the Hospital & Community Psychiatry Service brings multiple copies of *Hospital & Community Psychiatry* into member agencies every month, keeping staff up to date on developments and issues in the mental health field, offering new ideas and fresh perspectives, and serving as a useful resource in staff development and training programs.

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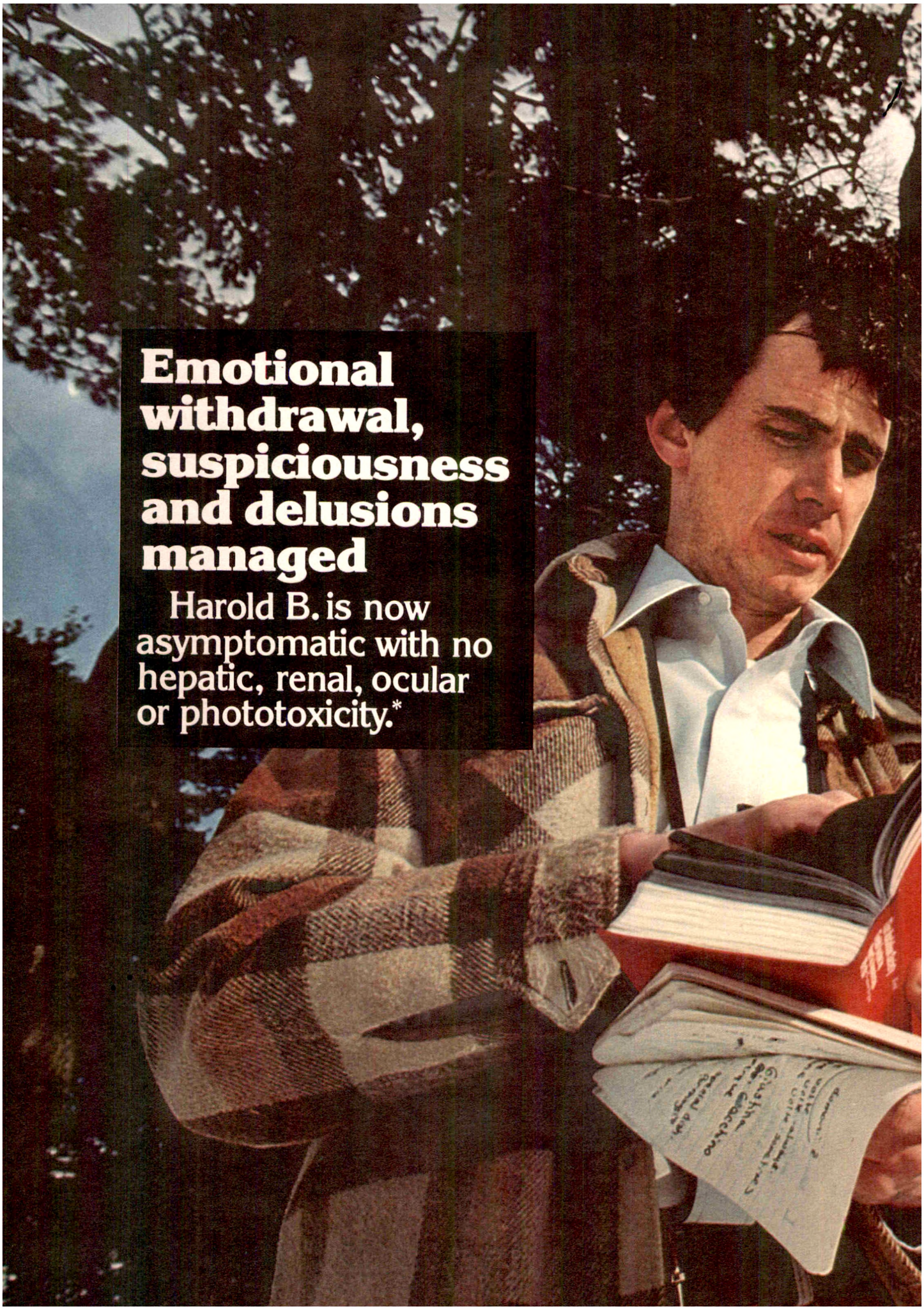
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Emotional withdrawal, suspiciousness and delusions managed
Harold B. is now asymptomatic with no hepatic, renal, ocular or phototoxicity.*



LOXITANE® Loxapine Succinate

Permits rapid management of Harold's suspiciousness and behavioral symptoms

Shy, retiring son forced to join family business after college. Became increasingly immobilized, sitting at desk and staring into space. Occasionally overheard muttering about the "powers killing me." Taken to psychiatrist's office, he was given LOXITANE® Loxapine Succinate capsules 50 mg initially. Scheduled for daily sessions and LOXITANE 20 mg t.i.d. Diagnosis: Acute schizophrenia.

Permits aggressive titration for rapid establishment of optimal symptom management

Short-term treatment goals focussed on symptom control. Within four days, dosage titrated up from 20 mg to 50 mg t.i.d. By the fifth day, Harold was able to visit psychiatrist alone. Delusions and conceptual disorganization began to subside.

Permits sustained symptom management with minimal risk of phototoxicity and other serious adverse effects

During third week, became aware of parental conflict. He began to explore his true career interest as a naturalist. Experienced no phototoxicity despite long hours out-of-doors. LOXITANE reduced to 30 mg t.i.d., with symptom management maintained.

Side effects are usually mild, transitory and easily managed

Consistence in schizophrenic symptom management and tolerance promotes patient compliance

Now, after six months on LOXITANE 20 mg t.i.d. and regular therapeutic sessions, Harold continues his outdoor interests, but has agreed to return part-time to his father's business on a trial basis. He remains under control and free from occurrence of side effects. Much less emotionally withdrawn, but lacks spontaneity.

*Transient liver enzyme changes not definitely related to LOXITANE have been reported. Although not reported to date, possibility of hepatic, renal, ocular, or phototoxicity cannot be ruled out at this time. Not an actual patient, but a simulation to illustrate the action of LOXITANE as reported in clinical studies.

See LOXITANE prescribing information on the following page for indications, warnings and precautions and for more detailed information concerning side effects.

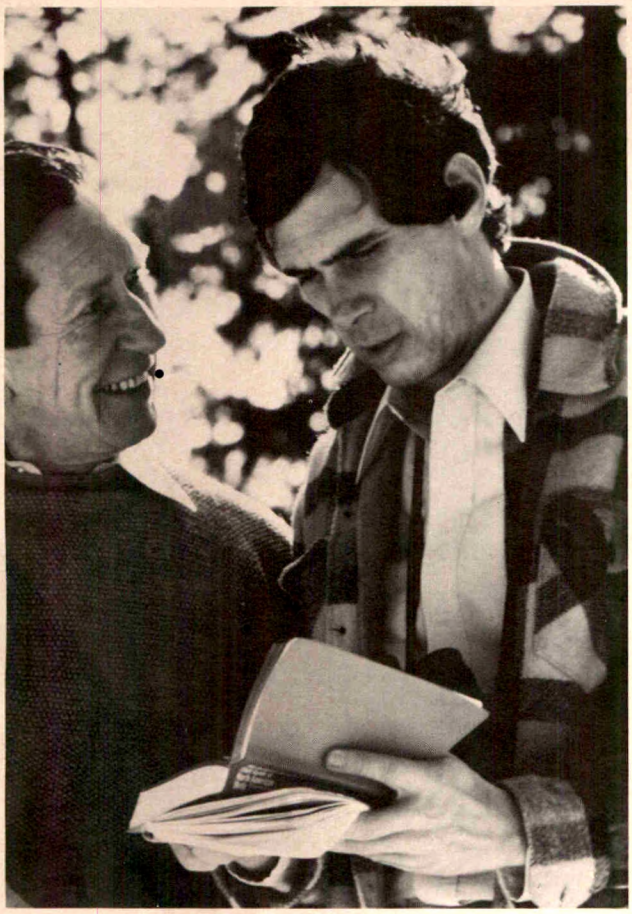
Loxitane®
LOXAPINE SUCCINATE

Lederle

Capsules: 5 mg, 10 mg, 25 mg, 50 mg

Maximum management... minimum risk

Maximum management... minimum risk



Loxitane®
LOXAPINE SUCCINATE
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Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		

Brief Summary
LOXITANE® Loxapine Succinate Capsules
LOXITANE® C Loxapine Hydrochloride Oral Concentrate
INDICATIONS: Manifestations of schizophrenia.
CONTRAINDICATIONS: Comatose or severely depressed states; hypersensitivity to the drug.
WARNINGS: Safe use during pregnancy or lactation has not been established; weigh potential benefits to possible hazards. Not recommended for use in children under 16. May impair mental and/or physical abilities especially during early therapy; warn ambulatory patients about activities requiring alertness and concomitant use of alcohol or other CNS depressants. Not recommended for management of behavioral complications in mentally retarded patients.
PRECAUTIONS: Use with extreme caution in patients with a history of convulsive disorders; use with caution in patients with cardiovascular disease or in those with glaucoma or a tendency to urinary retention particularly when on concomitant anticholinergic medication. Loxapine has an antiemetic effect in animals which might occur in man masking signs of overdosage of toxic drugs and obscuring intestinal obstruction or brain tumor. Since possible ocular toxicity cannot be excluded, observe carefully for pigmentary retinopathy and lenticular pigmentation.
ADVERSE REACTIONS: CNS effects, other than extrapyramidal, infrequent. Mild drowsiness may occur at beginning of therapy or upon dosage increase, usually subsides with continued therapy. Sedation, dizziness, faintness, staggering gait, muscle twitching, weakness and confusional states have been reported. Extrapyramidal reactions often occur early in treatment manifested by Parkinson-like symptoms (tremor, rigidity, excessive salivation, masked facies, akathisia) controllable by dosage reduction or antiparkinson drugs at usual dosages. Dystonic and dyskinetic reactions, while less frequently occurring, may be more severe, requiring dosage reduction or temporary withdrawal plus appropriate counteractive drugs. Persistent Tardive Dyskinesia may appear during prolonged therapy or following discontinuance, the risk greater in the elderly, especially females, on high dosage. Symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of the tongue, face, mouth and jaw sometimes accompanied by involuntary movement of extremities. Since there is no known effective treatment, discontinue all antipsychotic drugs if symptoms appear. Reinstitution of treatment, increased dosage, or switching to another agent may mask syndrome. The syndrome may not develop if medication is stopped when fine vermicular movements of the tongue first appear. Cardiovascular Effects: Tachycardia, hypotension, hypertension, lightheadedness and syncope. ECG changes, not known to be related to loxapine use, have been reported. Skin: Dermatitis, edema of face, pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of unknown etiology seen in a few patients in hot summer months. Anticholinergic: Dry mouth, nasal congestion, constipation, blurred vision (more likely to occur with concomitant use of antiparkinson agents). Other: Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of unknown etiology.

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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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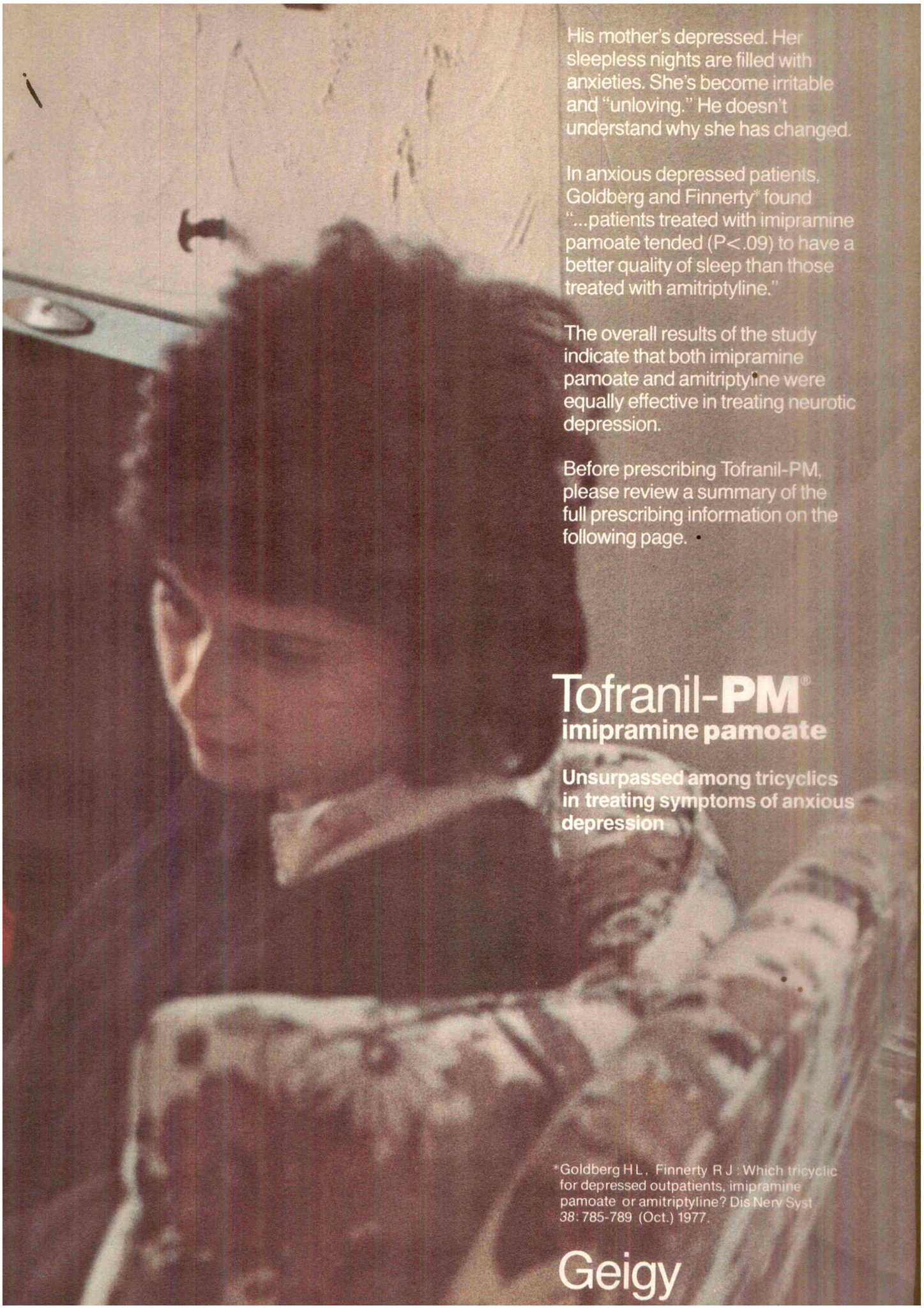
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"Please don't cry."



His mother's depressed. Her sleepless nights are filled with anxieties. She's become irritable and "unloving." He doesn't understand why she has changed.

In anxious depressed patients, Goldberg and Finnerty* found "...patients treated with imipramine pamoate tended ($P<.09$) to have a better quality of sleep than those treated with amitriptyline."

The overall results of the study indicate that both imipramine pamoate and amitriptyline were equally effective in treating neurotic depression.

Before prescribing Tofranil-PM, please review a summary of the full prescribing information on the following page.

Tofranil-PM®

imipramine pamoate

Unsurpassed among tricyclics in treating symptoms of anxious depression

*Goldberg H L, Finnerty R J: Which tricyclic for depressed outpatients, imipramine pamoate or amitriptyline? Dis Nerv Syst 38: 785-789 (Oct.) 1977.

Geigy

Tofranil-PM®
imipramine **pamoate**

Geigy

As anxiety, agitation, sleep disturbances, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic. Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)

(B) 98-146-840-A(9/75) 667120

For complete details, including dosage and administration, please refer to the full prescribing information.

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A66

Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
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- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

80pp. Paperbound

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Rapid onset of therapeutic effect in a wide range of psychotic symptoms.[1-4]

Helps avoid long-term hospitalization.[1-3,5]

Minimal risk of hypotension, oversedation, or troublesome anticholinergic effects.[1,4,6,7,9-11]
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Permits aggressive titration to effective dosage levels for optimal control.[5,6]

Often leaves patient with improved insight,
with a renewed interest in the environment, and more likely to participate in therapeutic efforts.[1,7]

Common side effects easily controlled.[1,4-6,8,9]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

References: 1. Ayd, F.J., Jr.: Med. Sci. 18:55 (Oct.) 1967. 2. Rapp, M.S.: Can. Psychiatr. Assoc. J. 15:73 (Feb.) 1970. 3. Rubin, R.: Ala. J. Med. Sci. 8:414 (Oct.) 1971. 4. Abuzzahab, F.S., Sr.: Psychosomatics 11:188 (May-June) 1970. 5. Man, P.L.: Dis. Nerv. Syst. 34:113 (Feb.) 1973. 6. Howard, J.S.: Dis. Nerv. Syst. 35:458 (Oct.) 1974. 7. Sugerman, A.A., et al.: Am. J. Psychiatry 120:1190 (June) 1964. 8. Darling, H.F.: Dis. Nerv. Syst. 34:364 (Oct.-Nov.) 1973. 9. Gerle, B.: Clin. Trials J. 3:380 (Feb.) 1966. 10. Snyder, S.H., et al.: Science 184:1243 (June 21) 1974. 11. Stimmel, C.L.: Dis. Nerv. Syst. 34:219 (Apr.) 1976.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Please turn page for summary of prescribing information.

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A tasteless, odorless, colorless liquid concentrate for better patient compliance: 2 mg per ml.

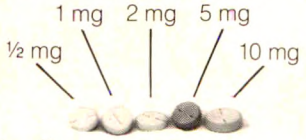
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A rapid-acting injection for psychiatric emergencies: 5 mg per ml,* with 1.8 mg methylparaben and 0.2 mg propylparaben per ml, and lactic acid for pH adjustment to 3.4 ± 0.2.



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5 tablet strengths for convenience in individualizing dosage:



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tablets/concentrate/injection

A dosage form for every therapeutic need

Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

HALDOL tablets are manufactured by McNeil Laboratories Co., Dorado, Puerto Rico 00646.

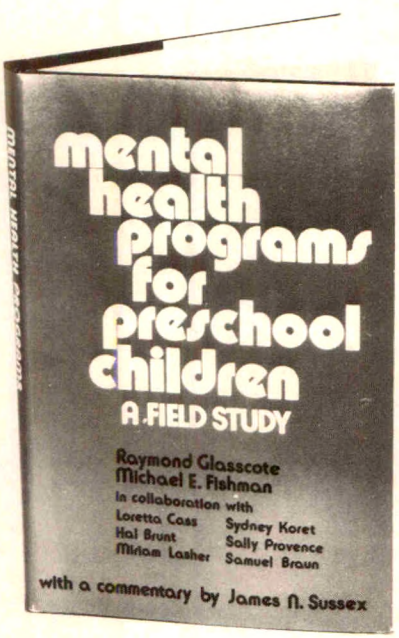
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This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

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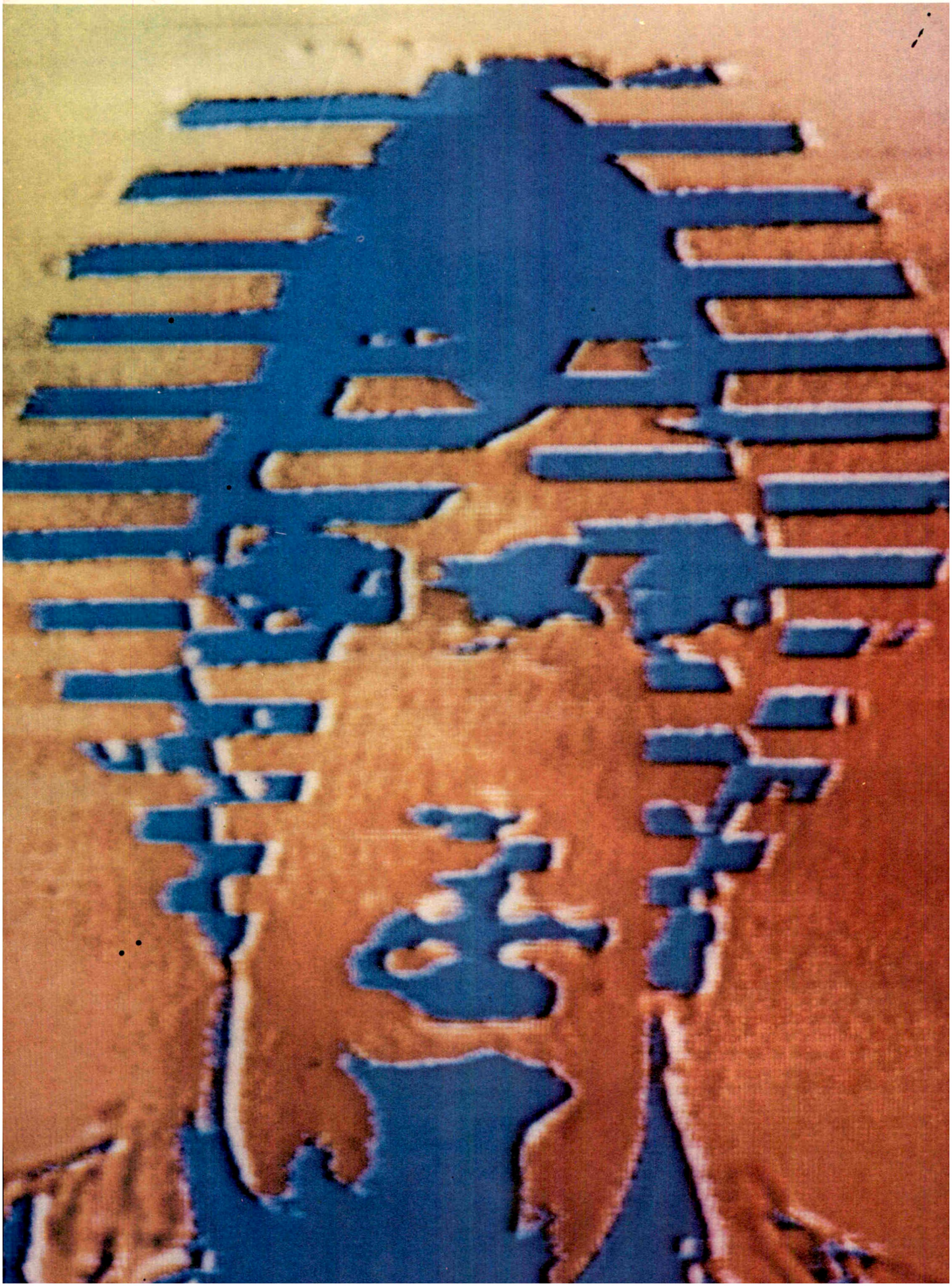
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NO incidence	Tolerance development Hepatotoxic changes Ophthalmological changes Excessive weight gain Impotence Decreased libido Skin pigmentation Thyroid function changes Persistent tardive dyskinesia
LOW incidence	Sedation Adverse drug interactions Hypotension Hematological changes Lactation Amenorrhea Autonomic changes Skin rash Nonpersistent symptoms of tardive dyskinesia
MODERATE incidence	Extrapyramidal reactions

*This table compiled from clinical and field reports, according to the best available information.

Further advantages with MOBAN®

The pharmacological effect from a single oral dose persists for 24-36 hours, permitting *h.s.* dosage, once the daily dosage level is established.

Several clinicians have reported that obese patients on MOBAN® frequently experienced a marked reduction in weight toward normal weight.

How to use MOBAN®

The usual starting dose is 50-75 mg/day. You can increase to 100 mg/day in 3-4 days. Titrate individually up or down depending on patient response. Some chronic, treatment-resistant patients may require an increase to 400 mg/day. (However, the long-term safety of 400 mg/day has not been established.) Please refer to complete prescribing information for further dosage instructions.

For complete prescribing information, please see next page.

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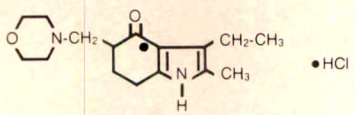
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MOBAN®
molindone HCl

5 mg	10 mg	25 mg
Endo	Endo	Endo

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN® (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN® being excreted in urine and feces.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in the pregnant patient have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN® Caution should be exercised where increased activity may be harmful.

MOBAN® does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN® has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological

Extrapyramidal Reactions

Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia

Motor restlessness may occur early.

Parkinson Syndrome

Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome

Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System

Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological

There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN® may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects

Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with MOBAN® therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN®.

Cardiovascular

Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological

Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN® (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN®.

Skin

Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN® usage alone.

MOBAN® (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN® is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia

Although rarely reported with MOBAN® (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50-75 mg/day.

—Increase to 100 mg/day in 3 or 4 days.

—Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.

—An increase to 225 mg/day may be required in patients with severe symptomatology.

—Some chronic, treatment resistant patients, may require up to 400 mg/day (however, the long term safety of 400 mg/day has not been established).

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

Dosages may be administered once a day.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidot must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN® in animals this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN® in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles of 100 and 1000 with potencie and colors as follows: 5 mg orange, 10 mg lavender, 25 mg light green.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

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New Hampshire Hospital, Concord, N.H., an 800 bed JCAH-Accredited psychiatric hospital, is seeking an Assistant Superintendent for Professional Services to be responsible for the direction and coordination of clinical programs for the treatment, care and management of mentally ill patients. New Hampshire Hospital, the only public psychiatric hospital in N.H., is located in Concord, a city of approximately 30,000 people, 75 minutes north of Boston, MA. Salary ranges from $30,740-$38,690, plus an additional $3,000 per year if board certified. Possibility of faculty appointment to major university. Housing and other fringe benefits are available. Candidate must be a physician-psychiatrist licensed or eligible for licensure as an M.D. in New Hampshire and certified or eligible for certification by the American Board of Neurology and Psychiatry.

Interested applicants should contact C. Clifton DuBois, Ph.D., Superintendent at New Hampshire Hospital, 105 Pleasant Street, Concord, New Hampshire 03301. Telephone (603) 271-2205.

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Computerized transaxial tomography (CT scan) is an important new noninvasive technique that can help in determining the etiology of epileptic seizures. Shown here is a single slice from a color-enhanced CT scan of the brain of a patient with epilepsy. The absence of vascula

A proven way to start seizure control

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A single entity with multiple anticonvulsant properties

Primidone itself has anticonvulsant activity. In addition, its major metabolite, phenylethylmalonamide (PEMA), exerts an anticonvulsant effect of its own and also enhances the anticonvulsant action of the second metabolite, phenobarbital. In patients refractory to phenytoin and phenobarbital, primidone may prove effective as the sole therapy.[1]

Multiple indications—grand mal, psychomotor, and focal epileptic seizures

In grand mal, MYSOLINE is comparable in efficacy to phenytoin and phenobarbital.[2,3] In psychomotor seizures, phenobarbital is rarely effective.[1] In focal seizures, phenytoin used alone is not indicated[4]—and, according to Forster,[3] MYSOLINE is markedly more effective than phenobarbital, which should dispel the misconception that MYSOLINE is "just a phenobarbital." (See chart below.)

Efficacy ratings* of anticonvulsants in epilepsy

Agent	Grand Mal	Psychomotor	Focal
Mysoline	++++	++++	++++
Hydantoinates (phenytoin)	++++	+++	+++
Barbiturates (phenobarbital)	++	++	++

*Relative effectiveness, ++++ being most effective. Adapted from Forster, F. M.[3]

Consider it first for major motor epilepsies

MYSOLINE® (primidone) Ayerst®

See next page for Brief Summary and references.

Start seizure control with MYSOLINE® (primidone)

Initial dose in children over 8 years of age and adults: 250 mg daily at bedtime

BRIEF SUMMARY
(For full prescribing information, see package circular.)
MYSOLINE®
Brand of **PRIMIDONE** *Anticonvulsant*

ACTIONS: MYSOLINE raises electro- or chemoshock seizure thresholds or alters seizure patterns in experimental animals. The mechanism(s) of primidone's antiepileptic action is not known.

Primidone *per se* has anticonvulsant activity as do its two metabolites, phenobarbital and phenylethylmalonamide (PEMA). In addition to its anticonvulsant activity, PEMA potentiates that of phenobarbital in experimental animals.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several weeks before it can be assessed.

Usage in pregnancy: The effects of MYSOLINE in human pregnancy and nursing infants are unknown.

Recent reports suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Data are more extensive with respect to diphenylhydantoin and phenobarbital, but these are also the most commonly prescribed anticonvulsants; less systematic or anecdotal reports suggest a possible similar association with the use of all known anticonvulsant drugs.

The reports suggesting an elevated incidence of birth defects in children of drug-treated epileptic women cannot be regarded as adequate to prove a definite cause and effect relationship. There are intrinsic methodologic problems in obtaining adequate data on drug teratogenicity in humans; the possibility also exists that other factors, *e.g.*, genetic factors or the epileptic condition itself, may be more important than drug therapy in leading to birth defects. The great majority of mothers on anticonvulsant medication deliver normal infants. It is important to note that anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and threat to life. In individual cases where the severity and frequency of the seizure disorder are such that the removal of medication does not pose a serious threat to the patient, discontinuation of the drug may be considered prior to and during pregnancy, although it cannot be said with any confidence that even minor seizures do not pose some hazard to the developing embryo or fetus.

The prescribing physician will wish to weigh these considerations in treating or counseling epileptic women of childbearing potential.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

PRECAUTIONS: The total daily dosage should not exceed 2 g. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 g per day. The initial dose is 250 mg. Increments of 250 mg are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 g. A typical dosage schedule for the introduction of MYSOLINE is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg daily at bedtime	*2nd Week* 250 mg b.i.d.
3rd Week 250 mg t.i.d.	*4th Week* 250 mg q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg, with gradual weekly increases of 125 mg a day, to a daily total usually between 500 mg and 750 mg.

In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances: for initiation of combination therapy; during "transfer" therapy; for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.).

HOW SUPPLIED: MYSOLINE Tablets—No. 430—Each tablet contains 250 mg of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg of primidone (scored), in bottles of 100 and 500.
MYSOLINE Suspension—No. 3850—Each 5 ml (teaspoonful) contains 250 mg of primidone, in bottles of 8 fluidounces.

References: 1. Woodbury, D. M., and Fingl, E., in Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics, ed. 5, New York, Macmillan Publishing Co., Inc., 1975, p. 222. **2.** Rodin, E., in Conn, H. F. (ed.): Current Therapy 1977, Philadelphia, W. B. Saunders Company, 1977, p. 716. **3.** Forster, F. M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. **4.** Physicians' Desk Reference, ed. 31, Oradell, N.J., Medical Economics Co., 1977, p. 1175.

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SUPERINTENDENT

Big Spring State Hospital

Physician, licensed to practice in Texas. Certified or Board eligible in psychiatry and must possess administrative ability. Base salary is $40,600/yr with house and utilities provided. Up to $3,000 additional salary may be granted by the Commissioner of TDMHMR and an additional $1,000 compensation for Board Certification. Fringe benefits include vacation, sick leave, and usual state benefits including a retirement program. Legal protection provided by state law. Applications must include a professional resume and must list not less than three references. Applications must be addressed to: John J. Kavanagh, M.D., Commissioner, Texas Department of Mental Health and Mental Retardation, Box 12668, Capitol Station, Austin, Texas 78711.



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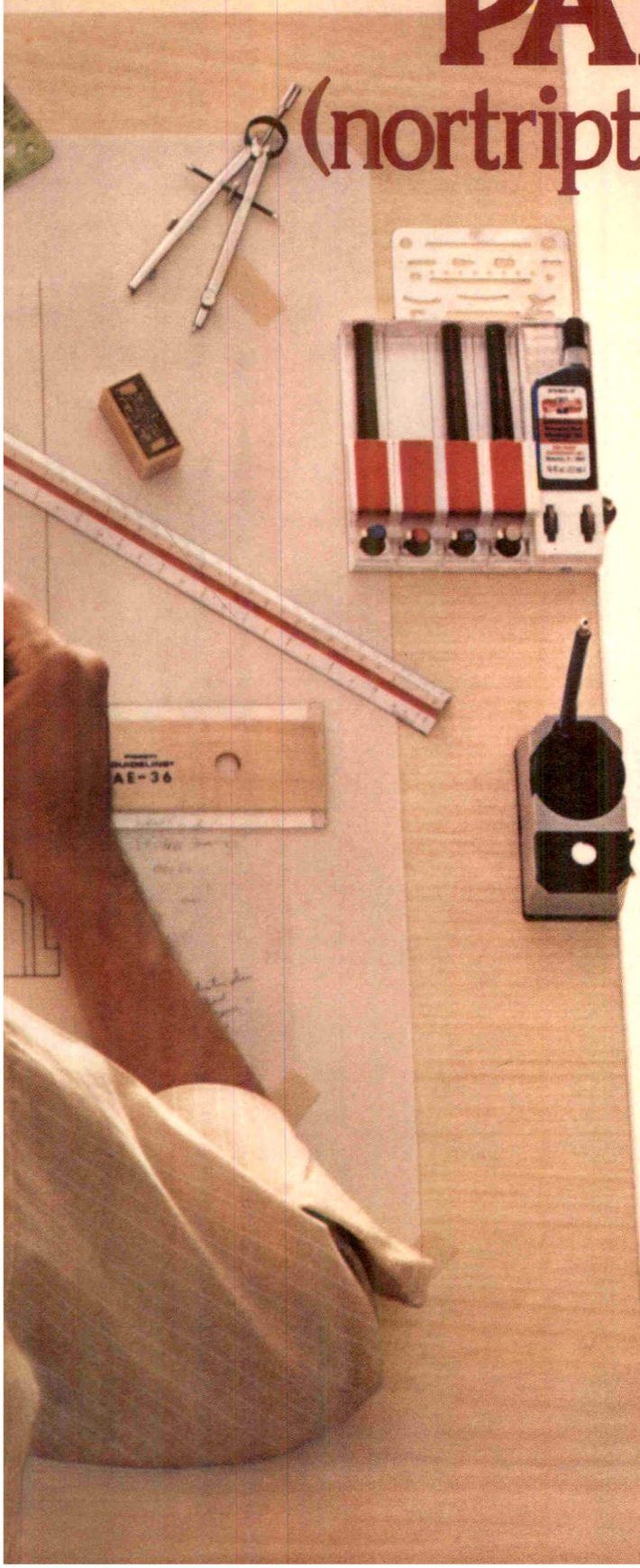
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For Brief Summary please see last page of this advertisement.

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The antidepressant for the patient who must remain alert and active.

- relieves depression without causing problem daytime drowsiness
- insomnia of depression begins to improve within a week
- PAMELOR therapy is well tolerated

Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose* – 25 mg. three or four times daily; dosage should begin at a low level and increase as required. As an alternate regimen, the total daily dosage may be given once-a-day. *Elderly and Adolescent* - 30 to 50 mg. per day, in divided doses, or the total dosage may be given once-a-day. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

SDZ-8-14

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Prescribing Information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats Oral Dose —
20 mg/kg/day — 10 days, no adverse effect
40 mg/kg/day — 10 days, no adverse effect

Pregnant Mice Oral Dose —
20 mg/kg/day — 10 days, slight increase resorptions
40 mg/kg/day — 10 days, slight increase resorptions

Pregnant Rabbits Oral Dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg/day.
- — Increase to 100 mg/day in three or four days.
- — Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- — An increase to 225 mg/day may be required in patients with severe symptomatology.
- — Some chronic, treatment-resistant patients may require up to 400 mg/day; (however, the long-term safety of 400 mg/day has not been established).

Elderly or debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four times a day.
2. Moderate — 10 mg to 25 mg three or four times a day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone hydrochloride) capsules are supplied in bottles of 100 in the following dosage strengths and colors:

5 mg (**NDC** 0074-5542-13) blue and cream
10 mg (**NDC** 0074-5543-13) red and cream
25 mg (**NDC** 0074-5544-13) brown and cream

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(Molindone HCl)

See overleaf for prescribing information

Significant gains are reported in control of hallucinations, conceptual disorganization, hostility, unusual thought content, emotional withdrawal.

Yet no reports of persistent tardive dyskinesia, excessive weight gain, impotence, lens opacities, skin pigmentation; and only rare cases of significant hypotension. Other side effects are similar to comparable agents. 8063266

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THE AMERICAN JOURNAL OF PSYCHIATRY

In this issue

The Psychodynamics and Developmental Psychology of the Borderline Patient: A Review of the Literature

By Edward R. Shapiro

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Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: The possibility of extrapyramidal reactions from 'Thorazine' may confuse the diagnosis of Reye's syndrome or other encephalopathy. Therefore, avoid use in children or adolescents with suspected Reye's syndrome.

Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or obscure diagnosis of conditions such as intestinal obstruction, brain tumor, and Reye's syndrome (see Warnings). Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.



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• FAMILY THERAPY AND EVALUATION THROUGH ART •

By **Hanna Yaxa Kwiatkowska,** *The George Washington Univ., Washington, D. C. Foreword by* **Lyman C. Wynne.** Written by the innovator of family art therapy, this book describes the use of art therapy with families for evaluation of family dynamics, treatment and research.

The author outlines the developments in the field from its inception to the present. She covers the steps required in assessing the problems of the troubled family and in carrying out the necessary treatment. Case material is presented which demonstrates the merits of this powerful approach. Methods of research arising from the material generated by the evaluative and therapeutic procedures are also examined.

The author stresses that family art therapy is unique in that individual family members and the family as a group communicate with one another and with the therapist through their expressive art. Their pictures and sculptures embody family process and suggest the etiology of the difficulties in the family relations. That is, the transactions of the family engaged in art procedures reproduce their transactions in day-to-day life, thus offering an unbiased view of the family system and interrelationships. Their individual and joint artwork, says the author, provides revelations and changes that cannot be denied. This is the source of the effectiveness of family art therapy.

The importance of this contribution to the practice of family therapy must not be underestimated. The book will appeal to a variety of therapists and counselors who are using or considering the use of family therapy and evaluation through art. *'78, 296 pp., 125 il. (12 in color), 7 tables, $21.75*

INTRODUCING NEUROPSYCHOLOGY: The Study of Brain and Mind *by* **Stuart J. Dimond,** *Univ. College, Cardiff, Wales.* This book delves into enigmas of mental life to arrive at an expanded perception of precisely how the mind and behavior are related to the underlying machinery of the brain. The initial portion of the text outlines the status of neuropsychology among the brain sciences, the methods used in this discipline, and fundamental brain systems. The effects of brain injury and disease, the development of the brain in relation to the mind, the split-brain condition in man, and other topics are discussed in this section. Remaining chapters focus on the cerebral cortex, the infant brain, the normal brain, mechanisms and functions of the higher mental states, neurological processes in psychiatric illness, and the development of technology for control of the brain. *'78, 236 pp., 12 il., 2 tables, $13.75*

THE HAND TEST: A New Projective Test with Special Reference to the Prediction of Overt Aggressive Behavior (5th Ptg.) *by* **Barry Bricklin** *and* **Zygmunt A. Piotrowski,** *both of The Jefferson Medical College of Philadelphia, Philadelphia, Pennsylvania, and* **Edwin E. Wagner,** *Univ. of Akron, Akron, Ohio.* This text describes a practical, projective personality test designed to make it possible to predict not only potentially aggressive overt behavior but many other facets of interpersonal behavior as well. It consists of pictures of human hands in various ambiguous poses. The subject is asked to tell what each hand may be doing. A large section is devoted to use of the test in daily clinical practice — including diagnostic hints and procedures pertaining to use of the test with individual cases. *'78, 112 pp. (5 3/4 x 8 3/4), 1 il., 25 tables, $7.50*

CAREER DEVELOPMENT AND COUNSELING OF WOMEN *edited by* **L. Sunny Hansen,** *Univ. of Minnesota, Minneapolis, and* **Rita S. Rapoza,** *Wilson Learning Corp., Eden Prairie, Minnesota. (46 Contributors)* This unique text includes articles on psychological and environmental variables which inhibit or facilitate women's development and affect their decisions, not only about work, but also about their life roles with respect to family, leisure, and community. The text also provides information which will help teachers, counselors, psychologists and others to find ways to help women expand their career and life-style options. Specific topics include occupational socialization, negotiating and planning for multiple roles, assessment of women's career interests, and cross-cultural perspectives of female career development. An extensive bibliography supplements the text. *'78, 672 pp., 57 tables, $24.75*

MICROCOUNSELING: Innovations in Interviewing, Counseling, Psychotherapy and Psychoeducation (2nd Ed.) *by* **Allen E. Ivey,** *Univ. of Massachusetts, Amherst, and* **Jerry Authier,** *Univ. of Nebraska Medical Center, Omaha. Foreword by* **Bernard G. Guerney, Jr.** *(3 Contributors)* The Second Edition of this groundbreaking introduction to interviewing and counseling updates the extensive research on the microcounseling model; presents new chapters on the cross-cultural and cross-racial implications of interviewing training; provides material illustrative of how microcounseling skills are used by various types of therapists; and includes a new section on operational definitions. The text continues to present a detailed analysis of the interview, specific methods for teaching and researching interviewing skills, and an explication of the videotape methods used in microcounseling. *'78, 624 pp., 13 il., $24.50*

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MAJOR MEDICINAL PLANTS: Botany, Culture and Uses *by* **Julia F. Morton,** *Univ. of Miami, Coral Gables, Florida. Forewords by* **Norman R. Farnsworth** *and* **Maynard W. Quimby.** This luxuriously produced volume describes the principal drug plants which have played an important medicinal role for many generations. Origin and geographical distribution are detailed along with areas of cultivation, yields, chemical constituents, economic uses and, where applicable, toxicity. The botany, culture, harvesting and handling of medicinal plants are explored in unprecedented depth. All plants are richly illustrated. Appendices cover those plants which provide pharmaceutical aids, and those species which have been dropped from official use in recent years. A bibliography composed of over 600 international research reports follows the text. *'78,,78, 4, 448 pp. (6 3/4 x 9 3/4), 109 il. (17 in color), 2 tables, $49.50*

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Overviews

Overview articles attempt to bring together important relevant information on a topic of general interest to psychiatry. They are usually written at the invitation of the Editor; authors who have ideas for overview articles are advised to check with the Editor to ensure that a similar work is not in preparation. Overviews should not exceed 25 double-spaced pages (about 7,500 words) and should have no more than 100 references. All overviews, including those written by invitation, are given the same peer review received by other papers.

Regular Articles and Brief Communications

The primary difference between these two types of papers is length. The "brief communication" designation does not imply less sophisticated or complete work: it merely means that the content can be expressed within the upper limit of 2,500 words. There is no difference in the review procedure or scheduling of these articles. Authors who submit unnecessarily long articles with redundancies and loose writing style will be asked to shorten them, which can delay publication. Authors should choose their words carefully, avoiding lengthy introductions containing material that is common knowledge, summaries that merely repeat the results, unnecessary tables, figures or references, etc. Single case reports will not be accepted as regular articles or Brief Communications; these papers should be submitted as Clinical and Research Reports.

Clinical and Research Reports

This section contains very brief articles (maximum of 1,000 words, 10 references, 1 table, and *no figures*) reporting new research findings, including preliminary data from pilot studies, and case reports that 1) describe new syndromes, 2) cast a new light on established ones, 3) indicate a new therapeutic procedure of potential value, or 4) describe adverse effects or previously unreported complications of drugs or therapeutic interventions. Because of the stringent criteria for this section, authors can expect more rapid publication than is possible in other sections. Submitted papers that substantially exceed the stated maximum length or contain figures will be returned to the author unreviewed.

Prompt Publication Policy

Articles submitted for prompt publication can be of either regular or Brief Communication length (a "prompt publication policy" is automatically in effect for Clinical and Research Reports). These papers are given priority in scheduling; however, authors should be aware that the minimum publication time is four months. Prompt publication papers must meet stringent criteria of originality, and authors must state their reasons for wanting rapid review in a cover letter to the Editor. It is important to think carefully about the nature of the paper before requesting prompt publication; a paper may be delayed if it is submitted inappropriately.

Other Sections

Letters to the Editor. Brief letters (maximum of 500 words and 5 references) will be considered if they include the notation "for publication" in the upper right corner. Letters critical of an article published in the *Journal* will automatically be sent to the authors for reply. The *Journal* is unable to notify authors of receipt of letters or to return those not published. All letters are subject to editing. Letters must be typed *double-spaced* throughout on letter-size bond paper; two copies are required. Letters that are not typed appropriately or do not conform to *Journal* reference style will be returned to the author for revision.

Book Reviews. Books for review or listing may be sent directly to the Editor, *American Journal of Psychiatry*, 1700 Eighteenth St., N.W., Washington, D.C. 20009. Book reviews are usually solicited by the editor. Authors interested in reviewing a particular book are urged to contact the editorial office to see if the Editor wishes the book to be reviewed and to ascertain whether it has been previously assigned to another reviewer. Reviews are usually less than 500 words long, seldom contain references, and will be edited.

TYPING AND ARRANGING THE PAPER

All manuscripts must be typed in upper- and lowercase on one side only of 22 × 28 cm (8½ × 11 inches) *nonerasable* bond paper. All parts of the manuscript, including case reports, quotations, references, and tables, must be double-spaced throughout. All 4 margins must be 3¾ cm (1½ inches). The manuscript should be arranged in the following order, with each numbered item beginning a new page: 1) title page, 2) précis, 3) text, 4) references, 5) tables, 6) footnotes to text, and 7) figure captions. All pages must be numbered, with the title page as number 1. Please consult the following section on *Journal* style specifications for criteria for each part of the paper.

STYLE SPECIFICATIONS

Title Page

Title. The title should be informative but as brief as possible. Headline style (declarative sentences) should be avoided. Abbreviations and acronyms are rarely used in titles.

By-line. Authors named in the by-line should be limited to principal researchers and/or writers; collaborators can be acknowledged in a footnote. Please include degrees (other than honorary or undergraduate) after the authors' names.

Previous presentation. If the paper has been presented orally, please give the name of the meeting, the place, and inclusive dates.

Author affiliations. The authors' position titles and affiliations should be given in a paragraph using complete sentences. A full address is necessary only for the author who is to receive reprint requests.

Acknowledgments. Acknowledgments should be in a separate paragraph on the title page. Grant support should include the full name of the granting agency as well as the grant number. Individual acknowledgments should be as brief as possible. Acknowledgments of companies that supplied drugs are used only in the case of experimental drugs or those unavailable in this country.

Other requirements. The number of words, tables, and figures should appear in the upper right corner and a phone number for the corresponding author in the lower right.

Précis

The précis should be up to 100 words for regular articles and Brief Communications (no précis is used in the Clinical and Research Reports section). The précis should be a single paragraph using complete, connected sentences, active verbs, and the third person. In most cases, the précis replaces a summary.

Text

Headings and subheadings should be inserted at reasonable intervals in all types of papers. Footnotes to text material should be typed on a separate page at the end of the manuscript. Summaries are rarely desirable, although a final "Comment" or "Conclusions" section may be used. The body of the paper should be written in the active voice and first person where appropriate.

Statistics. Authors should be sure that statistics used are both necessary and appropriate. Statistical tests should be described and a reference citation given if the tests are not generally known. It is not sufficient to say "differences between the groups were significant." In cases of lengthy complicated analyses, the authors may summarize results and indicate that details are available from them on request.

Abbreviations. All abbreviations (other than those for metric units) should be explained the first time they are used. Idiosyncratic abbreviations should be avoided: overuse of abbreviations may hinder rather than facilitate readability.

Drugs. Generic rather than trade names of drugs should be used; trade names may be given parenthetically if necessary.

Units of measurement. All measurements should be in metric units; standard abbreviations are used.

References

References should be restricted to closely pertinent material; a complete review of the literature is rarely desirable. *Accuracy of citation is the author's responsibility.* References should conform exactly to the original spelling, accents, punctuation, etc. Authors should be sure that all references listed have been cited in text; no bibliographies can be used.

References are numbered and listed by their order of appearance in text; the text citation is followed by the appropriate reference number in parentheses. Do *not* arrange the list alphabetically.

Personal communications, unpublished manuscripts, manuscripts submitted but not yet accepted, and similar unpublished items should not appear in the reference list. Such citations may be noted in text or in a footnote. It is the author's responsibility to obtain permission to refer to another individual's unpublished observations. Manuscripts that are actually "in press" may be cited as such in the reference list; the name of the journal must be included.

Type references in the style shown below and on the next page, double-spaced *throughout* (not just a line between references). List up to three authors; designate one or more authors past the third "et al." Abbreviations of journal names should conform to the style used in *Index Medicus*; journals not indexed there should not be abbreviated.

1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26

2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

Tables

Tables should be self-explanatory and should supplement rather than duplicate text. Authors may be asked to delete tables that present data which could be given succinctly in text or repeat information in the Results section. Tables are generally reserved for data presentation and not used as lists or charts. They should be no wider than 120 typewriter characters, including spaces. Consult recent issues of the *Journal* for table style. Please note that no vertical rules or internal horizontal rules are used. It is usually desirable to give both numbers and percentages where applicable. All units of measurement must be specified within the table. Metric measurements are used in tables and throughout the text. Each table should be identified numerically and include a concise title.

Figures

Figures should be used only for data that cannot be adequately presented in text or tabular form. Figures should be submitted as glossy prints with one photocopy attached to each manuscript copy. The author's name and the title of the paper should appear on a gummed label affixed to the back of each glossy. All figures should be able to withstand reduction to about 8 cm (3¼ inches). Authors are urged to consider carefully the necessity of figures; they may be asked to delete figures that repeat information available in text. It is often advisable to obtain professional assistance in the preparation of figures.

PROCESSING OF ACCEPTED MANUSCRIPTS

Authors will be notified of acceptance of their papers; papers will then be edited and sent to the first-named (or corresponding) author for corrections and answers to editorial queries. No galley proofs are sent to authors, so the edited manuscript should be read with extreme care. Prompt return of edited manuscripts is essential. Authors who expect to be away from their offices for a long period or who change address after notification of acceptance should so inform the *Journal* office.

Manuscripts are accepted with the understanding that the Editor has the right to make revisions aimed at greater conciseness, clarity, and conformity with *Journal* style.

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The December 1978 issue of

The American Journal of Psychiatry

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  By Ming T. Tsuang



Hallucinations... delusions... thought disorders... controlled

Navane (thiothixene) rapidly controls hallucinations as well as the agitation and hostility patterns they frequently generate.

Rapid control of psychotic symptoms on admission

Navane provides effective, rapid control of the major symptoms of psychosis including hallucinations, delusions, agitation, hostility, and thought disorders.[1-5] Navane promotes rapid progress, usually without the over-sedation or cardiovascular problems that can impede the therapeutic course.

Continued long-term improvement

Because Navane provides long-term outpatient control of psychotic symptoms, it can enable many patients to function effectively at home and on the job and to adjust rapidly to the community.[6]

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Navane allows patients to remain alert and active, seldom causing over-sedation or drowsiness.[7] Hypotension and other cardiovascular reactions[5,6,8] are seldom reported and the occurrence of unpleasant anticholinergic side effects such as dry mouth or constipation is rare.[9] If extrapyramidal symptoms occur, they are usually readily controlled through dosage adjustments or antiparkinson agents.

Rapid control
Long-term improvement

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(thiothixene) (thiothixene HCl)

Capsules 1 mg., 2 mg., 5 mg., 10 mg., 20 mg. Concentrate 5 mg./ml. Intramuscular 2 mg./ml.

For prescribing information, including adverse reactions and contraindications, please see following page of this advertisement.

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Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

BRIEF SUMMARY OF PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg
(thiothixene hydrochloride) Concentrate: 5 mg/ml.
Intramuscular: 2 mg/ml

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, 1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. Denber HCB, Turns D: Double blind comparison of thiothixene and trifluoperazine in acute schizophrenia. *Psychosomatics* 13:100-104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: The place of thiothixene in the treatment of schizophrenic patients. *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, 1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 130th Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Hil TM, Unverdi C, Wohlrabe J, Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, et al: Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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This 24 credit Category I course was designed by the Director and the Office of Continuing Education of the Institute of Pennsylvania Hospital. In the first stage of development, topic selection was confirmed by a nationwide sampling of 900 practicing psychiatrists. The director then invited seven outstanding psychiatrist/teachers to prepare lessons in their areas of recognized expertise. The published material will be tightly structured and clearly presented. At the conclusion of the course, comments and views of the participants will be published so that everyone can further benefit from a shared learning experience.

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The objective of this continuing medical education program is to provide an opportunity for the practicing psychiatrist to assess and further develop his own clinical skills by means of a home study program sponsored by the Institute of Pennsylvania Hospital. This first program will present in depth six topics of current interest and clinical usefulness and will include a series of questions designed to aid this assessment and to focus his reading. If he chooses, the psychiatrist may select additional articles for further study and may also develop a written dialogue with the editors for further discussion.

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The sophisticated computer response form not only presents scores and averages for personal and confidential peer comparison but in the case of a wrong answer will indicate sources of correct information both in the text and in key references. The questions and answers are keyed to the computer, personalizing responses while maintaining confidentiality. Our unique procedures encourage the development of a dialogue between the course participant and the editors.

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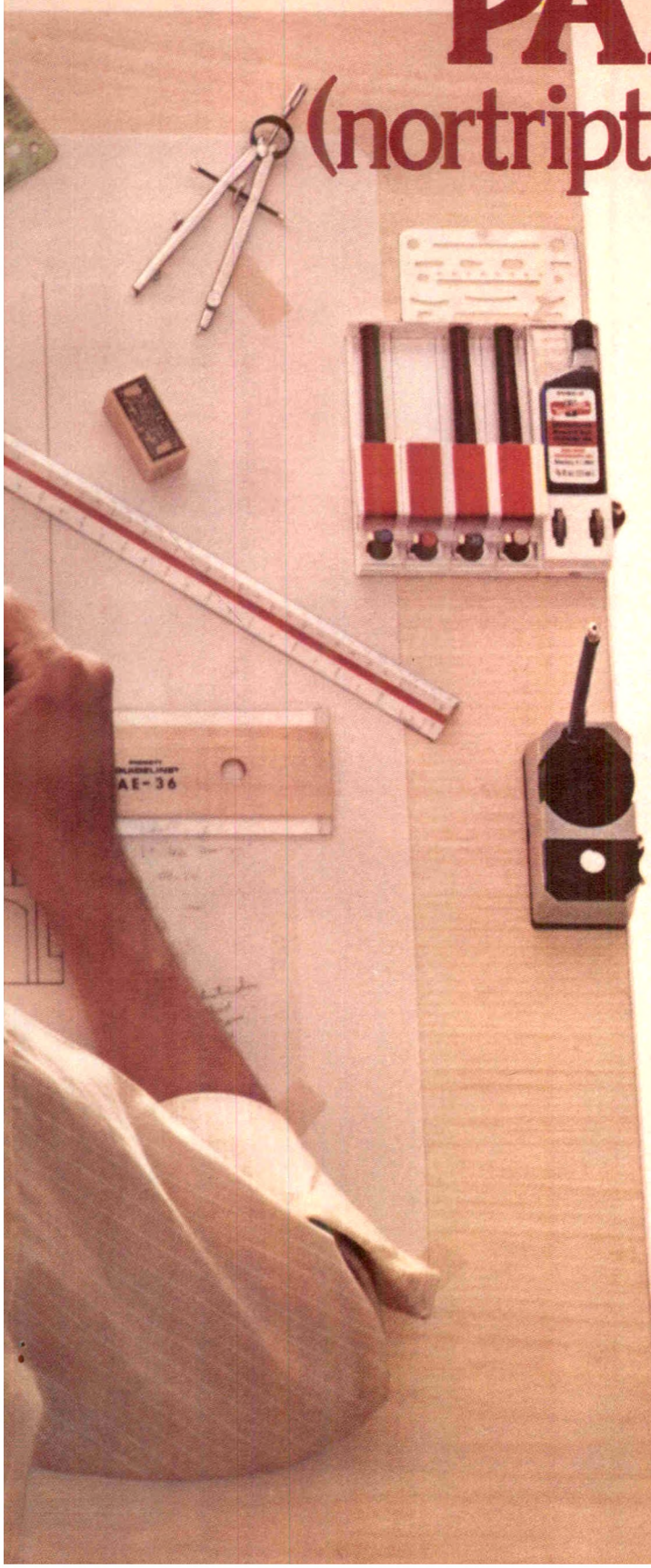
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The antidepressant for the patient who must remain alert and active.

- relieves depression without causing problem daytime drowsiness
- insomnia of depression begins to improve within a week
- PAMELOR therapy is well tolerated

Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose* – 25 mg. three or four times daily; dosage should begin at a low level and increase as required. As an alternate regimen, the total daily dosage may be given once-a-day. *Elderly and Adolescent* - 30 to 50 mg. per day, in divided doses, or the total dosage may be given once-a-day. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

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References: 1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. 2. Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. 3. Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. 4. Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. 5. Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. 6. Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. 7. Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. 8. Snyder, S.H., et al: Science *184*:1243 (June 21) 1974. 9. Stimmel, C.L.: Dis. Nerv. Syst. *34*:219 (Apr.) 1976.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

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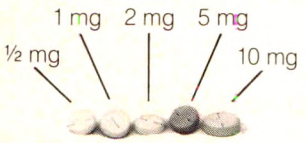
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Summary of Prescribing Information

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use With Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases.

Withdrawal Emergent Neurological Signs: Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn.

Persistent Tardive Dyskinesia: Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent.

Other CNS Effects: Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms.

Cardiovascular Effects: Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

Caution: Federal law prohibits dispensing without prescription.

Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

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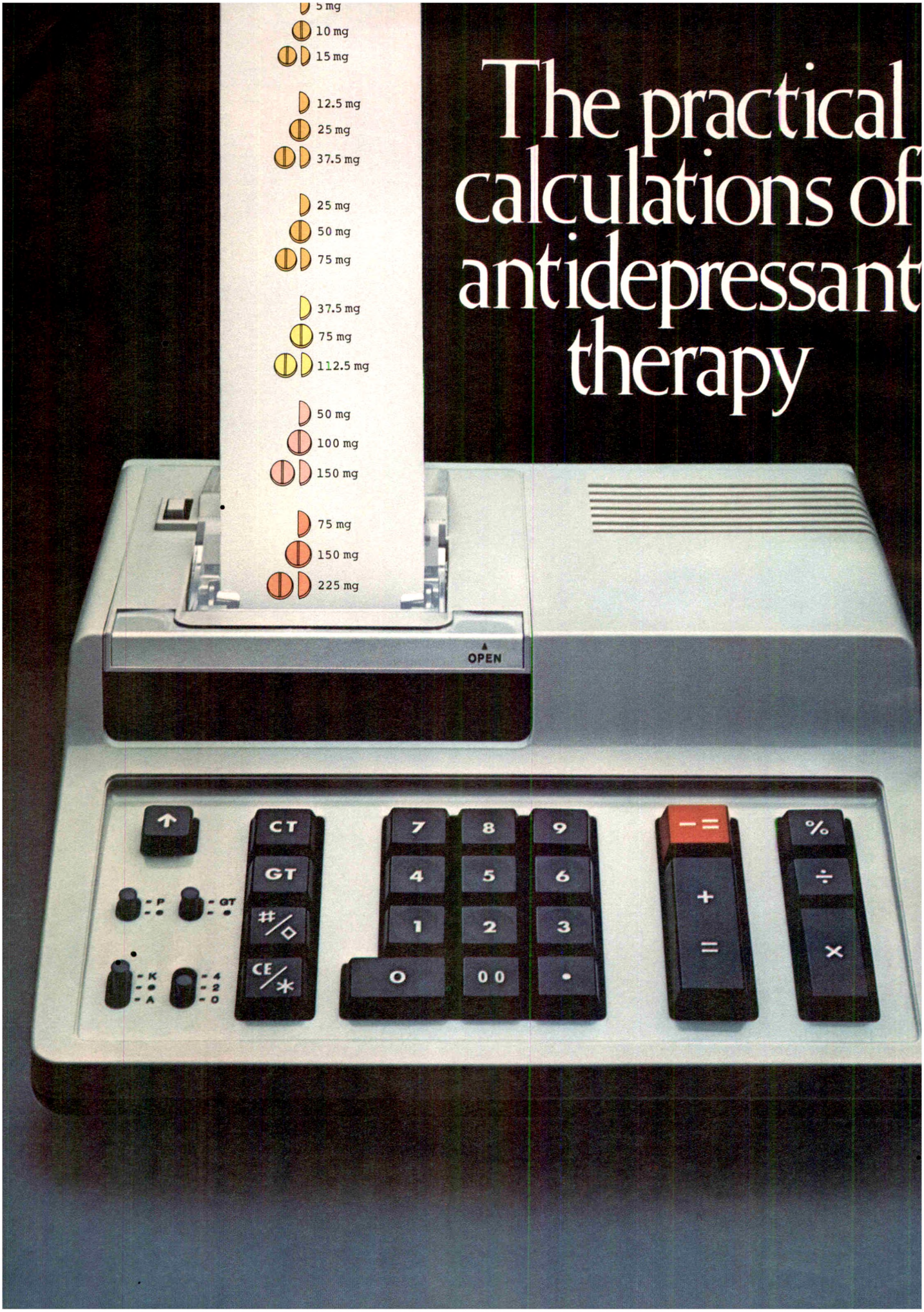
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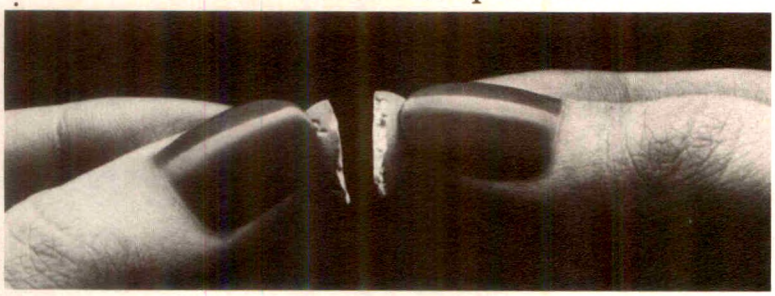
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Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

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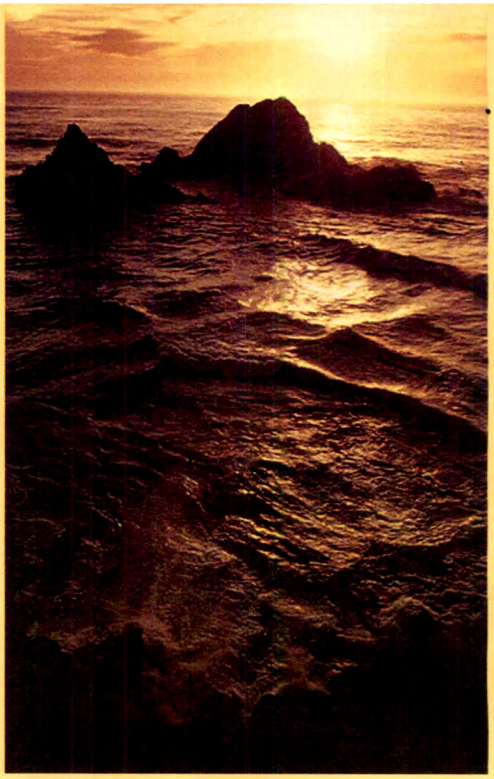
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Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./1., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./1. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage.

Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./1. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./1. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./1. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity of inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

How Supplied: 300 mg. capsules in bottles of 100.

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- Virtually no drowsiness* in most Verstran patients (less than 10%)
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- The simplicity of a single-strength 10-mg scored tablet, for easy titration and improved patient compliance
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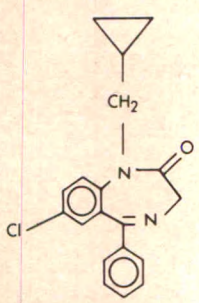
TO ENHANCE PATIENT RESPONSIVENESS CONSIDER

VERSTRAN (prazepam) Ⓒ IV

VERSTRAN® (prazepam) Ⓒ IV

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (±15 s.d.) hours before and 70 (±10 s.d.) hours after multiple dosing — a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

The effectiveness of Verstran (prazepam) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51) and 1000 (N 0047-0276-60).

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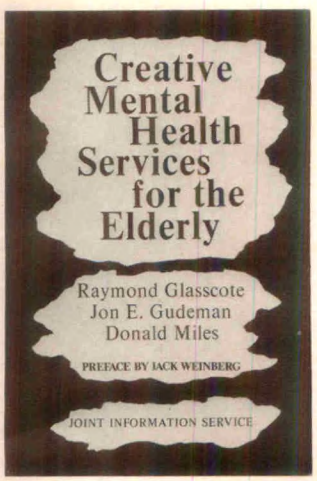
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The Joint Information Service of the American Psychiatric As and the Mental Health Association releases Two New Volum

Focus on the Elderly



This carefully documented book reports a field study of ten programs and six in England and Scandinavia, which serve as practical de creative mental health services for the elderly. They include:

- a novel "Neighborhood Family" providing vigorous support to elc several trailer parks
- a high school for the elderly which sends its students abroad for
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- a "Lucy Booth," patterned after the Peanuts comic strip, whic access to many kinds of services
- and many other innovative, successful approaches.

Each program is subjected to intensive scrutiny. The resulting docu handbook, and, as well, a forum through which some of the world's n practitioners of "the psychiatry of old age" present their views.

Dr. Wilma Donohue, director of the International Center for Soc characterizes this volume as "a masterful job of putting everything guidelines for action are right here."

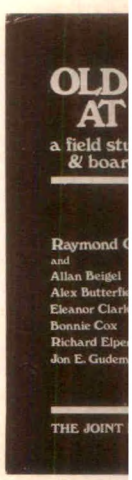
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Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

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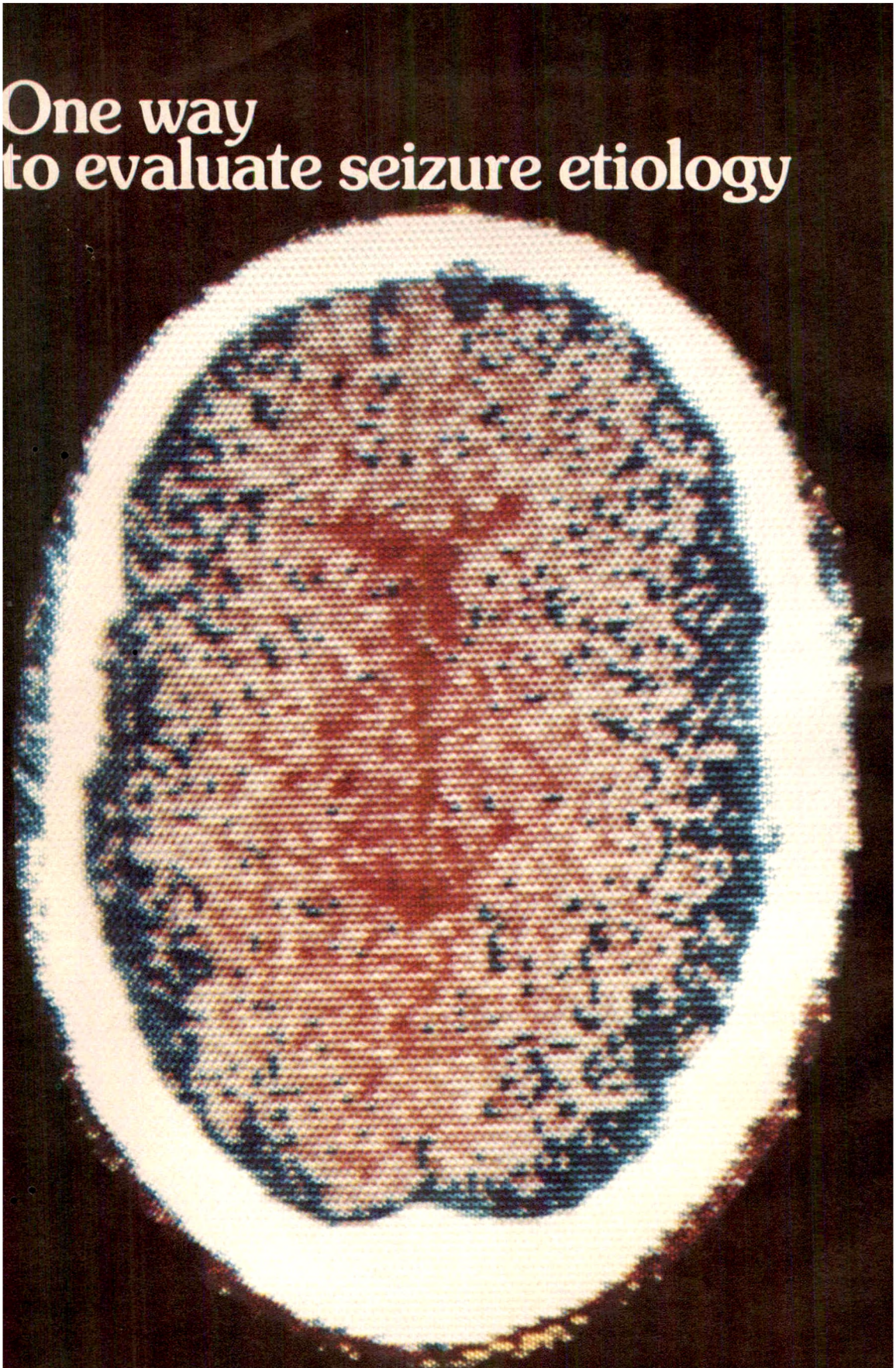
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One way to evaluate seizure etiology



Computerized transaxial tomography (CT scan) is an important new noninvasive technique that can help in determining the etiology of epileptic seizures. Shown here is a single slice from a color-enhanced CT scan of the brain of a patient with epilepsy. The absence of vascular abnormalities, tumor, or other lesions is compatible with a diagnosis of idiopathic epilepsy.

A proven way to start seizure control

MYSOLINE®
(primidone) Tablets 50 mg, 250 mg Suspension 250 mg/5 ml

A single entity with multiple anticonvulsant properties

Primidone itself has anticonvulsant activity. In addition, its major metabolite, phenylethylmalonamide (PEMA), exerts an anticonvulsant effect of its own and also enhances the anticonvulsant action of the second metabolite, phenobarbital. In patients refractory to phenytoin and phenobarbital, primidone may prove effective as the sole therapy.[1]

Multiple indications—grand mal, psychomotor, and focal epileptic seizures

In grand mal, MYSOLINE is comparable in efficacy to phenytoin and phenobarbital.[2,3] In psychomotor seizures, phenobarbital is rarely effective.[1] In focal seizures, phenytoin used alone is not indicated[4]—and, according to Forster,[3] MYSOLINE is markedly more effective than phenobarbital, which should dispel the misconception that MYSOLINE is "just a phenobarbital." (See chart below.)

Efficacy ratings* of anticonvulsants in epilepsy

Agent	Grand Mal	Psychomotor	Focal
Mysoline	++++	++++	++++
Hydantoinates (phenytoin)	++++	+++	+++
Barbiturates (phenobarbital)	++	++	++

*Relative effectiveness, ++++ being most effective.
Adapted from Forster, F. M.[3]

Consider it first for major motor epilepsies

MYSOLINE® (primidone) Ayerst®

See next page for Brief Summary and references.

Start seizure control with MYSOLINE® (primidone)

Initial dose in children over 8 years of age and adults: 250 mg daily at bedtime

BRIEF SUMMARY
(For full prescribing information, see package circular.)
MYSOLINE®
Brand of **PRIMIDONE** *Anticonvulsant*

ACTIONS: MYSOLINE raises electro- or chemoshock seizure thresholds or alters seizure patterns in experimental animals. The mechanism(s) of primidone's antiepileptic action is not known.

Primidone *per se* has anticonvulsant activity as do its two metabolites, phenobarbital and phenylethylmalonamide (PEMA). In addition to its anticonvulsant activity, PEMA potentiates that of phenobarbital in experimental animals.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several weeks before it can be assessed.

Usage in pregnancy: The effects of MYSOLINE in human pregnancy and nursing infants are unknown.

Recent reports suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Data are more extensive with respect to diphenylhydantoin and phenobarbital, but these are also the most commonly prescribed anticonvulsants; less systematic or anecdotal reports suggest a possible similar association with the use of all known anticonvulsant drugs.

The reports suggesting an elevated incidence of birth defects in children of drug-treated epileptic women cannot be regarded as adequate to prove a definite cause and effect relationship. There are intrinsic methodologic problems in obtaining adequate data on drug teratogenicity in humans; the possibility also exists that other factors, *e.g.*, genetic factors or the epileptic condition itself, may be more important than drug therapy in leading to birth defects. The great majority of mothers on anticonvulsant medication deliver normal infants. It is important to note that anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and threat to life. In individual cases where the severity and frequency of the seizure disorder are such that the removal of medication does not pose a serious threat to the patient, discontinuation of the drug may be considered prior to and during pregnancy, although it cannot be said with any confidence that even minor seizures do not pose some hazard to the developing embryo or fetus.

The prescribing physician will wish to weigh these considerations in treating or counseling epileptic women of childbearing potential.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

PRECAUTIONS: The total daily dosage should not exceed 2 g. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 g per day. The initial dose is 250 mg. Increments of 250 mg are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 g. A typical dosage schedule for the introduction of MYSOLINE is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg daily at bedtime	*2nd Week* 250 mg b.i.d.
3rd Week 250 mg t.i.d.	*4th Week* 250 mg q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg, with gradual weekly increases of 125 mg a day, to a daily total usually between 500 mg and 750 mg.

In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances: for initiation of combination therapy; during "transfer" therapy; for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.).

HOW SUPPLIED: MYSOLINE Tablets—No. 430—Each tablet contains 250 mg of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg of primidone (scored), in bottles of 100 and 500.
MYSOLINE Suspension—No. 3850—Each 5 ml (teaspoonful) contains 250 mg of primidone, in bottles of 8 fluidounces.

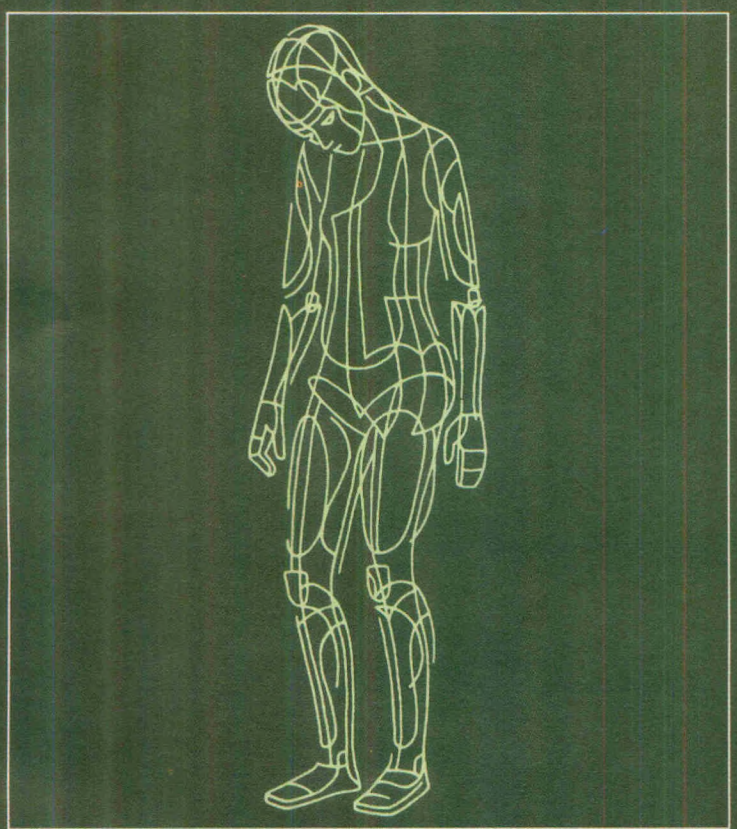
References: 1. Woodbury, D. M., and Fingl, E., in Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics, ed. 5, New York, Macmillan Publishing Co., Inc., 1975, p. 222. **2.** Rodin, E., in Conn, H. F. (ed.): Current Therapy 1977, Philadelphia, W. B. Saunders Company, 1977, p. 716. **3.** Forster, F. M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. **4.** Physicians' Desk Reference, ed. 31, Oradell, N.J., Medical Economics Co., 1977, p. 1175.

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Provides the well established antidepressant effect of amitriptyline with the proven antianxiety effect of Librium® (chlordiazepoxide HCl)

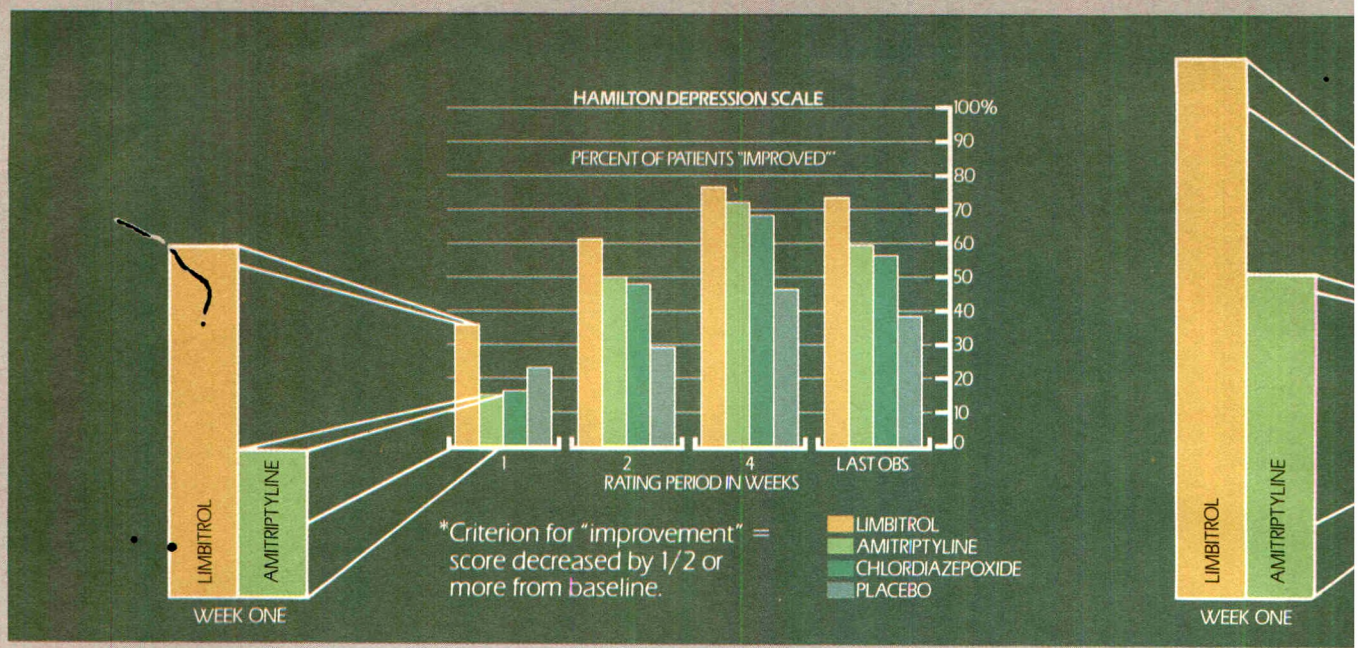


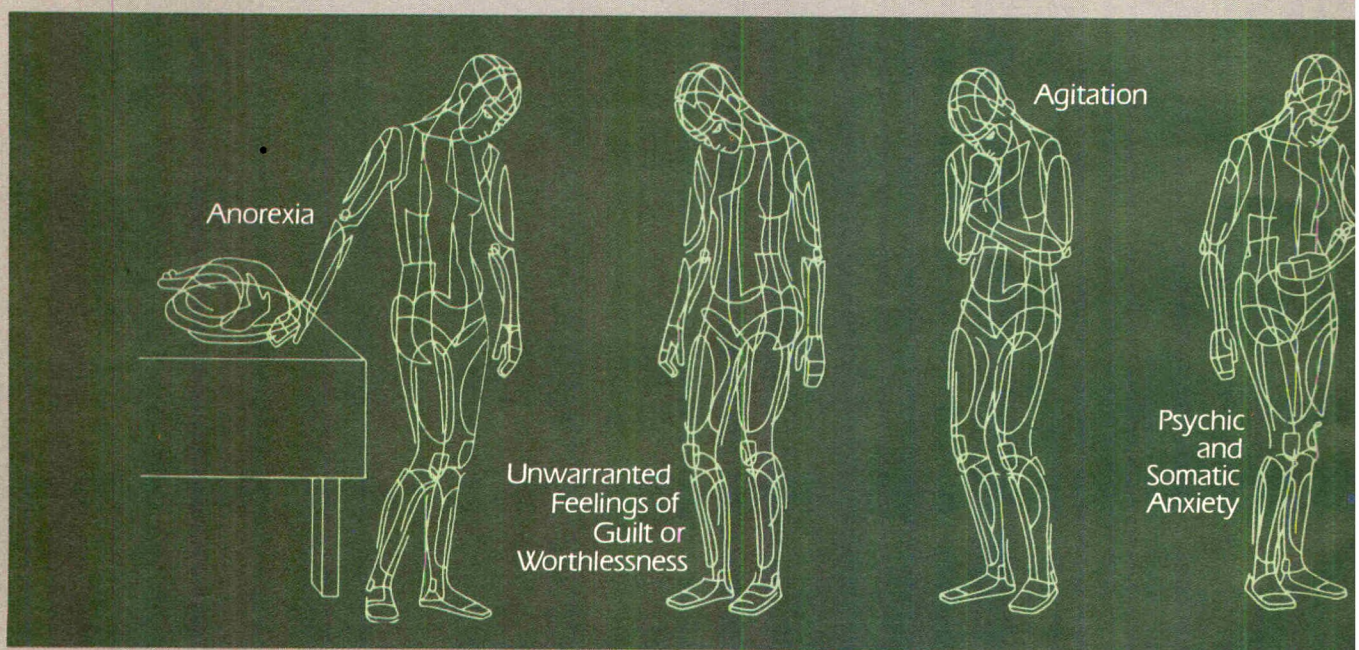
For rapid relief of symptoms in moderate to severe depression and anxiety

In a rigidly controlled nationwide multicenter study, when compared to amitriptyline alone, Limbitrol achieved these superior results...

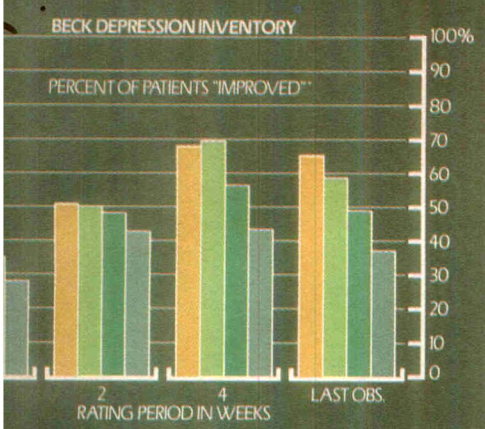
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ROCHE


HAMILTON DEPRESSION SCALE
PERCENT OF PATIENTS "IMPROVED"*
100%
90
80
70
60
50
40
30
20
10
0
1
2
4
LAST OBS
RATING PERIOD IN WEEKS
LIMBITROL
AMITRIPTYLINE
WEEK ONE
*Criterion for "improvement" = score decreased by 1/2 or more from baseline.
LIMBITROL
AMITRIPTYLINE
CHLORDIAZEPOXIDE
PLACEBO
LIMBITROL
AMITRIPTYLINE
WEEK ONE


Anorexia
Unwarranted Feelings of Guilt or Worthlessness
Agitation
Psychic and Somatic Anxiety





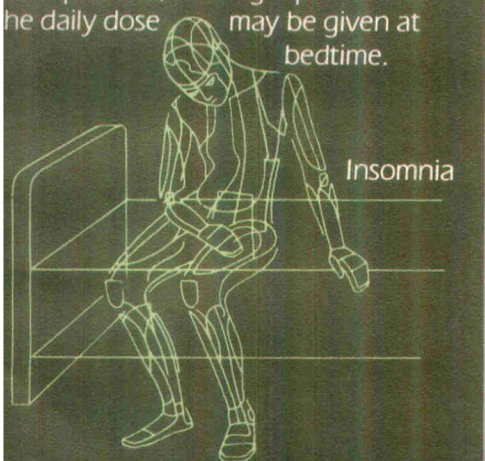
iterion for "improvement" =
ore decreased by 1/3 or
ore from baseline.

Greater patient improvement

Within the first week—

twice as many patients achieved significant relief—sooner than with amitriptyline alone

nsomnia, a major target symptom, can
e relieved sooner by taking advantage
f the sedative effect of Limbitrol. For
ome patients, the larger part or all of
he daily dose may be given at bedtime.



Better relief of symptoms

Within the first week—

symptom severity was reduced to a greater extent than with amitriptyline alone

As measured by the Hamilton Depression Scale

- insomnia
- psychic anxiety
- somatic anxiety
- agitation
- suicidal ideation
- worthlessness

As measured by the Beck Depression Inventory

- pessimism
- dissatisfaction
- guilt
- social withdrawal
- anorexia

Limbitrol®

blets 10-25 each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt)
blets 5-12.5 each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)

or rapid relief of
noderate to severe
epression and anxiety

Greater patient acceptance

- Despite a higher incidence of drowsiness, there was a lower dropout rate due to side effects with Limbitrol than with amitriptyline
  (Limbitrol 4%, amitriptyline 13%, chlordiazepoxide 1%)
- The dropout rate due to therapeutic failure was lower with Limbitrol than with amitriptyline
  (Limbitrol 2%, amitriptyline 6%, chlordiazepoxide 12%)

Please see summary of complete product information on last page of this advertisement.

ROCHE

How to Initiate and Maintain Therapy

Select dosage strength appropriate for each patient

- Limbitrol 10-25 is recommended for most patients
- Limbitrol 5-12.5 may be indicated for those who do not tolerate higher doses and for elderly patients

Specify daily dosage based on symptom severity

- An initial dosage of three tablets is recommended
- Dosage may be increased to six tablets or decreased to two tablets daily as necessary
- Once a satisfactory response is obtained, evaluate response, and if necessary, reduce dosage to smallest amount needed to maintain remission

Utilize dosage options to best accommodate individual patient needs

- T.I.D. or Q.I.D., familiar regimens best suited for patients who tolerate medication without undue drowsiness
- Two tablets one hour before bedtime and one tablet midday may minimize daytime drowsiness and help relieve a common target symptom—insomnia
- Entire dosage h.s. to take maximum advantage of the sedative effect

How to make each patient an informed patient

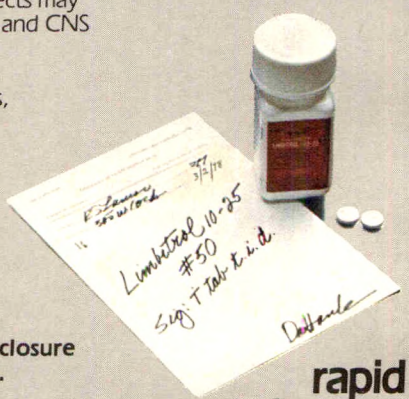
1. Discuss with patients the probability that they will experience drowsiness, especially during the first week.
2. Reassure your patients that drowsiness is one indication that the medication is working and that it may help alleviate their insomnia.
3. Encourage patients to report if drowsiness becomes troublesome so that, if necessary, dosage schedule can be adjusted or the symptom treated.
4. Caution patients about the combined effects with alcohol or other CNS depressants. Let them know that the additive effects may produce a harmful level of sedation and CNS depression.
5. Caution patients about activities requiring complete mental alertness, such as operating machinery or driving a car.
6. Warn pregnant patients and patients of childbearing age that the safety of Limbitrol in pregnancy has not yet been established.

Please see complete product disclosure for other pertinent information.

Limbitrol should not be used under the following circumstances

1. Hypersensitivity to benzodiazepines or tricyclic antidepressants.
2. Should not be given with an MAO inhibitor. To replace an MAO inhibitor with Limbitrol, discontinue MAO inhibitor for a minimum of 14 days before cautiously initiating Limbitrol therapy.
3. During the acute recovery phase following myocardial infarction.

Limbitrol



For rapid relief of moderate to severe depression and anxiety

LIMBITROL® TABLETS
Tranquilizer–Antidepressant

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of moderate to severe depression associated with moderate to severe anxiety.
Contraindications: Known hypersensitivity to benzodiazepines or tricyclic antidepressants. Do not use with monoamine oxidase (MAO) inhibitors or within 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Contraindicated during acute recovery phase following myocardial infarction.
Warnings: Use with great care in patients with history of urinary retention or angle-closure glaucoma. Severe constipation may occur in patients taking tricyclic antidepressants and anticholinergic-type drugs. Closely supervise cardiovascular patients. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, especially high doses. Myocardial infarction and stroke reported with use of this class of drugs.)

Usage in Pregnancy: **Use of minor tranquilizers during the first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Since physical and psychological dependence to chlordiazepoxide have been reported rarely, use caution in administering Limbitrol to addiction-prone individuals or those who might increase dosage; withdrawal symptoms following discontinuation of either component alone have been reported (nausea, headache and malaise for amitriptyline; symptoms [including convulsions] similar to those of barbiturate withdrawal for chlordiazepoxide).
Precautions: Use with caution in patients with a history of seizures, in hyperthyroid patients or those on thyroid medication, and in patients with impaired renal or hepatic function. Because of the possibility of suicide in depressed patients, do not permit easy access to large quantities in these patients. Caution patients about possible combined effects with alcohol and other CNS depressants and against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Periodic liver function tests and blood counts are recommended during prolonged treatment. Amitriptyline component may block action of guanethidine or similar antihypertensives. Concomitant use with other psychotropic drugs has not been evaluated: sedative effects may be additive. Discontinue several days before surgery. Limit concomitant administration of ECT to essential treatment. See Warnings for precautions about pregnancy. Limbitrol should not be taken during the nursing period. Not recommended in children under 12.
In the elderly and debilitated, limit to smallest effective dosage to preclude ataxia, oversedation, confusion or anticholinergic effects.
Adverse Reactions: Most frequently reported are those associated with either component alone: drowsiness, dry mouth, constipation, blurred vision, dizziness and bloating. Less frequently occurring reactions include vivid dreams, impotence, tremor, confusion and nasal congestion. Many depressive symptoms including anorexia, fatigue, weakness, restlessness and lethargy have been reported as side effects of both Limbitrol and amitriptyline. Granulocytopenia, jaundice and hepatic dysfunction have been observed rarely.
This list includes adverse reactions not reported with Limbitrol but requiring consideration because they have been reported with one or both components or closely related drugs:
Cardiovascular: Hypotension, hypertension, tachycardia, palpitations, myocardial infarction, arrhythmias, heart block, stroke.
Psychiatric: Euphoria, apprehension, poor concentration, delusions, hallucinations, hypomania and increased or decreased libido.
Neurologic: Incoordination, ataxia, numbness, tingling and paresthesias of the extremities, extrapyramidal symptoms, syncope, changes in EEG patterns.
Anticholinergics: Disturbance of accommodation, paralytic ileus, urinary retention, dilatation of urinary tract.
Allergic: Skin rash, urticaria, photosensitization, edema of face and tongue, pruritus.
Hematologic: Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia.
Gastrointestinal: Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, black tongue.
Endocrine: Testicular swelling and gynecomastia in the male, breast enlargement, galactorrhea and minor menstrual irregularities in the female and elevation and lowering of blood sugar levels.
Other: Headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, jaundice, alopecia, parotid swelling.
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. I.V. administration of 1 to 3 mg physostigmine salicylate has been reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestation and treatment.
Dosage: Individualize according to symptom severity and patient response. Reduce to smallest effective dosage when satisfactory response is obtained. Larger portion of daily dose may be taken at bedtime. Single *h.s.* dose may suffice for some patients. Lower dosages are recommended for the elderly.
Limbitrol 10-25, initial dosage of three to four tablets daily in divided doses, increased up to six tablets or decreased to two tablets daily as required. Limbitrol 5-12.5, initial dosage of three to four tablets daily in divided doses, for patients who do not tolerate higher doses.
Supplied: Limbitrol 10-25 tablets each containing 10 mg chlordiazepoxide and 25 mg amitriptyline (as the hydrochloride salt) and Limbitrol 5-12.5 tablets each containing 5 mg chlordiazepoxide and 12.5 mg amitriptyline (as the hydrochloride salt)—bottles of 100 and 500, Tel-E-Dose® packages of 100; Prescription Paks of 50.

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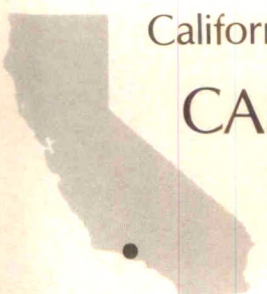
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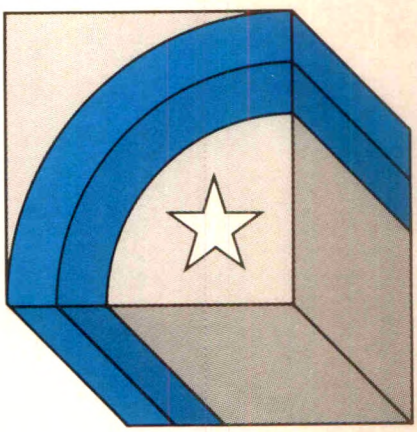
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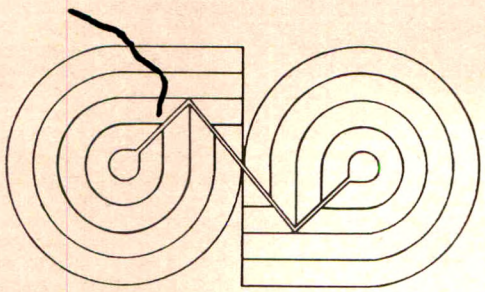
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a consideration beyond efficacy alone...

Infrequent akathisia and other disabling extrapyramidal effects...

The rapidly rising readmission rate among discharged psychotic patients is mainly due to noncompliance with antipsychotic drug therapy. And this, in turn, may be largely attributed to disabling extrapyramidal side effects, notably akathisia.[1]

Although extrapyramidal effects are characteristic of antipsychotic agents in general, with Mellaril (thioridazine) such effects are infrequent. Adding an antiparkinsonian agent—which can cause its own side effects—can usually be avoided. Mellaril (thioridazine) is contraindicated in patients with severe hypotensive or hypertensive heart disease.

Extrapyramidal Effects of Selected Antipsychotic Agents*[2]

DRUG	EXTRAPYRAMIDAL EFFECTS
Chlorpromazine	Moderate
Perphenazine	High
Prochlorperazine	High
Fluphenazine	High
Acetophenazine	Moderate
Trifluoperazine	High
Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2-6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170-171.

Mellaril® (thioridazine)

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP
CONCENTRATE: 100 mg per ml

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.
Store and dispense Concentrate below 86°F; use tight, amber glass bottle. Just prior to administration, Concentrate may be diluted with distilled water, acidified tap water, or suitable juices; preparation and storage of bulk dilutions is not recommended.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.
It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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Infrequent akathisia and other disabling extrapyramidal effects...



a consideration beyond efficacy alone for the discharged patient who needs an antipsychotic agent

Mellaril® [thioridazine]

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP
CONCENTRATE: 100 mg per ml

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For brief summary, please see preceding page.

SANDO
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The Psychodynamics and Developmental Psychology of the Borderline Patient: A Review of the Literature

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The author reviews the psychodynamics and developmental psychology of the borderline patient as described in the literature on intensive therapy, early mother-child interaction, and family interaction. Focusing on the borderline patient's characteristic difficulties in intimate relationships, he describes the patient's use of splitting and projective identification as seen in the characteristic transference-countertransference interaction in intensive therapy. These primitive defensive mechanisms, which are also utilized by family members, appear to contribute to a failure of empathic responses both during the child's early development and in the family interactions during his adolescence. The author concludes that conceptual attempts to relate adult and child phenomena, although highly speculative, create new and useful perspectives for the treatment of the borderline patient.

THERE IS a growing body of work on the dynamics and developmental psychology of the borderline patient. The literature springs from three main sources: observations of the transference-countertransference interaction in intensive psychotherapy or analysis, observation of the preverbal infant in interaction with his mother, and study of the family in interaction. Despite the active controversy about the nondefinitive boundaries of the diagnostic classification of borderlines (1–3) and the continual attempts to reach bett[illegible] diagnoses (4), clinicians have found [illegible] clarity about the underlying dynamic [illegible] mental issues of great use in wor[illegible] therapeutically with these patients and [illegible] ing and putting into perspective the ter[illegible] velop in the work.

In this paper I will survey the major [illegible] ture and attempt to synthesize the curre[illegible] ing of the many dynamic and developme[illegible] volved. The literature is large and is fi[illegible] condensed metapsychological abstracti[illegible] resent attempts to make sense of subt[illegible] data. I will attempt, when possible, to [illegible] concepts into clinical language without [illegible] sary violence to the complexity of the [illegible]

The focus of the review will be on [illegible] patient's characteristic difficulties in int[illegible] ships (5). There are specific element[illegible] psychopathology that engage both [illegible] and therapists in unique ways. I will a[illegible] these observations as they have bee[illegible] both the dynamic and the developmen[illegible]

CONTRIBUTIONS OF DESCRIPTIVE PSYCHOLOGICAL STUDIES

Although the attempt to apply a des[illegible] to the presence and frequency of sym[illegible] observable abnormal behavior of the [illegible] tient is important (2, 4, 6), it may be [illegible] of the characteristic behavioral and [illegible] sponses of these patients seem to app[illegible] of stress, while at other times the pa[illegible] tively asymptomatic (7). Nonetheless [illegible] from descriptive studies and from re[illegible] logical test reports are congruent wit[illegible] vations from intensive therapy.

Gunderson (4) and Grinker and We[illegible]

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most discriminating data in their descriptive studies to be in the observation of the difficulties borderline patients have in interpersonal relationships, particularly in terms of their pervasive sense of aloneness and their tendency to see difficulties as emanating from outside themselves. Gunderson[1] suggested from his data that these attributes, as well as an inability to acknowledge wants and to discriminate them from needs, are "core conflicts" in borderline patients.

On unstructured projective tests that provide the opportunity for regressive responses, borderline patients reveal evidence of primary process thinking (7), a strong tendency to add too much and too specific affect to simple perceptions, and a propensity to produce these peculiar responses without concern or embarrassment. Margaret Singer's review of series of test reports (8) revealed that these responses do not occur on more structured tests, suggesting that these patients can utilize an external organizing structure to support their thinking. This finding has been corroborated by repeated clinical observations (2, 9–11). Melvin Singer (12) suggested the presence of a cognitive disturbance in which these patients fail to structure, delay, detour, and check their impulses and affects through "cognitive binding" (i.e., through contemplation, fantasy, reflection, and symbol formation).

These findings suggest that borderline pathology is related to difficulties in the management of impulse and affect, which is perceived most characteristically in interpersonal relationships and emerges most clearly in relatively unstructured settings. The intensive, relatively unstructured therapeutic relationship, then, is an ideal setting in which to study in detail the elements of the disorder.

TRANSFERENCE DEVELOPMENT AND PROBLEMS

The response of borderline patients in intensive therapy is characteristically marked by gross fluctuations in perception, thinking, and feeling about themselves and the therapist, without awareness of the contradictions (13, 14). Margaret Singer (8) found this tolerance and bland acceptance of contradictory thoughts a unique attribute of these patients.

Kernberg (7) described the borderline patient as having little capacity for realistic appraisal of others and, in the absence of a relationship, as experiencing others as distant objects to whom he can compliantly adapt. In therapy borderline patients present narrow, rigid, unmodulated affects (15) and show little ability to tolerate guilt, concern, or internalized depressive affect (16, 17). They have difficulty in discriminating and reporting body sensations and emotions (13) and manifest little capacity for anxiety or frustration tolerance, delay, or impulse control (7). Their unpleasant affects readily escalate to panic without triggering reliable defensive operations (18), and they manifest a profound sense of identity diffusion, with little sense of inner continuity from past to present to future (15). Unlike the majority of psychotic patients, they are capable of higher level defenses and good reality testing in many situations, but they are likely to regress to transient psychotic states under stress or within the transference relationship, readily developing fantasies of extreme neediness, destruction, and abandonment (7, 19).

The transference of the borderline patient has been described as immediate, intense, and chaotic, only gradually developing over time into discernible patterns (7, 15). Modell (20–22) described the patient's diagnostic transference response to the therapist as similar to the infant's response to the "transitional object" (e.g., blanket or teddy bear). He suggested that although the patient recognized the therapist as existing outside the patient (as is not the case in the transference of many psychotic patients), the quality of this perceived existence is determined by processes arising within the patient. Although neurotic patients also attribute certain qualities of their own to a therapist, they seem to have the capacity to recognize the origins of these perceptions within themselves, while borderline patients do not (5).

The transference response described by Modell is seen most clearly in the borderline patient's difficulty in asking the therapist for help in a way that would imply an acknowledgment that the therapist as a separate person might reasonably refuse or be unable to respond to the request. For the borderline patient, to consider the possibility of the therapist's refusal is to become aware of anxious, rage-filled fantasies of abandonment that are perceived as potentially arising from the therapist. To avoid these fantasies, the patient does not ask directly for help. Instead, he either angrily demands a response or overtly ignores the therapist in the session while maintaining an illusion of comfort from the therapist's presence. This response is seen as similar to the child's response to his blanket.

Winnicott (23) described the child's blanket as something that possesses attributes of its own (warmth, texture, something "not me") yet is not allowed to change unless changed by the child. It is "used" by the child—both loved and mutilated—and must survive both (24). As in the therapeutic relationship described by Modell, the blanket is not "asked" for help. It is, instead, symbolically placed as a protective shield between the child and the dangers of the outside world; the child is comforted by it. Several authors (23, 25–27) have described the relationship with the transitional object as representing a phase in the child's gradual separation from his mother, a gradual disengagement in which the blanket partly represents his mother (and the child) and partly represents the outside world. Abelin (28) described the father as occupying this position during the child's separation-individuation period.

[1]Gunderson JG: Discriminating characteristics of borderlines and their families (unpublished paper, 1977).

In the early stages of therapy, some borderline patients use inanimate objects, animals, or certain fantasies as transitional ways of relating to the therapist, metaphorically putting these objects between them and the therapist both to protect them from a dangerous intimacy and simultaneously to allow a relationship to exist (25, 26).

Borderline patients demonstrate their characteristic unmodified, polarized responses in the therapeutic relationship (15, 22). They have magical expectations from therapy, often anticipating improvement without their own active participation. They may experience the therapist as benevolent but demonstrate the constant fear that he or she will be transformed into the opposite. Under stress, they may experience the therapist as attacking and may fear that they will be dominated, controlled, or abandoned by him (19, 29). In the latter experiences they feel alone, helpless, and needy and appear unable to maintain a reliable sense that the therapist remains with them in a caring way (19). Because of these tensions, they present the manifest conflict of extreme dependence and intense fear of closeness, the solution to which appears to be a constant preoccupation with maintaining the "proper" distance from the therapist in order to regulate their anxiety about both distance and closeness.

EGO DEFENSES

The use of specific primitive ego defenses has been described as characteristic of the borderline patient in intimate relationships. Kernberg (7, 30), in describing the borderline patient's disjointed, contrasting behaviors and fantasies in intensive therapy, suggested that there is an underlying pattern of sharply polarized fantasied relationships that is "activated" in the transference. The polarization of these fantasies is determined by the defensive maneuver labeled "splitting." Kernberg suggested that borderline patients' core difficulty lies in their inability to bring together and integrate loving and hating aspects of both their self-image and their image of another person. Kernberg's observation was that these patients cannot sustain a sense that they care for the person who frustrates them. Kernberg saw this characteristic failure in the achievement and tolerance of ambivalence and in the modification of affects as diagnostic. He suggested that loving fantasied relationships and hating ones are internally "split" for the borderline patient to prevent the anxiety that would result if they were experienced simultaneously. These positive or negative stereotyped fantasies are activated in a relationship depending on the degree of gratification or frustration perceived by the patient. In a gratifying relationship, the patient develops positive fantasies, with the negative ones dissociated or "split off" and therefore unavailable. When frustrated, the patient elaborates negative fantasies and loses all memory of the positive relationship.

Other authors (1, 4, 13, 31, 32) ha[illegible] comfortable with the idea of splitting [illegible] mechanism for borderline patients. Mo[illegible] acknowledge the patient's failure to [illegible] modify impulses and affects as an imp[illegible] istic.

Splitting as a defense is to be distin[illegible] nial, which is the disavowal of whole ob[illegible] substitution of a wish-fulfilling fanta[illegible] sion, which is the repulsion of menta[illegible] consciousness due to a linkage of that [illegible] conflicted memory (33). In splitting, th[illegible] negative fantasied relationships remai[illegible] consciousness with the complement[illegible] ated.

Defensive splitting also contributes t[illegible] alternating view of the therapist as on[illegible] valued. In these responses to the thera[illegible] experiences no real dependency on h[illegible] love or concern. Instead, the patient [illegible] sudden shift from total need to total de[illegible]

A second major defense of borderline [illegible] associated with both splitting and trans[illegible] ness, is that of projective identificatio[illegible] defense involves the dissociation of [illegible] pects of the personality (elements of i[illegible] age, or superego) and the projection o[illegible] other person, resulting in an identific[illegible] other person because of having attribu[illegible] the self to him. Included in this intrap[illegible] personal mechanism, particularly for [illegible] tients, is the unconscious attempt to [illegible] behavior in the other that conform w[illegible] and the willingness (conscious or not) [illegible] of these projections to accept these at[illegible] of himself (32).

The person using this defense [illegible] attentive to the real aspects of the oth[illegible] tradict or invalidate the projection. [illegible] the other (now seen as possessing the [illegible] acteristics) is consciously identified as [illegible] (36), while an unconscious relationship [illegible] which the projected attribute can be vi[illegible] rienced.

This defense differs from projection [illegible] vidual maintains some contact with the [illegible] jected. The projections are not simply [illegible] other; an unconscious attempt is mad[illegible] relationship with the other and to invol[illegible] lusive partner in conforming to the v[illegible] perceived (37, 40).

Projective identification is a defens[illegible] spectrum of psychopathology from norm[illegible] ic. The severity of the pathology is det[illegible] content of the projection, the capacity [illegible] test reality and to differentiate himse[illegible] person, and the intensity of the subj[illegible] need to avoid conflict by disavowing a [illegible] of himself. Depending on the interactio[illegible] tors, projective identification can end[illegible]

ship with emphatic qualities or create a delusional distortion and give a binding quality to the interaction (38).

Of central importance in the borderline patient's use of this defense are its consequences in terms of a weakening of his or her ego functioning. Klein (34) noted that projection of aggressive aspects of the self may result in a loss of attributes important for the development of power, potency, strength, and knowledge. Kernberg (30) suggested that the resultant failure of integration leads to incomplete development of internalized standards (e.g., guilt and internal delay of impulses) and other internal "structures" (18).

Similarly, according to Klein (34), the chronic projection of positive aspects of the self (as seen in primitive idealization) may result in extreme dependency, loneliness, fear of parting, and fear of the loss of the capacity to love. Kernberg (7) suggested that the borderline patient's use of idealization in its primitive form involves no regard for the real person who is idealized but represents a use of the other as a "thing"—an "all positive" magical protection in a dangerous world (3, 7).

The borderline patient has been described as having a peculiar "empathy" for the unconscious impulses of other people (41, 42) and as having little or no ability to recognize the mature defenses, reaction formations, or other adaptive ego functions manifested by the other person in his struggle with these impulses (7, 43). This intuitive sensitivity allows for a powerful coercive use of projective identification because there is often some degree of perceptual accuracy in the projection that touches a conflicted area of the recipient. When frustrated and projecting their anger in therapy, for instance, borderline patients may utilize this intuition to provoke a response in such a way as to justify their distrust and may then destroy, devalue, or reject all the therapist has to offer (44).

COUNTERTRANSFERENCE

The use of these primitive defenses contributes to the transference-countertransference difficulties often seen in intensive treatment. Many authors have described the powerful countertransference responses evoked by the borderline patient (11, 13, 14, 19, 20, 22, 45). Unlike the more gradually developed, complex, empathic understanding that characterizes his or her response to neurotic patients, the therapist's response to the borderline patient's transference appears to be more rapid, intense, and stereotyped. Different therapists discover similar responses to these patients; the response seems less related to specific problems of the therapist's past and more to the patient's poorly integrated impulses (7).

Because of their defensive inability to integrate loving and angry aspects of the same internal image, borderline patients are inordinately sensitive to minor frustrations in treatment. In the face of such frustrations, their characteristic response is to withdraw all affectively positive perceptions of the therapist and reinvest them elsewhere. At this point, patients "lose" all memory of positive experiences with the therapist and feel abandoned. Through the mechanism of projective identification, they experience the therapist as attacking and rejecting and respond defensively to control their now projected rage (46). Their defensive response may include rage, devaluation, withdrawal, or paranoid thinking and an inability to call up the previously experienced image of a comforting therapist (19).

If the therapist exposed to these raw affective interchanges undergoes an "empathic regression" (7) in an attempt to keep in touch with the patient, he may become vulnerable to countertransference guilt and anxiety. Many therapists who have devoted their lives to healing are concerned at some level about their capacity to hurt, which borderline patients unconsciously sense. In his introspective attempt to understand the patient, the therapist might find a punitive aspect of himself at whom the patient might "accurately" be angry. Such a counteridentification might evoke guilt in the therapist which, when perceived by the patient, would validate the patient's projection, making it difficult for the therapist to work on it collaboratively with the patient as a projection (46, 47). This blurring of ego boundaries between patient and therapist might contribute to the familiar regressive transference psychosis in which patients' reality testing is lost, their unmodified rage and guilt increase, and they experience the therapist as a reincarnation of the fantasied early frustrating parent.

Similarly, the unexpected and uncontrolled intensity of the patient's rage may make the therapist vulnerable to his own anxiety, which is potentially disruptive to the therapeutic work. Such anxiety in the therapist could be related to the possible loss of the relationship with the patient (with specific countertransference implications) or the need to manage his own hatred and need to retaliate (45, 48). In response to these countertransference feelings, Kernberg (7) suggested that otherwise sophisticated therapists may at times experience an almost masochistic submission to the patient's aggression, disproportionate doubts in their own capacity, and exaggerated fears of criticism by third parties.

In their description of the therapeutic task with borderline patients, several authors have discussed the need for the therapist to withstand these unmodified affects without withdrawal or retaliation. The therapeutic position is characterized as the provision of a "holding environment" (49), a response that includes the need 1) to reflect, absorb, transform, and feed back the patient's responses (7), 2) to maintain contact, concern, and emotional availability (50), 3) to set limits for the patient to help him or her manage internal and external stimuli, and 4) to accept the patient's ambivalence with continuing concern (19). This description of the therapeutic response in situations in which inter-

pretation appears to have no effect (51) has its parallels in the developmental observations described below.

OBJECT RELATIONS THEORY

Much of the data derived from the above observaitons has been used by theorists in the development of object relations theory (7, 21, 33, 52–55). The basic assumption behind this theory is that one can understand the relationships between people through an examination of the internal images they have of one another. In the healthiest people, these images correspond rather accurately to the reality of the other person and are continually reshaped and reworked as new information is perceived and integrated. In less psychologically healthy people, the images are stereotyped, rigid, and relatively unchanged by new information. The theory proposes a model for the developmental shaping of these images as the child begins to separate the image of himself from that of his mother. Anna Freud (56) characterized this developmental line as proceeding from dependency to emotional self-reliance to adult relationships.

According to the theory, there are at least three major developmental tasks in this progression: 1) self-object differentiation, 2) the integration of loving and hating images and the development of object constancy, and 3) the further integration of these images into flexible psychic structures (for example, superego and ego ideal).

These ideas are based primarily on clinical observations of transference relationships with psychotic, borderline, and neurotic patients and are inferentially related to early child development. Self-object differentiation refers to the child's capacity to separate intrapsychically the boundaries of his own experience from his experience of his mother. Failure in this task is presumably seen in psychotic patients who have difficulty in recognizing the therapist's independent existence. The attainment of object constancy (or the "depressive position") (16, 34) is a consequence of the child's capacity to recognize himself simultaneously as both loving and hating in response to the image of another who is both gratifying and frustrating. Accomplishment of this task presumably allows the child to tolerate and master separation and loss by being able to maintain a comforting image of the mother (a "constant object image") despite her frustrating the child by her absence (11, 56). Further, the capacity to acknowledge angry feelings toward the image of a loved person leads to the development of the capacity for reality testing and the capacity to tolerate guilt, concern, and "internalized depression" (11, 56). "Internalized depression" in this sense refers to an experience of mourning and regret over lost "good" internal images, rather than the more primitive sense of impotent rage and defeat by external forces (7). Failure in this achievement of object constancy is presumably characteristic of borderline patients who have mastered self-object differentiation but ca[illegible] ol[illegible] separation or ambivalence without regre[illegible]

This object relations view of borderline [illegible]ology of interest in that it suggests a basis for [illegible] object constancy. Presumably, the child [illegible] strong enough image of his positive rel[illegible] his mother to be able to tolerate the inte[illegible] images of negative interactions with his [illegible] out being overwhelmed. Kernberg (7, [illegible] that this failure of integration and the [illegible] splitting is due to excessive aggression, [illegible] tutional or from excessive or chronic [illegible] mental frustration.

Horner (57) described the person who [illegible] these tasks as "a separate individual w[illegible] sense of self and other, who is able to [illegible] as whole persons rather than simply [illegible] who can tolerate ambivalence . . . and [illegible] ability to sustain his own narcissistic ec[illegible] resources within the self." Further integ[illegible] cation, and "depersonification" of thes[illegible] relationships evolves into such psychic [illegible] superego and ego ideal (7, 30).

DEVELOPMENTAL PSYCHOLOGY

Many psychoanalytically oriented re[illegible] study preverbal infant development foc[illegible] tion on the intimate mother-child relatio[illegible] of particular interest to the student of [illegible] chopathology. The literature, howeve[illegible] warnings about the dangers of direc[illegible] from infant observations to adult phen[illegible] Since the infant has a unique cognitive [illegible] little capacity for conscious memory a[illegible] bolization, it has been suggested that th[illegible] er's inferences are likely to be distort[illegible] spective falsifications and adultomorph[illegible] pathy" (62). Several observers (63, 64) h[illegible] that the closest one can get to preverba[illegible] the adult patient is that which is filte[illegible] fantasies of the oedipal period, when [illegible] tal and verbal capacities begin to app[illegible] closely those of the adult. Theodor[illegible] warned of the danger of the "great or[illegible] which suggests that the first year of lif[illegible] what follows that its stamp is irrepara[illegible] the alternative hypothesis that each [illegible] stage presents a new opportunity for [illegible] zation that allows earlier experiences [illegible] toxic.

Others (34, 54, 65) have argued that [illegible] ness of the infant's mental apparatus a[illegible] all-encompassing internal struggle to d[illegible] self from a symbiotic unity with the m[illegible] such a core, global, organizing expe[illegible] traces must be seen (particularly if u[illegible] subsequent stages. At the very least, [illegible] derived from this struggle seem to be a[illegible]

petitive dynamic issues with which the borderline patient struggles.

As described above, the clearest, most comprehensive formulation about the relationship between borderline psychopathology and developmental psychology is that of Kernberg (30) and the object relations theorists (54). Kernberg suggested that borderline pathology is a consequence of a developmental failure occurring after self-object differentiation but before the development of object constancy. He described this failure as marked by a continuing defensive use of splitting, with resultant limitations in the capacity to test reality, to tolerate anxiety and frustration, and to sustain a stable, integrated relationship with an underlying attitude of basic trust (7, 46).

This formulation is apparently applicable to a large number of borderline patients, but it has been questioned by some authors, who have found two discernible groups or at least two different qualities of experience within the borderline population. These two qualities are variously described as schizoid versus aggressive (19), constricted versus expansive (12), compliant versus aggressive (13), obsessive ("as-if," "false self") versus hysterical (66), and "those who have given up" versus "those who are still searching" (3). Although Kernberg's formulation appears to apply to the second group in each of these pairs, it does not seem adequately to describe members of the first group, with their distant, "pseudoautonomous" functioning and their difficulty in engaging the other in anything but a compliant manner (13). Clearer formulations about both groups emerge from the developmental data of the symbiotic and separation-individuation periods (ages 0–3), when the issues of intrapsychic separation and the development of formed images first appear.

Symbiotic Period

Mahler (6) described the symbiotic period as extending from the second month (when the infant begins tactilely and visually to explore the mother) to the fifth month (when the infant begins to explore beyond the mother). She defined this period as characterized by the infant's acting as though his internal experience of himself and his mother were joined by a common boundary with no recognition of separateness.

During this period the tasks of the mother are to satisfy the infant's needs, to buffer and modify the stimuli he receives (both internal and external), and in effect to act as an auxiliary ego. Preparation for these tasks is facilitated by a regression in the mother (49, 68) that increases her empathy, reawakens fantasies of her own childhood experiences, and helps develop her capacity to provide a "holding environment" (49) in which the infant's "absolute need for empathy" is met.

Mahler (67) described the "mutual cuing" that must take place between infant and mother for normal symbiosis to take place. The infant's contribution to this interaction requires attributes of both perceptual ability (i.e., the capacity to receive and seek nurturance and attachment and to link patterns of experience) and stimulus sensitivity (i.e., sensitivity to touch and visual responses). The mother's tasks require her recognition and acceptance of her own infantile impulses, with a resultant continuing empathic responsiveness to similar impulses in her child.

Unacknowledged conflict in the mother in interaction over time with specific attributes of the child may result in an empathic failure that interferes with this "optimal symbiosis." Some authors (39, 42, 57, 69–71) have suggested that in such situations the infant may prematurely inhibit his dependency on his mother in order to respond to her defensive needs, a response that may result in premature ego development. Failure in the mother-child interaction during this period has been suggested as the origin of pathology for the first quality of experience within the borderline spectrum (i.e., the "pseudoautonomous" complaint, detached response).

If, for example, because of unconscious conflict about her own needs, the mother disavows them, sees them as "bad," and projects them onto her child, she may persistently withdraw from or be relatively unresponsive to the child's actual needs because of the confusion with hers (projective identification). Several authors have suggested that such chronic nonresponsiveness is experienced by the child as a meaningless interruption, resulting in his eventual detachment (39, 70, 71) and formation of a "false self" (51) in which the child compliantly adapts to his mother's needs with relative abandonment of his own, since they represent a threat to a mother whom the child absolutely needs and who is relatively immune to his feedback (70).

This detached self is the consequence of a premature severing of the symbiotic tie before any internalization of maternal functions can occur. It leaves the child with a sense of detachment (72), a profound emptiness, and an undeveloped potential for the direct and personal experience of living (70). It is a distancing maneuver, presumably developed by the child to ward off the negative experience of the mother's chronic empathic failure and misinterpretation. The pseudoself is that which validates the mother's projection (70). According to Friedman (70), Winnicott suggested that the failure of interpretation in the therapy of patients with such false self-organization is due to their perception of the interpretation as another "act of attribution" by a nonempathic parent.

Separation-Individuation

Mahler and associates (27) divided the separation-individuation period (5 months–3 years) into four subphases: 1) differentiation (5–8 months), 2) "practicing" (8–16 months), 3) rapprochement (16–25 months), and 4) object constancy (25 months–3 years). Within this period the child's task is to emerge gradually from the symbiotic unit and to develop increasing autonomy both internally and externally. Successful

completion of this developmental period results in the capacity for frustration tolerance, the mastery of separation anxiety, and the maintenance of self-esteem.

It is during these subphases of separation-individuation that the issues central to the dynamics of borderline psychopathology first appear. It is here that the conflict between the push for autonomy and the wish to unite is for the first time apparent in the behavior of the child.

During the differentiation subphase (5–8 months), the child remains anchored to the mother but develops an increasing alertness, an outer orientation, and finally, a social smile that increasingly focuses on the mother (59, 73). In her absence the child gets restless and unhappy, but he smiles on her return. This fact suggests the initial development of "recognition memory": the ability for perception to revive a memory trace (73, 74).

At 8 months the infant develops "stranger anxiety." Mahler and associates (27) observed that if there is a good symbiotic period the infant reacts to strangers with curiosity and wonderment; if it is less good the infant demonstrates more anxiety. These observations are congruent with Kernberg's formulation (30) that a greater quantity of frustration during symbiosis would threaten the infant's loving experiences, resulting in a prolonged defensive splitting and projection of fearsome negative experience onto the "stranger."

In the "practicing" subphase (5–16 months), the infant develops the capacity for locomotion with an exuberance and excitement that temporarily enables him to ignore the presence of his mother. Exhausted by autonomous movements, the infant periodically returns to the mother for "emotional refueling" (27), a brief contact that rapidly enables the infant to restore his or her energy and momentum. During this subphase there is evidence that the child begins to notice and tolerate his mother's departure, since he tones down his activities ("low keyedness") in her absence (27).

Kernberg (7, 30) suggested that the child begins to be able to differentiate between positive images of himself and his mother during this period; similar self-object differentiation within images of negative experiences occur somewhat later. He suggested, however, that this differentiation of images is still tentative at this point and vulnerable to "regressive refusion" at times of stress.

The rapprochement subphase (16–25 months) is the period during which the conflict between autonomy and reunion is demonstrated most clearly in the child's behavior and response. Even the language of the descriptions evokes images familiar to therapists of borderline patients. It is in this phase that the toddler's behavior indicates a sense of increased separateness, loneliness, helplessness, a constant concern with the mother's presence, and increased separation and stranger anxiety. It is during this period, according to Kernberg (7, 30), that the child develops firm boundaries between the experience of himself and that of his mother. The infant now responds to [illegible] fueling place, he responds to *her*.

Observers of the child in this subp[illegible] scribed the toddler's active interest [illegible] presence and his developing need for [illegible] tively in his autonomous achievemen[illegible] noted two normal patterns: shadowing [illegible] and darting away expecting to be swep[illegible] Inferred from this behavior is both th[illegible] ion and the fear of engulfment.

In children who have difficulties [illegible] phase, Mahler (58) observed increase [illegible] havior manifested by rapidly alterna[illegible] and repudiation of the mother. McD[illegible] that the less a child can assert himse[illegible] independent play, the greater the freq[illegible] sive clinging and repudiation. The imp[illegible] observations is that significant conflic[illegible] in the mother-child relationship if [illegible] does not support the child's develop[illegible] ward autonomy.

With the development of the capacity [illegible] tational thought" (75) and symboliza[illegible] develops the use of transitional objects [illegible] use his relationship with his father mo[illegible] as a way to mediate the shift from t[illegible] with his mother toward autonomy (28

At about 18 months the infant develo[illegible] both to retain the memory of a concret[illegible] has disappeared ("object permanence" [illegible] the image of the mother in her abse[illegible] memory") (74, 75). Throughout mo[illegible] ment, although the image of the moth[illegible] cur to the child, it does not seem to p[illegible] absence, suggesting an incomplete in[illegible] the mother's caring functions and a[illegible] on her actual presence. Kernberg (7[illegible] that when the child is frustrated by h[illegible] ture, he is overwhelmed with rage an[illegible] image of the loving relationship with h[illegible] To preserve this comforting fantasy [illegible] sociates it and sees himself as a "ba[illegible] doned by an angry mother. The child [illegible] profound separation anxiety in the fo[illegible] ness and a negative sense of self, with [illegible] the "good" images.

The task of the mother during thi[illegible] tolerate sufficiently her own wishes [illegible] and autonomy so that she can respon[illegible] pathic way to corresponding wishes [illegible] out seeing them as "bad" or dangero[illegible] ing with retaliation or withdrawal (4-9 [illegible] associates (76) described two kinds o[illegible] ure. One group of mothers who had d[illegible] ating their own dependency responde[illegible] anger to their child's clinging by say[illegible] minute ago you didn't want me, no[illegible] you." Presumably, this response wou[illegible] in the child about his wishes for nurtu[illegible] group of mothers with conflicts abo[illegible]

sponded to the child's autonomous moves with, "You think you can manage on your own, well go ahead." This response threatens the child with abandonment and inhibits his freedom to explore because of the reactive fantasy: "If I grow up, I'll be all alone."

The implication drawn from these observations is that this kind of chronic withdrawal represents the mother's defensive inability to distinguish between her own unresolved needs (for the child's response to her) and her recognition of the needs of her child for autonomy and support. This defensive response from the mother is contributed to by her own conflicts and by the increased aggressiveness of her 2-year-old during the anal period.

Adler (44) described the "good enough mother" (49) of this subphase as one who is not afraid of her own or her child's anger and can be firm when she needs to frustrate. She has confidence in her basic goodness and a capacity to care without having to retaliate for old hurts. The descriptions of Mahler and associates (27) indicate that there is a broad spectrum of "good enough" mothering responses. Typical failures in the interaction between mother and child, then, are likely to represent chronic empathic insensitivity from mothers at the ends of this spectrum in interaction with particularly sensitive infants.

Toward the end of rapprochement, the child's cognitive apparatus sufficiently matures so that he can notice that the same mother both gratifies and frustrates. Winnicott (24) suggested that if the mother can "contain" the child's aggression (by nonanxious, firm limit setting) while continuing to provide love and understanding, she will demonstrate to him that she is not created or changed by his impulses and that she is separate from him and not a creation of his projections. Such a response allows the child to develop a relationship with her as a real and autonomous person and helps the child to tolerate his anger and put it in perspective by allowing him to recognize that it cannot destroy his loving mother (by turning her into a bad, angry, or anxious mother). In addition, the mother's response will help strengthen the child's conviction in the strength of his good self-image and that of his mother and will decrease the child's fear of his own aggressive tendencies (7).

Alternatively, if the mother, because of unacknowledged conflict revived by the interaction, retaliates for her infant's aggression or withdraws from his demands, she will confirm his fear of aggressive fantasies, making it difficult for him subsequently to integrate them. Responding to the withdrawal, the infant generates increased rage and greater demands (e.g., clinging), which contributes to a spiraling crisis in their interaction and an intensification of the infant's need for defensive splitting and projection, the phenomena characteristic of borderline patients.

With successful negotiation of rapprochement the child develops the capacity for ambivalence and object constancy (27, 74). The ambivalently held image of the mother is more stable in the face of frustration, providing the infant an inner sense of comfort and contributing to his or her capacity to experience guilt, concern, depression, and increasing anxiety tolerance. The infant's image of himself now begins to develop increasing complexity, affects are modulated, ego control of impulses is increased, and there is a clearer sense of self and others. Observers of the child during this subphase (59, 73) have noted that with this development there is a shift from self-centered, demanding, clinging behaviors to more mature expressions of affection, trust, confidence, regard for the interests and feelings of others, cooperation, sharing, making sacrifices, and offering gifts.

Latency

The relative absence of clinical descriptions of borderline phenomena in the literature of the latency period (ages 6–10) suggests a relative decrease in symptomatic behavior during this period. Chiland and Lebovici (77), however, described the two borderline qualities of inhibition and excitability as emerging in some children during latency in response to the disciplinary and learning requirements of school. These children were described as rejecting attempts at communication and refusing to carry out tasks. Despite high learning performance, they were described as exploding periodically in rage. The authors commented on the characteristic contrast between good intellectual development and poor relationship modalities in these children that marked their "developmental disharmony." Again, in these descriptions, the two types of borderline presentation were seen: cold, detached, and removed versus subject to unpredictable outbursts of rage. The authors also commented on the probable use of projective identification by these children, noting that "the interlocutor himself feels the whole burden of anxiety."

Adolescence

Several authors (65, 78, 79) have perceived a recapitulation of the dynamic themes of separation-individuation during the adolescence of the borderline child, a period when borderline symptomatology often becomes apparent. With my colleagues at NIMH (78, 79) I observed a family group regression that occurs during the adolescence of the borderline patient in which family members respond with retaliation or withdrawal to autonomous or dependent behaviors of the adolescent. The conflict between the striving for autonomy and the fulfillment of dependency needs revived in adolescence is inadequately resolved in these families because of a regressive, shared use of projective identification (38) in which the adolescent becomes the bearer of disavowed aspects of the parents. In some families the child's dependent wishes are seen as devouring demands from which the family members withdraw. This withdrawal often represents parental anxiety about their own wishes to be given to, which they cannot acknowledge. In other families, the adolescent's autonomous strivings are interpreted as a

hateful abandonment of the family, which the parents cannot accept. In these families parental strivings for autonomy are disavowed, seen as bad, and projected onto the adolescent, resulting in a defensive inability to respond to the adolescent's autonomous development with the necessary support.

Masterson and Rinsley (65, 80) described the latter group in terms of the adolescent's "abandonment depression," which is defended against by splitting, acting out, and other pathological maneuvers. They viewed as the "basic dynamic theme" of the borderline patient that "the borderline mother withdraws emotional supplies at her child's efforts to separate and individuate." This is a characteristic phenomenon observed by several authors (42, 78). The consequence of this maternal withdrawal in the face of separation is that the child is torn between the engulfment of symbiosis if he moves toward his mother and the experience of loss and abandonment if he moves away. Zinner and my study of the families of these patients in interaction (78) revealed that this retaliatory withdrawal of supplies is a shared family dynamic that is only one element in a family regression involving all family members.

The borderline adolescent's alienation, aggressive responses, and inadequately structured self-image (81) contribute to this family turmoil, but parental contributions are also significant. Our analysis of family interactions (79) revealed that these parents depend heavily on projective identification for their own defensive organization and that in this process there is considerable collusion from the child. We also observed a blurring of parental capacity to experience themselves as separate from the particular child in the areas of conflict and an idiosyncratic but often overlapping content of parental projections involving disavowed wishes for dependency or autonomous strivings (78, 79).

Although Masterson (82) found the parents of the borderline patient to be borderline themselves, this finding does not agree with our observations of significant variability in parental psychopathology (79) or with the observations of Margaret Singer (8), who found that 84% of borderline patients had normal or neurotic parents according to psychological tests. Although some authors (6, 65)[1] have discussed these parents in descriptive terms (i.e., "ineffectual, passive father," "intrusive, dependent mother"), other authors (78, 79, 83) have described a more dynamic, shifting tension between parent and child.

The latter observers illustrated how family members' use of primitive defenses may be encapsulated in response to issues involving certain offspring. In interaction with siblings or the outside world, they may use more advanced ego defenses and greater maturity but may regress to fixation points relating to deficient resolution of symbiosis and identity formation in relation to the designated borderline child. Singer and Wynne (83) commented on the discrepancy between the severity of the disturbance when family members interact compared with the relatively mild distu[illegible] family member shows individually. They [illegible] that in attempting to understand the famil[illegible] it is insufficient to look only at the indivi[illegible] relation to the individual child. Their [illegible] (78, 79, 83) indicate that there are contri[illegible] the entire family group that affect the nat[illegible] velopmental interference.

DISCUSSION

The continuing study of the psychopat[illegible] borderline patient has inevitably led to [illegible] interest in and examination of a two-per[illegible] gy. The remnants of early symbiotic int[illegible] in the interpersonal use of projective id[illegible] the borderline patient are evident in the [illegible] pist interaction, the mother-child respo[illegible] family life of the borderline adolescent. [illegible] the complex fantasy life of the neurotic [illegible] his more differentiated and articulated p[illegible] intrapsychic conflict (usually involving [illegible] tasies), the borderline patient presents [illegible] typed, primitive difficulties, frequently [illegible] nonverbally. Often the key to a comp[illegible] these difficulties lies in the affects and [illegible] tasies of the caretaker (e.g., therapist o[illegible]

The growing experience in intensive w[illegible] patients has led to a closer study [illegible] transference phenomena (7, 30), the setti[illegible] work" of the therapeutic interaction (2[illegible] ture of the "holding environment" (10, [illegible] components and complexities of empat[illegible] ures (10, 24, 36–40, 44, 48, 57, 62, 71, 7[illegible] conscious communicative process invo[illegible] interaction (78, 79, 83). In addition, the [illegible] chological defense has been reevaluate[illegible]

Although Klein (34, 35) focused her st[illegible] tive identification on the nature of the [illegible] fantasies, more recent work has focused [illegible] orative nature of this defense (38, 39, [illegible] 85). Much of this work supports the 'de[illegible] an entire range of interpersonal defense[illegible] collaboration of a particular other is nec[illegible] tain defensive equilibrium.

Certainly, unconscious conflict in t[illegible] ternalized in her relationship with the [illegible] to be related in some way to difficulties [illegible] of "adequate symbiosis" as well as in [illegible] choreographed movements required fo[illegible] ration-individuation. Unconscious fant[illegible] fensive collaborative use of particular fa[illegible] to represent disavowed aspects of inne[illegible] family of the borderline adolescent seem[illegible] icant impediment to development. It [illegible] seen how these family interactions affec[illegible] mental matrix in which the infant's ear[illegible] self develops. This question remains a re[illegible] plored frontier of developmental theory

Although there remains significant controversy about the applicability of child observation to adult phenomena as well as serious debate about the possibility of recreating elements of the relationship between infant and mother in the therapeutic situation (86), conceptual attempts to make these comparisons have produced fascinating new insights. The delineation of the therapist's "holding" function, of his task of "containing" the patient's affects and projections, and of the hazard of his own projections interfering with the therapeutic process (46, 84, 85) have all emerged in part from this study of the borderline patient and his development. All seem in some way to have relevance to the developing understanding of the complex preverbal interaction between infantile impulses and human environmental responses from which personality evolves.

The increasing overlap of the observations, formulations, and inferences derived from the three different areas of study reviewed above suggests at least an approximation to reality that future follow-up studies should address. The data from the current studies have already made an important contribution to the therapist's ability to listen better to particular elements of these patient's productions, to develop more accurate empathic responses, and to provide borderline patients with some coherent organization for their chaotic experience.

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Bilateral and Unilateral ECT: Effects on Verbal and Nonverbal Memory

BY LARRY R. SQUIRE, PH.D., AND PAMELA C. SLATER

The memory loss associated with bilateral and nondominant unilateral ECT was assessed with verbal memory tests known to be sensitive to left temporal lobe dysfunction and with nonverbal memory tests known to be sensitive to right temporal lobe dysfunction. Bilateral ECT markedly impaired delayed retention of verbal and nonverbal material. Right unilateral ECT impaired delayed retention of nonverbal material without measurably affecting retention of verbal material. Nonverbal memory was affected less by right unilateral ECT than by bilateral ECT. These findings, taken together with a consideration of the clinical efficacy of the two types of treatment, make what appears to be a conclusive case for unilateral over bilateral ECT.

ELECTROCONVULSIVE THERAPY (ECT) has long been considered an effective treatment for depressive illness (1, 2). The memory loss associated with this treatment has been well documented (3–5). For example, following conventional bilateral treatment, memory loss can extend to events that occurred many years before treatment as well as to events that occur during the weeks after treatment. Memory functions gradually improve as time passes after treatment (6).

It has been generally accepted that right unilateral ECT is a clinically effective treatment that produces less impairment of new learning capacity and less amnesia for remote events than bilateral ECT (7–13). However, since right unilateral ECT is specifically associated with impairment in nonverbal memory (e.g., memory for spatial relationships, faces, designs, and other material that is difficult to encode verbally (14–17), and since most studies of ECT and memory loss have employed verbal memory tests, the actual extent of memory loss associated with right unilateral ECT has remained somewhat unclear. It has been suggested that the amnesic effects of left or right unilateral ECT may be similar to the effects of left or right temporal lobe dysfunction (18). Accordingly, if memory were assessed with nonverbal tests specifically sensitive to right temporal lobe dysfunction, the amnesic effect of right unilateral ECT might prove to be as great as or even greater than that of bilateral ECT.

Only two studies have addressed this issue directly, employing verbal and nonverbal memory tests with patients receiving bilateral or right unilateral ECT. In the first study (15) impairment in one nonverbal test was somewhat greater after bilateral ECT than after unilateral ECT, but this difference was not statistically significant. In the second study (16) the results were ambiguous. Impairment in a nonverbal test was greater in the unilateral group after 4 treatments, but greater in the bilateral group 3 months after treatment. That study was further complicated by the fact that one-third of the patients given unilateral treatment did not have a grand mal seizure. Finally, since it was not clear how patients with identified right unilateral lesions would perform on the nonverbal tests used in these two studies, it was difficult to be sure how specifically sensitive the tests were to right hemispheric dysfunction.

The present study investigated memory functions in patients receiving bilateral or right unilateral ECT. Assessments of memory were made with two verbal tests known to be sensitive to left temporal lobe dysfunctions and two nonverbal tests known to be sensitive to right temporal lobe dysfunction.

METHOD

Subjects

The subjects were 72 psychiatric inpatients (53 women and 19 men), from 4 private hospitals, who had been prescribed a course of ECT. The diagnoses as recorded upon admission by the psychiatrists were depression (N=55; this diagnosis included designations of primary affective disorder, involutional melancholia, manic-depressive, and psychotic depression),

Some of the data reported here were included in a review of ECT and memory loss presented at the 129th annual meeting of the American Psychiatric Association, Miami Beach, Fla., May 10–14, 1976.

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neurotic depression (N=11), schizo-affective disorder (N=5), and hysterical personality (N=1). Patients with neurological disorders, schizophrenia with depression, depression secondary to alcoholism or drug abuse, and patients who had received ECT during the previous 12 months were excluded from the study. Most of the patients (N=45) had not received ECT before; 27 had received ECT 1 to 15 years earlier.

The 72 patients in the study were assigned to 3 groups (table 1). Group 1 consisted of 33 patients who had been prescribed bilateral ECT. Group 2 consisted of 21 patients who had been prescribed right unilateral ECT. The choice of bilateral or unilateral ECT depended on the preferences of the individual psychiatrists and was therefore not random. However, since the patients about to receive bilateral or unilateral treatment did not differ measurably on their memory test scores before ECT (figure 1), it seems reasonable to assume that group differences emerging after ECT can be attributed to the type of ECT administered. Group 3, a control group, consisted of 18 randomly selected patients who were only tested before receiving a course of ECT. Fourteen of these patients were scheduled to receive bilateral ECT and 4 right unilateral ECT. All subjects were determined to be strongly right-handed; they reported that they did not use their left hand for any everyday activity and had no left-handed parent or sibling.

TABLE 1
Characteristics of 72 Patients Receiving ECT

Item	Group	
	Bilateral ECT (N=33)	Right Unilat ECT (N 2
Age (years)		
Mean	43.2	34 7
Range	19–64	22 64
Sex		
Male	12	3
Female	21	18
Years of education		
Mean	12.8	12 8
Range	10–18	8 18
Number with prior history of ECT	10	8

ECT

ECT was administered three times a week on alternate days following medication with atropine, methohexital sodium, and succinylcholine. Bilateral and unilateral treatments were administered using a Medcraft B-24 machine. For bilateral treatment electrode placement was temporal-parietal; for unilateral treatment both electrodes were placed on the right side of the head, as described by McAndrew and associates (19) (N=19) and by D'Elia (7) (N=10). Amnesic effects of nondominant unilateral ECT have been reported to be similar despite wide variation in electrode placement (20, 21). The stimulus parameters (140–170 v for .75–1.0 seconds) were sufficient to induce a grand mal seizure throughout the course of all treatments.

Tests and Procedures

Two memory tests, each consisting of a verbal and a nonverbal portion, were employed.

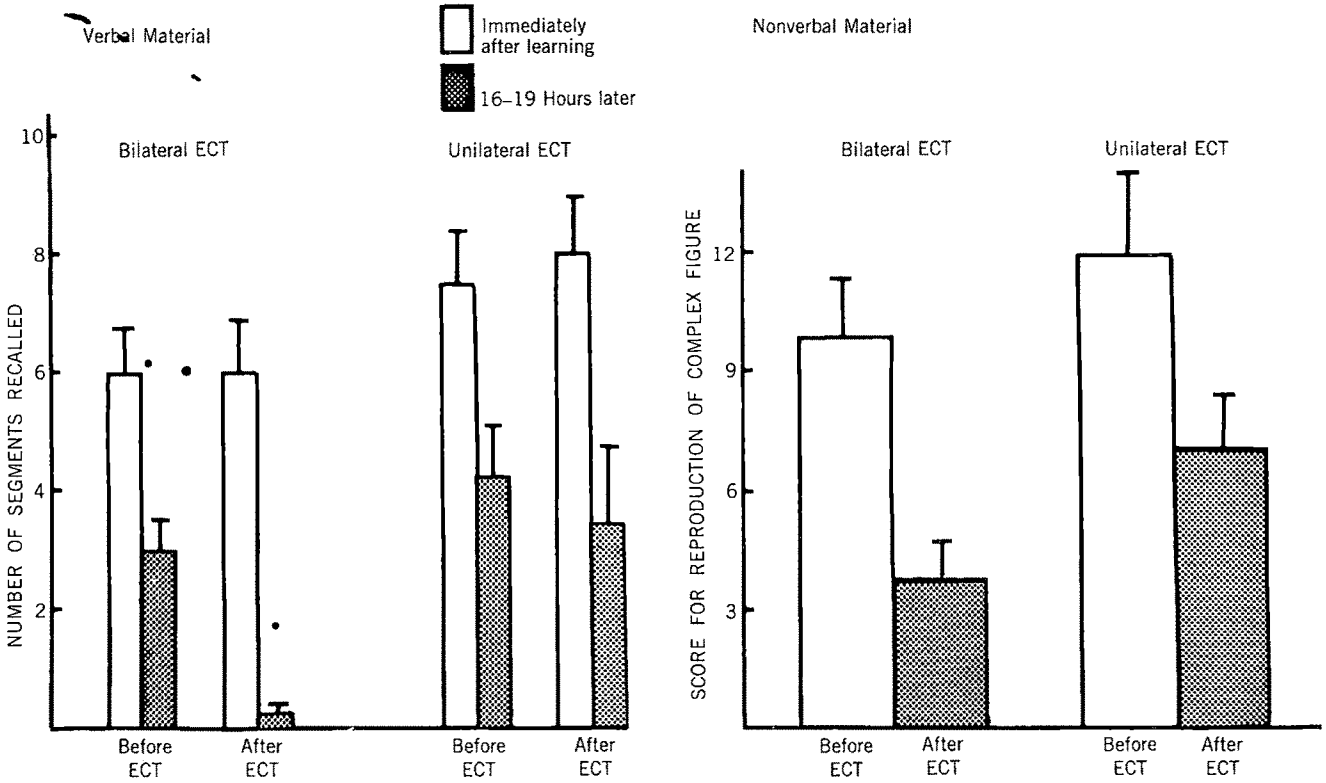
Test 1A (verbal portion: story recall). A short paragraph was read to the subject (6). Patients with identified dysfunction of the left temporal lobe are known to perform more poorly on this test than patients with dysfunction of the frontal parietal or right temporal region (22). Immediately after hearing the story, and again the next day (16–19 hours later), subjects were asked to recall as much as they could remember of it. The paragraph was divided into 20 segments, and the score was the number of segments recalled. Eighteen patients receiving bilateral ECT and 13 receiving right unilateral ECT were tested before treatment and again, with an equivalent form of the te after the fifth treatment of the series.

Test 1B (nonverbal portion: memo figure). Subjects copied a complex geo (the Rey-Osterrieth figure [23] or the [24]) and were then asked to reproduce ry 16–19 hours later. Patients with righ sions are known to be deficient on this patients with left temporal lesions exhi ment (25). The score for this test de number of properly placed line segmen score=36 points). The same patients (above) were tested with one of these ECT and with the other one 6–10 hour treatment.

Test 2A (verbal portion: short-te tractor test). Subjects were shown a gram, distracted for a variable interva seconds), and then asked to recall the c Patients with left temporal lesions are i task; patients with right temporal lesions Subjects received 8 trials at each rate and their score was the number of co rectly recalled without regard to order. score was 24. Fifteen patients receiving were tested on two occasions with equi this test. These sessions were schedule ter the first treatment and 2–3 hours treatment of the series. In addition, 8 p ing right unilateral ECT were tested 2- their first and third treatments. Fina were tested on one occasion 1–2 days b treatment.

Test 2B (nonverbal portion: spatial jects attempted to remember the posi circle located along an 8-inch horizonta with right temporal lesions are impair patients with left temporal lesions are jects inspected the circle on the line for then were distracted for 6, 12, or 24 seco ing strings of random digits into numeric subjects attempted to mark on a differe the remembered position of the circle

FIGURE 1
Retention of Test 1 Verbal and Nonverbal Material by Patients Receiving Bilateral ECT (N=18) and Right Unilateral ECT (N=13)



trials were given, with 8 at each of the three retention intervals. The score on each trial was the distance (in millimeters) between the position of the originally presented circle and the position of the circle as marked by the subject. The score on the test at each retention interval was the total error (in millimeters) for all 8 trials. Test 2B was given on the same occasions and to the same patients as test 2A (above).

RESULTS

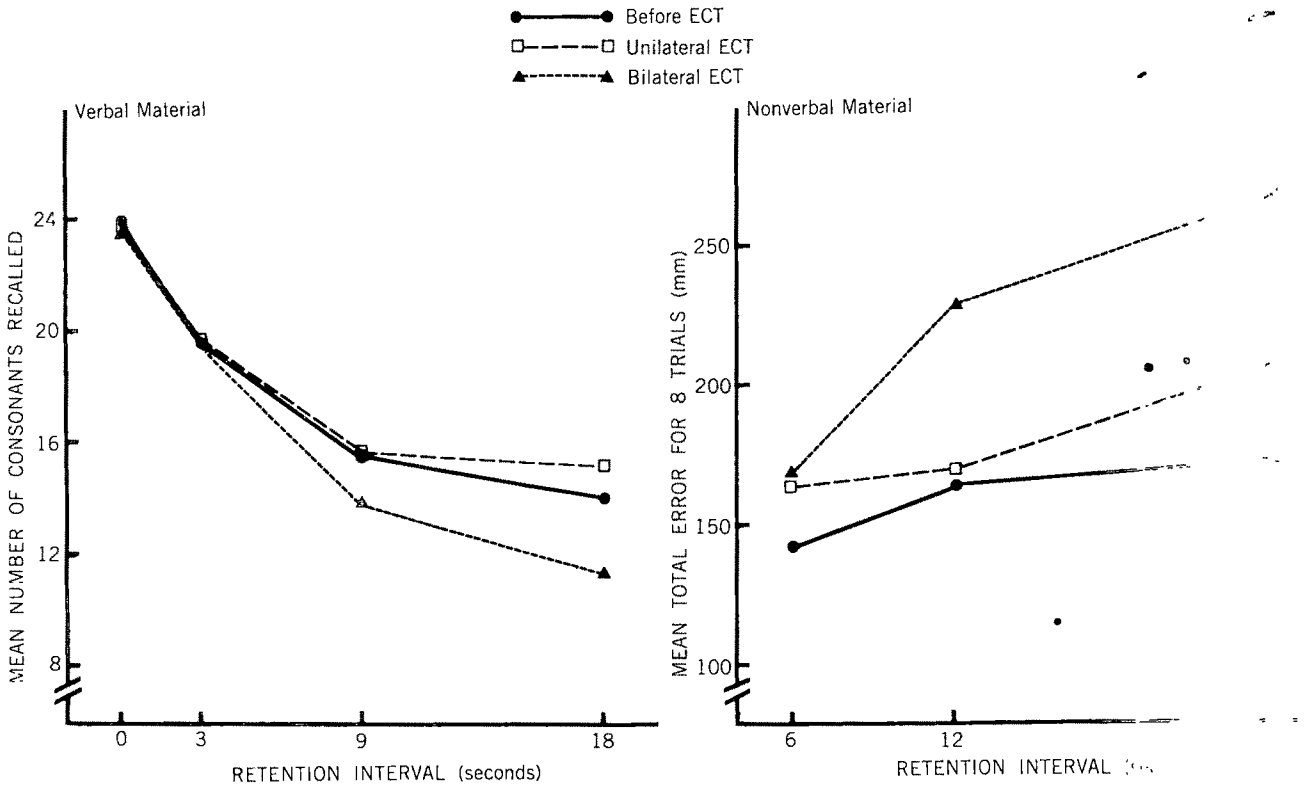
Figure 1 shows the results with test 1 for patients who received bilateral or unilateral ECT. Before ECT these two groups of patients did not differ from each other on any of the measures of immediate or delayed recall (for the verbal test, $t<1.5$, $p>.10$; for the nonverbal test, $t=0.7$, $p>.10$). After ECT patients receiving bilateral treatment were able to remember verbal material immediately after hearing it as well as they could before ECT (before ECT versus after ECT, $t=0.1$, $p>.10$), and they were able to copy a complex figure as well as before ECT ($t=0.1$, $p>.10$). However, their performance was severely impaired on delayed tests of verbal and nonverbal memory (verbal test: before ECT versus after ECT, $t=5.6$, $p<.01$; nonverbal test: before ECT versus after ECT, $t=3.7$, $p<.01$).

Right unilateral ECT did not affect verbal memory, as measured by test 1A. That is, the delayed recall scores of patients receiving right unilateral treatment were about the same after ECT as before ($t=0.6$, $p>.10$). However, nonverbal memory was significantly impaired by right unilateral ECT (test 1B). Before unilateral ECT the score for reproducing the geometric figure after a delay was 11.9, and after unilateral ECT the corresponding score was 7.1 ($t=2.7$, $p<.05$). This impairment in nonverbal memory associated with unilateral ECT was not as great as the impairment in nonverbal memory associated with bilateral ECT ($t=2.1$, $p<.05$).

Figure 2 shows the results with test 2 for patients receiving bilateral ECT, patients receiving right unilateral ECT, and a control group of patients about to begin a course of bilateral or unilateral ECT. For the short-term memory distractor test, patients receiving bilateral ECT were impaired, but patients receiving right unilateral ECT performed normally. An analysis of variance with repeated measures on one factor (28) indicated that the scores of bilateral patients were significantly lower than those of both unilateral patients ($F=10.8$, $p<.01$) and control patients ($F=5.7$, $p<.01$). The scores of unilateral patients and control patients were not measurably different ($F=0.8$, $p>.10$).

For the spatial memory test bilateral ECT also produced a marked impairment (bilateral group versus control group, $F=22.4$, $p<.01$). The scores of unilateral patients were also poorer than those of control patients, although this difference fell short of significance

FIGURE 2
Recall of Test 2 Verbal and Nonverbal Material by Patients Receiving Bilateral ECT (N=15), Right Unilateral ECT (N=8), and by Control Subjects About to Begin a Scheduled Course of ECT (N=18)



(F=2.64, p=.12). Finally, the effect on nonverbal memory associated with unilateral ECT was not as great as the effect associated with bilateral ECT (F=9.6, p<.01).

DISCUSSION

The results can be summarized by three main conclusions.

1. Bilateral ECT markedly impaired the ability to retain both verbal and nonverbal material.
2. Right unilateral ECT impaired the ability to retain nonverbal material without measurably affecting memory for verbal material.
3. The impairment in nonverbal memory associated with right unilateral ECT was less than the impairment in nonverbal memory associated with bilateral ECT.

The findings that bilateral ECT markedly affected memory and that right unilateral ECT exerted a material-specific effect on nonverbal memory are consistent with the results of a number of studies of ECT and memory loss (3–5, 7). However, it should be noted that the extent to which bilateral or right unilateral ECT impairs memory depends on the sensitivity of memory tests to the effects of ECT. For example, in the present study right unilateral ECT had no measurable effect on verbal memory; yet performance on some verbal memory tests can be impaired by right u[illegible] ment (10, 12). Accordingly, it is diffic[illegible] the amnesic effects of bilateral and ri[illegible] ECT unless these effects are assesse[illegible] study using the same tests.

The present study employed memory te[illegible] be sensitive to either left or right temp[illegible] function. The results clearly indicated tha[illegible] right unilateral ECT on both verbal a[illegible] memory was less than that of bilateral[illegible] sometimes been assumed that right u[illegible] produces as much memory dysfunction[illegible] ECT on those aspects of memory functi[illegible] with the right hemisphere. To our kno[illegible] study reported here is the first to clea[illegible] strate that right unilateral ECT produc[illegible] ory dysfunction for nonverbal materia[illegible] ECT.

The therapeutic efficacy of bilateral [illegible] ECT has been compared in a large num[illegible] (for reviews see references 29 and 30). T[illegible] these studies indicate that courses of b[illegible] lateral ECT are approximately equivale[illegible] to similar reductions in depressive sym[illegible] sociated with similar relapse rates, and [illegible] efficacy at follow-up. One review (29) [illegible] that the slight disadvantage in immed[illegible] sometimes reported for unilateral treatme[illegible]

the apparently widespread impression[1] that unilateral ECT is not as effective as bilateral ECT, may be due to occasional failures to produce a maximal seizure with the unilateral technique. Since the therapeutic effect of ECT is bound to the seizure (32), even one submaximal seizure during a course of unilateral treatment could account for reported slight differences between unilateral and bilateral ECT. Several practical suggestions to ensure that unilateral ECT produces a grand mal seizure have been outlined (29).

When given properly, unilateral ECT seems to be clearly preferable to bilateral ECT since the risks to verbal and nonverbal memory are less than for bilateral treatment. It should be noted that some risks to memory exist even for unilateral ECT. The benefits to be derived from this procedure should therefore be weighed carefully against these risks and against the possible risks of alternative therapies to form a basis for clinical judgment.

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[1]A recent survey of members of the American Psychiatric Association conducted by the APA Task Force on ECT indicated that of 3,000 respondents, 75% of those who used ECT used bilateral ECT for all of their patients (31).

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Withdrawal Syndromes Associated with Antipsychotic Drugs

BY GEORGE GARDOS, M.D., JONATHAN O. COLE, M.D., AND DANIEL TARSY, M.D.

Withdrawal symptoms frequently follow abrupt discontinuation of antipsychotic compounds. In addition to other somatic symptoms, withdrawal-emergent dyskinesias may be observed. "Covert dyskinesia" refers to a masked form of tardive dyskinesia that becomes clinically detectable only after antipsychotic drugs are withdrawn or their dosage is reduced. Withdrawal dyskinesia appears under similar circumstances but disappears spontaneously in 6 to 12 weeks. Cholinergic overactivity and changes in dopamine-acetylcholine balance in the basal ganglia may underlie these withdrawal syndromes. The principal value of the concept of covert dyskinesia is in the secondary and tertiary prevention of tardive dyskinesia through early discovery and treatment.

THE EFFECTS of withdrawal of antipsychotic drugs have not been adequately studied. In usual clinical practice, antipsychotics are withdrawn abruptly with no expectation of untoward effects other than the possibility of psychotic relapse. There are at least two compelling reasons to focus attention on the sequelae of drug discontinuation. Withdrawal symptoms that develop may in some cases mimic symptoms of psychotic exacerbation and therefore may result in unwarranted resumption of drug therapy. In addition, dyskinesias may be seen following antipsychotic drug withdrawal, including a form of tardive dyskinesia that is suppressed during drug administration. The dose-related suppression of dyskinetic movements by dopamine-blocking antipsychotic drugs is probably the major reason for the widely differing prevalence rates of tardive dyskinesia (ranging from .5% to 40%, according to Crane [1]) reported in the literature. Withdrawal-emergent dyskinesia, like withdrawal symptoms, may be confused with signs of psychotic relapse (2); hence the careful assessment of pa[illegible] postwithdrawal weeks is more than an exercise.

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AUTONOMIC AND BEHAVIORAL SYM[illegible] WITHDRAWAL

During the first week or two followi[illegible] antipsychotic drugs, a number of soma[illegible] may occur. These symptoms, aptly labe[illegible] effects" by Lacoursiere and associates [illegible] consist of one or more of the following: [illegible] ing, diarrhea, perspiration, restlessness, [illegible] norrhea, headaches, increased appeti[illegible] ness. Although some drug studies have [illegible] withdrawal symptoms (4–6), it seems [illegible] that such symptoms were not looked [illegible] sequently may have been missed. In [illegible] where careful observations were made [illegible] incidence of withdrawal symptoms rang[illegible] to 75%.

Symptoms typically begin during the [illegible] after drug discontinuation, peak during th[illegible] and disappear by the end of the second [illegible] affected patients have only one or two [illegible] which are usually mild enough to be elic[illegible] direct questioning. Symptoms usually fa[illegible] occasional severe cases may require tem[illegible] ment with antihistamines or sedatives. [illegible] withdrawal appears more likely to induce [illegible] symptoms than gradual withdrawal (9). [illegible] relationship has been demonstrated [illegible] withdrawal dosage and the incidence o[illegible] symptoms (3, 7, 13). In one study (11) a [illegible] greater proportion of women than men ga[illegible] syndrome.

Antipsychotic drugs all possess centra[illegible] eral antimuscarinic properties to varying [illegible] 15). It is tempting to postulate that at lea[illegible] drawal symptoms may be due to a choline[illegible] occurring when antipsychotic drugs are [illegible] The following observations support this [illegible] with marked anticholinergic properties, [illegible] promazine and thioridazine, are assoc[illegible] highest rates of withdrawal symptoms (3[illegible] same may be true for clozapine, a very [illegible] cholinergic new antipsychotic agent (16[illegible] drawal of piperazine phenothiazines, whi[illegible] weaker anticholinergic agents (14), rare[illegible]

symptoms unless antiparkinsonian drugs are simultaneously withdrawn (3, 17, 18). Imipramine, an antidepressant with marked anticholinergic effects, also yields a high incidence of withdrawal symptoms (19). Finally, most of the common withdrawal symptoms, e.g., nausea, vomiting, and sweating, can be produced by the acute administration of physostigmine, a drug with marked central and peripheral cholinergic actions (20, 21); these same symptoms can be abolished by anticholinergic agents (20).

EXTRAPYRAMIDAL COMPLICATIONS OF WITHDRAWAL

Parkinsonism

Drug-induced parkinsonism tends to improve following drug withdrawal (22). However, cases often persist for at least 4 months (23, 24), and, in rare cases, subtle signs have persisted for as long as 9–18 months (25). When antiparkinsonian drugs are withdrawn at the same time as antipsychotics, parkinsonian symptoms of rigidity, tremor, and akinesia may recur (17, 18), usually peaking at about 4 days after drug withdrawal, with gradual fading thereafter. Simpson and associates (17) have suggested that the temporary exacerbation of parkinsonism is the result of unopposed dopamine blocking effects of the more slowly excreted phenothiazines in the absence of protection by the more rapidly excreted antiparkinsonian drug.

Withdrawal Dyskinesia

A syndrome resembling tardive dyskinesia may appear for the first time following antipsychotic drug discontinuation but, in contrast to tardive dyskinesia and like other withdrawal symptoms, is self-limited. Case reports have described dyskinesias occurring after withdrawal of chlorpromazine and fluphenazine (26), mesoridazine (27), and haloperidol (28, 29). In the above cases, dyskinetic symptoms uniformly appeared within a few days after drug withdrawal and lasted for 1–22 weeks. Degkwitz and associates (11) observed 9 cases of withdrawal dyskinesia among 53 patients withdrawn from antipsychotics. In the 6 male patients dyskinesia disappeared in 4–7 weeks; in the 3 female patients dyskinesia occurred later and lasted somewhat longer (8–13 weeks).

Withdrawal-emergent dyskinesia has also been reported in children (30). Fourteen of 34 schizophrenic children aged 6–12 developed choreiform dyskinesia and ataxia, primarily affecting the extremities, trunk, and head, when withdrawn from antipsychotics. In half of the children the dyskinesias disappeared spontaneously within 5 weeks; in the other half the dyskinesias were suppressed by the reintroduction of antipsychotic drugs.

Similar to prevailing concepts of the pathophysiology of tardive dyskinesia, withdrawal dyskinesia is thought to be due to a temporary hyperdopaminergic state in the basal ganglia following the discontinuation of dopamine-blocking antipsychotic drugs. Experimentally induced supersensitivity to dopamine in rats dissipates within a few weeks (31) and, therefore, may be regarded as a useful animal model for withdrawal dyskinesia.

Covert Dyskinesia

According to a review by Crane (1), 5% to 40% of neurologically asymptomatic patients develop dyskinesia upon discontinuation of antipsychotic drugs. Because of the relatively short period of observation of these patients, it is unclear whether such dyskinesias are more commonly transient or persistent. The term "covert dyskinesia" refers to a dyskinesia that is not detectable during drug administration but develops in response to dose reduction or drug discontinuation, does not disappear spontaneously, and may in fact become permanent.

Degkwitz and associates (11) found that 6 of 53 patients withdrawn from antipsychotics developed dyskinesia that persisted over the 14-week study period. In another study by the same group (12), 30 of 130 patients developed dyskinesia after drug withdrawal although, since the length of postwithdrawal observation was not specified, some of these patients may have manifested transient withdrawal dyskinesias. One continuing study has found that 20% (9 of 43) of a group of young patients (mean age=25 years) developed distinct orofacial and other types of dyskinesias on antipsychotic drug withdrawal.[1] Extremity movements tended to dominate the clinical picture, as they do the withdrawal-emergent dyskinesia of children.

Hoff and Hoffman (32) found a 10% incidence of withdrawal dyskinesia in 190 patients withdrawn from antipsychotics. In the studies by Small and Hoff and Hoffman, patients were returned to drugs because of increased symptoms; thus it was not possible to differentiate withdrawal from covert dyskinesias. Hershon and associates (24) withdrew trifluoperazine in 23 patients. Five new cases of dyskinesia, characterized most frequently by motor restlessness of the trunk and extremities, emerged and persisted for 16 weeks. Crane (33) also described a case in which massive dyskinesia with diffuse restlessness dominated the picture 3 weeks after antipsychotic drug withdrawal. Other reports (29, 34) provide additional documentation of cases of covert dyskinesia in which dyskinesia appearing after drug withdrawal tended to persist for many months.

RELAPSE INTO PSYCHOSIS

Withdrawal of antipsychotic medication is frequently followed by the reappearance of schizophrenic symptoms. The time at which relapse occurs is highly variable (2). Donlon and Blacker (35) described four distinct stages during decompensation: denial and anx-

[1]Small JG: Personal communication, March 24, 1978.

TABLE 1
Effects of Antipsychotic Drug Withdrawal

Type of Syndrome	Onset	Symptoms	Contributing Factors	Course	Treat...
"Medical" withdrawal symptoms	Within 2 weeks, usually during first week.	Nausea, vomiting, insomnia, restlessness, sweating, diarrhea.	Higher incidence with anticholinergic phenothiazines (chlorpromazine, thioridazine).	Symptoms disappear in 1–3 weeks.	Sedatives ... cholinerg... ally requ...
Extrapyramidal symptoms					
Parkinsonism	Within 2 weeks.	Rigidity, tremor, akinesia.	Only occurs when antiparkinsonian drugs are also withdrawn.	Usually disappears within weeks.	Retreatme... parkinso...
Withdrawal dyskinesia	Within 6 weeks. Often during first week.	Motor restlessness (akathisia), choreoathetoid movements of face, mouth, trunk, and extremities.	May develop later and last longer in females.	Self-limited. Complete remission is the rule.	No treat... Severe cas... quire sev... weeks.
Covert dyskinesia	Within 6 weeks.	Those of tardive dyskinesia (choreoathetosis, tics, grimaces).	Those of tardive dyskinesia (antipsychotic drug treatment, age, etc.).	Most cases show gradual improvement.	No treatm... cases. Ch... drugs an... antagonist... vere pro...
Psychotic decompensation	Usually within 12 months.	Increase in schizophrenic symptoms.	Sudden withdrawal, high dosage.	Tends to persist and intensify.	Retreatme... psychot... therapies

iety, depression and intensification of defense mechanisms, internal chaos, and subjective relief. Some of the early symptoms of relapse such as anxiety, tension, and insomnia overlap withdrawal symptoms, and in some patients differentiating these two syndromes may not be easy. The withdrawal syndromes described above are summarized in table 1.

TARDIVE, WITHDRAWAL, AND COVERT DYSKINESIA

It may be argued that no convincing evidence indicates that covert dyskinesia is distinct from tardive dyskinesia. The introduction of "covert dyskinesia" as a diagnostic entity might, therefore, confuse rather than clarify clinical thinking. There are other problems with the concept of "covert dyskinesia." By definition, the diagnosis can only be made after lengthy drug withdrawal during which withdrawal-emergent dyskinesia fails to disappear. Obviously a lengthy period of drug withdrawal is an impractical diagnostic procedure because of the risk of psychotic relapse (36). A further objection is that many patients with "covert dyskinesia" might in fact show subtle early signs of dyskinesia if they were thoroughly examined while still on drugs, and therefore should be considered as examples of undiagnosed tardive dyskinesia.

Our view regarding the interrelationships of the three clinical types of dyskinesia may explain the emphasis accorded to covert dyskinesia. We suggest that late appearing antipsychotic drug-induced dyskinesias form a continuum from mild, completely reversible cases ("withdrawal dyskinesias") to ob... sistent, and sometimes irreversible tar... nesias. We also assume that with advanc... continued drug treatment patients may pr... this continuum, although specific examples ... this assumption have not yet been reporte... ous that some patients may fall between t... tremes. Whether such patients are given ... "atypical dyskinesia" (37), withdrawal dy... covert dyskinesia may be academic.

Recognition of covert dyskinesia is difficult ... it must be distinguished from withdrawal ... According to our definition, if a withdraw... dyskinesia shows no sign of amelioratio... weeks, one is dealing with covert dyskine... dyskinesia differs clinically from tardive ... that during maintenance antipsychotic the... dyskinesia is entirely hidden, whereas tar... nesia is manifested by observable abnor... ments. If drug therapy is continued, cove... may break through and become overt. A... distinction would appear to be straightfo... fact often blurred in practice. Many pa... subtle early signs of tardive dyskinesia ... degrees of restlessness, choreoathetoid m... the tongue or fingers, and increased blink... easily be missed unless specifically looked ... cases dyskinesia may become quite evide... age is reduced or the drug is discontinued.

Clinical awareness of covert dyskinesia ... able in several ways. First, and foremos... may become increasingly concerned about ... bility that patients maintained on moder... ...

doses of antipsychotics may be developing the neuropathological substrate for tardive dyskinesia, although overt movements are suppressed by the drug. We hope that such concern will lead to attempts to reduce dosage whenever feasible (2). The awareness of covert dyskinesia may also induce clinicians to be vigilant for very early signs of overt dyskinesia. Acceptance of the notion that a proportion of drug-treated patients harbors an early hidden form of tardive dyskinesia may stimulate efforts to develop diagnostic tests to uncover such patients. One promising approach is the exploration of administration of challenge doses of central anticholinergic and dopaminergic substances to patients receiving maintenance antipsychotics. L-Dopa (38) and benztropine (39) given intravenously have been tried for this purpose. The assumption is that those patients in whom dyskinesia is unmasked by exposure to dopaminergic or anticholinergic drugs might be suspected to harbor covert dyskinesia.

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Tricyclic Antidepressants: Plasma Levels and Clinical Findings in Overdose

BY DAVID N. BAILEY, M.D., CRAIG VAN DYKE, M.D., RENE A. LANGOU, M.D., AND PETER I. JATLOW, M.D.

Following amitriptyline or imipramine overdose by 30 patients, total plasma concentrations ranged from 29 to 1732 ng/ml but did not correlate well with physical findings or most electrocardiographic changes. Only those patients with a QRS interval greater than 0.1 second had significantly elevated plasma levels. However, a plasma level ratio of the parent drug (amitriptyline, imipramine) to its respective N-desmethyl metabolite (nortriptyline, desmethylimipramine) greater than or equal to 2.0 was associated with an acute overdose. This ratio was more useful than total plasma levels in differentiating an overdose from a therapeutic dose with associated toxicity and an elevated steady-state plasma level.

ONE OF THE major risks in treating depressed patients with tricyclic antidepressants is that of administering a class of cardiotoxic and neurotoxic compounds to those patients who are most likely to attempt suicide. Electrocardiographic abnormalities, coma, convulsions, and death have all been reported following tricyclic overdose (1-7). Despite the seriousness and frequency of tricyclic overdose, little information is available to the clinician for making critical decisions concerning the patient's care. How serious is the overdose (or is it an overdose)? Does the overdose warrant hospitalization on a medical ward, and if it does, how long should the cardiac rhythm be monitored, and when can discharge safely occur? Byck (8) has suggested that all patients who have ingested an overdose of tricyclics have their cardiac rhythm monitored for 72 hours. However, a few deaths from sudden arrhythmias have been reported as long as 6 [illegible] ingestion (1, 2, 9).

Since there are few firm guidelines to [illegible] swers to the clinical questions usually [illegible] teraction between the internist and the [illegible] and their perspectives often differ. The [illegible] not want to hospitalize the patient on a [illegible] if the patient must be admitted because [illegible] diac arrhythmias, the internist is eager to [illegible] to a psychiatric ward. The psychiatrist, [illegible] hand, prefers to admit the patient to a [illegible] til it is clear that there will be no sudden [illegible] plications from the overdose.

The development of sensitive and spec[illegible] the accurate measurement of tricyclics [illegible] 13) may provide an additional parame[illegible] this problem. In two different publicatio[illegible] associates (6, 14) presented data on tric[illegible] trations following self-poisonings. How[illegible] and economical reasons have general[illegible] analysis of plasma tricyclic levels to a [illegible] laboratories. With the recent applicatio[illegible] sensitive detectors, which permit the acc[illegible] atively inexpensive measurement of pla[illegible] levels, such analyses may become more [illegible] able to clinicians.

We present the results of a retrospectiv[illegible] cases of overdose with amitriptyline (1[illegible] imipramine (11 cases), the most widely [illegible] cyclics. Plasma concentrations of the p[illegible] (amitriptyline, imipramine) and their res[illegible] macologically active *N*-desmethyl meta[illegible] triptyline, desmethylimipramine) were [illegible] by gas-liquid chromatography using a nitr[illegible] detector (11, 12). Plasma levels were co[illegible] the physical findings in each case and wit[illegible] sion ECGs in 21 cases.

METHOD

The clinical history for all 30 cases was [illegible] rospectively by review of the medical an[illegible] records. Patients who had ingested signifi[illegible] of drugs other than tricyclics were exclu[illegible] study. Initial plasma samples were obta[illegible]

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puncture within 2–4 hours following the patient's admission to the medical ward, and later samples were sometimes available for analysis.[1] Twenty-one patients had ECGs performed within 4–6 hours of admission, and 14 of these also had at least one subsequent ECG. The ECGs were reviewed by a cardiologist (R.A.L.) without knowledge of either the clinical history or the tricyclic plasma levels. The electrocardiographic criteria of the Minnesota Code (15) were used in the evaluation of the ECGs: class IV, for the ST segment; class V, for T waves; class VI, for A-V conduction; class VII, for ventricular conduction; and class VIII, for arrhythmias. We considered the QRS duration to be prolonged if it exceeded 0.1 second and the QT interval to be prolonged if it exceeded that predicted for the particular heart rate and sex of the patient by the Ashman, Hull, and Lepeschkin formula (16). Plasma levels of tricyclics were compared with both the physical findings and the ECG data. Levels of significance were determined using Student's t test and the Pearson product-moment correlation method. We considered $p<.05$ as significant.

RESULTS

Nineteen of the 30 cases involved amitriptyline, and 11 involved imipramine. The ingestions frequently included other drugs as well; only 7 patients had taken amitriptyline alone and 6 imipramine alone. However, there was not a significant difference between the mean plasma level of patients taking a pure overdose and those taking a mixed overdose. All but 7 patients had psychiatric histories, and 18 had been receiving tricyclics on a long-term basis. The patients ranged from 13 to 73 years of age; 6 were teenagers, and 24 were women. Although total plasma tricyclic levels did not correlate with age or differ with sex, the levels did correlate weakly with the estimated dose ($r=.40$, $p<.05$).

On admission, the patients' plasma levels of amitriptyline ranged from 29 to 1080 ng/ml, nortriptyline from 0 to 337 ng/ml, and the total level (the sum of the parent drug and the active metabolite) from 29 to 1260 ng/ml. Similarly, plasma imipramine concentrations ranged from 92 to 1515 ng/ml, desmethylimipramine from 31 to 350 ng/ml, and the total level from 123 to 1732 ng/ml. The ratio of the concentration of parent drug to that of the active *N*-desmethyl metabolite (P/M) ranged up to 14.4 (mean±SE=5.4±1.0) for amitriptyline and to 12.9 (3.4±1.1) for imipramine. For the entire series, 19 patients had P/M ratios greater than or equal to 2.0, and an additional 2 patients had no detectable metabolite at the time of drug analysis. Nine of the patients with a P/M ratio greater than or equal to 2.0 had plasma concentrations approaching those reported after therapeutic doses. In 8 patients with more than one determination, the P/M ratio decreased with time.[2]

The most common physical findings were tachycardia (pulse rate >110/minute), 70%; coma (grade 2 or 3[3]), 53%; and pupillary dilatation (estimated subjectively on admission but not quantified), 40%. Hyperreflexia and hypotension, although noted in some patients, were less frequent. Pupillary dilatation occurred more often with amitriptyline overdose (53%) than with imipramine (18%). Hypotension was more frequent with imipramine overdose (63%) than with amitriptyline (16%). We compared the mean total tricyclic levels for all patients with a given physical finding with the mean levels of those patients without the finding. The differences in means for all findings were not significant.

All patients had depressed levels of consciousness when first seen; 14 were grade 1, and 16 were grade 2 or 3. To exclude the possible effects of other drugs, we looked at the 13 cases of pure tricyclic overdose separately. The 6 lethargic patients (grade 1) had a mean total level of 364 ng/ml (range=187–555 ng/ml), and the remaining 7 (grade 2 or 3) had a mean total level of 809 ng/ml (range=189–1732 ng/ml). Because of the overlap in plasma concentrations, patients in coma (grade 2 or 3) showed only a trend toward having higher plasma concentrations than those patients not in coma ($t=1.96$, $p<.10$). However, 5 of the 6 cases in the study with plasma levels greater than 900 ng/ml were in grade 2 coma or worse regardless of whether the ingestions were pure or mixed.

Of the 21 patients who had ECGs on admission, 19 demonstrated prolonged QT interval, 7 had a widened QRS complex, and 7 manifested abnormal ST segments and flat T waves. Of these patients, 14 had at least one subsequent ECG. For 6 patients (including 5 with QRS widening) the ECGs reverted to normal, and the remaining 8 patients still demonstrated a prolonged QT interval at the time of their last ECG (i.e., 24–48 hours after admission). Prolonged PR intervals and right bundle-branch block were each seen in only 4 patients. Arrhythmias were not observed; however, our patients did not always have continuous monitoring of their ECG.

When the mean total tricyclic level for patients with a widened QRS complex (821±180 ng/ml) was compared with that for patients without a widened QRS complex (396±67 ng/ml), the difference was significant ($t=2.71$, $p<.02$). The same statistical comparison performed for the other ECG parameters showed the difference in means not to be significant. Furthermore,

[1]All samples had already been submitted to our own clinical laboratory for routine clinical chemical and/or toxicological analyses required for patient care, and no samples were drawn solely for analysis of plasma tricyclics. The plasma remaining after such routine analyses was frozen (−15°C) until the time of analysis for tricyclics. This protocol was approved by the Yale Human Investigations Committee and the National Institute on Drug Abuse.

[2]A table of data is available from Dr. Bailey on request.

[3]Grade 1=lethargic but arousable with nonpainful stimulation; grade 2=responsive to painful stimulation only; grade 3=nonresponsive to painful stimulation.

when the total tricyclic levels were compared with the magnitude of each ECG parameter using the Pearson product-moment correlation method, no significant correlation was noted.

All patients in this series recovered completely, including 3 who suffered cardiac arrests. Treatment consisted of supportive care alone. One patient also received parenteral physostigmine.

COMMENT

Previous studies of tricyclic overdose have predominantly been single-case reports. Recently, Spiker and associates (6, 14) reported two series of tricyclic overdose in which plasma levels were compared with physical findings and ECG changes. In the first series only the duration of the QRS complex was strongly correlated with total tricyclic levels; more specifically, levels greater than or equal to 1000 ng/ml were associated with a QRS interval greater than or equal to 0.1 second. Plasma levels correlated weakly with maximum pupil size but did not correlate with maximum heart rate, lowest blood pressure, degree of unconsciousness, or changes in the PR interval or ST-T wave of the ECG. In the second series, Spiker and associates noted that mean tricyclic plasma levels were significantly higher in patients who died, had a cardiac arrest, required respiratory support, were unconscious, had a grand mal seizure, had a ventricular rate greater than or equal to 120/minute, had cardiac arrhythmias, had a QRS interval greater than or equal to 0.1 second, or had bundle-branch block.

Our study is subject to several limitations. The patient population consisted only of those who were considered to have taken an overdose serious enough to warrant hospitalization on a medical ward. We do not have information on the less seriously ill patients who were not hospitalized, nor do we have preoverdose baseline data (e.g., ECG pattern) to compare with the data following overdose. It is possible that tricyclic plasma levels correlate best with the difference between baseline parameters and those monitored after overdose. For instance, after therapeutic doses of nortriptyline, Ziegler and associates (17) demonstrated a significant relationship between plasma levels and the increase in heart rate above predrug baseline values. Our patients also varied in the elapsed time between ingestion of the overdose and arrival at the hospital where plasma samples were obtained, and some had taken small amounts of other drugs as well. Nevertheless, these are exactly the conditions that the clinician faces when having to make judgments about the care of such patients in the emergency room. Patients arrive in the emergency room at varying times after the overdose. The history is often vague as to the precise amount of tricyclic ingested or the types and amounts of other drugs taken, and rarely are there baseline data to compare with the patient's ECG and physical findings after the overdose.

A review of the literature clearly indic[illegible] dividual variations in plasma concentrat[illegible] therapeutic doses. In healthy individual[illegible] tion is largely under genetic control (18, [illegible] plasma tricyclic concentrations are not u[illegible] to adjust dosage, and it is not uncommo[illegible] receiving chronic therapeutic doses to [illegible] total plasma tricyclic concentrations in [illegible] as observed after overdose. Half of ou[illegible] total tricyclic levels that approached thos[illegible] ter therapeutic doses (11, 12, 20, 21), and [illegible] had higher levels. Therefore, using [illegible] plasma levels alone to distinguish a pa[illegible] acutely ingested an overdose from one [illegible] therapeutic dose is difficult because of [illegible] overlap in plasma concentrations in thes[illegible] tions.

As a means of differentiating these c[illegible] noted that 21 of our 30 patients had P M[illegible] than or equal to 2.0 (mean± SE=4.6 ±0[illegible] contrast to a P/M ratio less than 2.0 o[illegible] tients stabilized on therapeutic doses (1[illegible] In a report by Gram and associates (2[illegible] P/M ratio was 0.82 (range=0.09 to 2.5) [illegible] maintained on therapeutic doses of imi[illegible] larly, a study of Braithwaite and asso[illegible] vealed an average P/M ratio of 1.17 (ran[illegible] in 15 patients treated for 6 weeks with [illegible] amitriptyline. Although they did not co[illegible] our calculation of this ratio from the dat[illegible] associates (6) demonstrated a P/M rat[illegible] equal to 2.0 in 11 of 12 patients with pu[illegible] or imipramine overdose (range=0.41 t[illegible] much higher ratios following overdose p[illegible] insufficient time for mono-*N*-demethylat[illegible] ent drug.

A parent drug-to-metabolite ratio gr[illegible] equal to 2.0 is more meaningful than the [illegible] cyclic level for the diagnosis of an acute [illegible] presence may be of particular value in [illegible] overdose when the tricyclic level is in t[illegible] served after therapeutic doses and the[illegible] or physical findings characteristic of to[illegible] trast, a P/M ratio less than 2.0 is more c[illegible] high steady-state plasma levels follow[illegible] dosages in spite of ECG or other clinic[illegible] toxicity. Being able to distinguish these t[illegible] is important for the acute management [illegible] well as having implications for their lo[illegible] ment. Patients who have acutely overd[illegible] quire either medical or psychiatric hospi[illegible] in the future they may need particularly c[illegible] tion in times of stress. On the other hand[illegible] levels or toxicity following a therapeut[illegible] require the patient's hospitalization on a [illegible] and a reduction in dosage, but psychi[illegible] ization is probably not a consideration [illegible] P/M ratio greater than or equal to 2.0 stro[illegible] a tricyclic overdose, its absence does n[illegible] exclude it.

The limited relationship between total plasma levels and either physical findings or most ECG changes was disappointing. This was consistent with the earlier report by Spiker and associates (6) but in contrast to their later report (14). When we analyzed the 13 cases of pure overdose, the patients in coma did not have a significantly higher plasma level. Nevertheless, 5 of 6 patients with plasma levels greater than 900 ng/ml were in coma. One possible explanation for this limited relationship is that an acute overdose of drug does not represent a steady-state condition. Tissue tricyclic levels may be transiently higher than plasma levels (22).

The single most common finding in our series was a prolonged QT interval; however, it did not correlate with plasma concentrations of tricyclic, nor did it always return to normal within 24 to 48 hours following admission. Widening of the QRS complex occurred in 7 patients and was associated with significantly elevated total plasma concentrations. This is consistent with the observations of Spiker and associates (6) but, unlike these authors, we found that 6 of our 7 patients with QRS widening had levels less than 1000 ng/ml. Of clinical importance, the QRS interval had returned to normal within 24 to 48 hours in all 5 patients for whom we had follow-up ECGs. Bigger and associates (23) have proposed tricyclics as possible antiarrhythmic agents because of their "quinidine-like effect" in widening the QT interval and thereby increasing the refractory period of the ventricle. In therapeutic doses quinidine prolongs the QT interval; in toxic doses it increases the duration of the QRS complex (24). We feel that, following a tricyclic overdose, widening of the QRS complex (i.e., QRS greater than 0.1 second) may be viewed as an index of serious cardiotoxicity that resolves with falling plasma levels, whereas a prolonged QT interval represents a more sensitive indicator of tricyclic effect that takes longer to return to normal limits.

Unlike many other drug poisonings (e.g., barbiturate, ethanol, and salicylate), where drug levels represent a cornerstone of clinical management, tricyclic overdoses cannot be judged as to their seriousness simply by evaluating the plasma levels. We found that a parent drug-to-metabolite ratio greater than or equal to 2.0 is associated with an acute overdose and may be useful in differentiating an overdose from a therapeutic dose with toxicity and a high steady-state plasma level. Monitoring the ECG appears to be a more cost-effective method and possibly more relevant parameter for gauging the seriousness of the overdose. Prolonged QRS complex, in addition to arrhythmias, is associated with significantly increased tricyclic plasma levels and represents serious cardiotoxicity.

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Paradoxical Reaction to L-Dopa in Schizophrenic Patients

BY MURRAY ALPERT, PH.D., ARNOLD J. FRIEDHOFF, M.D., LUIS R. MARCOS, M.D., AND FLORENCE DIAMOND

The authors administered 6 g of L-dopa to 8 schizophrenic patients and 750 mg of chlorpromazine to 7 schizophrenic patients. Chlorpromazine showed only a modest advantage over L-dopa and only on some Brief Psychiatric Rating Scale factor scores, and at maximum dosage the thought disturbance factor score in the L-dopa-treated group was not worse than at baseline. The results suggest that L-dopa is associated more with toxic than with schizophreniform symptoms and that there is adaptation to its effects. The authors discuss implications of these findings for the dopamine hypothesis of schizophrenia.

ALTHOUGH DIRECT SUPPORT for the dopamine hypothesis of schizophrenia has remained elusive (1, 2), there is a substantial body of evidence consistent with the major indirect prediction of the hypothesis: Agents that reduce central dopamine action tend to be therapeutic.

The converse prediction, the expectation that schizophrenics will be differentially sensitive to agents that increase central dopamine, also has considerable support. However, there have been a number of studies in which L-dopa has been administered to schizophrenic patients with results that have not been entirely consistent with the dopamine hypothesis. Unfortunately, these reports are difficult to interpret because of methodological differences among the studies.

LITERATURE REVIEW

For example, in an early study, Yaryura-Tobias and associates (3) reported on 4 schizophrenic patients who showed extrapyramidal side effects while on neuroleptic medication despite the use of anticholinergics. Their interest was in evaluating the efficacy of L-dopa on the extrapyramidal side effects, and, continuing the neuroleptic treatment, they discontinued anticholinergic medication [illegible] L-dopa treatment. They reported mark[illegible] tion of mental status with no improve[illegible] worsening of extrapyramidal side effects

Two characteristics of this study sho[illegible] mind while one is considering these [illegible] tient population was selected for the pre[illegible] pyramidal symptoms and treatment [illegible] cholinergics had been stopped just before [illegible] regard to the first issue, Crowley and ass[illegible] ported that patients who developed e[illegible] side effects while taking neuroleptics [illegible] amounts of free dopamine in their urine [illegible] tiation of the antischizophrenic medic[illegible] tients who did not develop extrapyramid[illegible] and Chase and coworkers (5) found that [illegible] nal fluid concentrations of homovanillic [illegible] droxy-indolacetic acid were not as eleva[illegible] thiazines in a group of schizophrenics w[illegible] drug-induced extrapyramidal side effects [illegible] tion of a group of schizophrenics with e[illegible] symptoms not responsive to anticholin[illegible] have produced a biologically unique study [illegible]

In regard to the second issue men[illegible] abrupt termination of anticholinergics j[illegible] study also may have influenced the e[illegible] noted that abrupt termination of anticho[illegible] have negative effects on the clinical pic[illegible] Fleming and associates (7) reported di[illegible] the relationship between the abrupt te[illegible] anticholinergics and L-dopa. They studi[illegible] receiving fluphenazine decanoate plus [illegible] hydrochloride. Maintaining this schedul[illegible] 3–5 g/day of L-dopa, they found no imp[illegible] extrapyramidal side effects but, rather, s[illegible] of improved mental state. However, whe[illegible] idrine was replaced by a placebo, the[illegible] oration in both mental state and ex[illegible] symptoms. Thus, the effects Yaryura[illegible] associates (3) attributed to L-dopa may [illegible] more complex mechanisms.

There have been a number of recent re[illegible] tive therapeutic effects of L-dopa on sch[illegible] tients. These reports have been based on [illegible] (8–11) and controlled (7, 12, 13) studies, [illegible] 316 chronically hospitalized schizoph[illegible] and have found positive therapeutic effec[illegible] in the areas of communication and rapp[illegible] dopa was added to the treatment regime[illegible] it appears that L-dopa—as little as 600 mg [illegible]

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up to 5 g/day (7) and in study periods as brief as 2 weeks (9) or as long as 18 months (11)—may actually be therapeutic, at least in relation to deficit schizophrenic symptoms. There is also a report (14) suggesting that schizophrenic children with autistic features may be helped by L-dopa.

Angrist and associates (15) studied the behavioral effects of L-dopa on 10 relatively acute schizophrenic volunteers with mixed diagnoses who were taking only chloral hydrate or sodium amytal. They interpreted their results as being consistent with the prediction, based on the hyperdopamine hypothesis of schizophrenia, that L-dopa should cause behavioral worsening. However, despite their use of higher doses (up to 6 g/day), they felt the intensity of the effects noted was somewhat less than that reported by Yaryura-Tobias and associates (3), who used doses of up to 2.6 g/day. Their report contained a brief description of the course of the response to L-dopa for each of the subjects, and it is interesting to note that worsening of symptoms was not always monotonic. For patient 3, depression and suicidal ideation cleared at 1,500 mg/day, whereas patient 4 showed disappearance of auditory hallucinations at 5000 mg/day.

Our interest in the action of L-dopa in schizophrenia grew out of our exploration of a novel treatment approach in tardive dyskinesia (16–18), a condition thought to result from supersensitivity of dopamine receptors consequent to prolonged neuroleptic blockade. Assuming that supersensitivity is a biphasic process, we attempted to reduce the sensitivity of dopamine receptors by increasing the amount of available transmitter. To achieve this, we treated our volunteers with L-dopa. Early in the treatment we noted some worsening of both mental condition and dyskinetic symptoms, which had been expected. After some weeks, in a number of cases maintained on a fixed dose of L-dopa we observed a salutary habituation and reduction in dyskinetic symptoms, with the maximum response occurring after the L-dopa was discontinued. In several patients the chronic schizophrenic process appeared to follow the same course as the dyskinesia.

Prior reports on the use of L-dopa have not been designed to reveal whether adaptation to the behavioral effects occurred as the L-dopa was continued over a period of time, nor have comparison groups been treated with neuroleptics in parallel and evaluated off drug at termination. Finally, when behavioral worsening with L-dopa has been reported, it has been difficult to determine whether the schizophrenic process has become more florid or whether a toxic process has been superimposed. To further evaluate these questions, we initiated the following pilot study.

METHOD

Subjects

Newly admitted schizophrenic volunteers below the age of 55 were drawn from the adult inpatient services of Bellevue Psychiatric Hospital. Diagnoses were made independently by ward clinicians and by a clinician with the research group and were based on DSM-II definitions (19). All of the patients had at least one prior admission for the same condition. Patients with complicating medical or drug- or alcohol-abuse histories were excluded. All patients signed consent forms after being informed of the study's research nature and treatment risks.

Eighteen patients were randomly assigned to either the chlorpromazine or L-dopa group. One patient in the L-dopa group was lost from study because of persisting nausea, and 2 patients were lost from the chlorpromazine group, one because an acute exacerbation of psychotic symptoms in the first week of study made it necessary to depart from the medication protocol and the second for administrative reasons unrelated to the study.

The average age of the chlorpromazine group was 34.0 years, with a range of 21–48 years. It included 4 men and 3 women. Five of the schizophrenic patients were diagnosed as chronic undifferentiated type, one as paranoid schizophrenic, and one as having an acute schizophrenic reaction. The average age of the L-dopa group was 33.9 years, with a range of 26–50 years. It included 4 men and 4 women. Five patients were diagnosed as chronic undifferentiated type and three as paranoid schizophrenics.

Additional information about the subjects is contained in figure 1, which shows that at baseline both groups obtained their highest Brief Psychiatric Rating Scale (BPRS) (20) factor scores in the area of "thought disturbance." However, as is typical with our recently re-exacerbated patients, a broad range of pathology is represented, and all the factors are elevated.

Dose Schedule

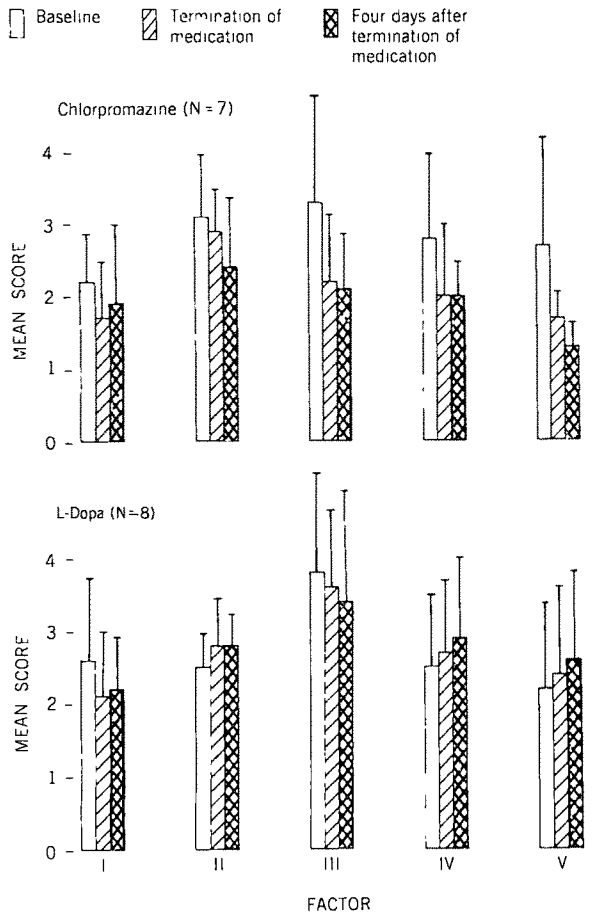
Medication was managed by a nonblind psychiatrist and administered in divided doses three times a day. The initial L-dopa dosage was 0.5 g and was increased 0.5 g every other day until day 11 (3 g); at this point it was increased 0.5 g each day until day 17 (6 g). This dose was maintained through day 24, when it was discontinued. Chlorpromazine was started at 300 mg, increased to 450 mg at day 4, to 600 mg at day 6, and to 750 mg at day 8. At day 11 the dose was adjusted at "doctor's choice." Chloral hydrate was permitted throughout. No other medication was permitted for the 5 days before baseline or for the 4 days after the last day of medication.

The Brief Psychiatric Rating Scale was completed by two independent raters at baseline, on the last day of medication (day 24), and after 4 days without study medication (day 28).

RESULTS

Figure 1 presents the average BPRS factor scores for each measurement period and the standard devia-

FIGURE 1
Brief Psychiatric Rating Scale Factor Scores of Schizophrenic Patients Treated with Chlorpromazine and L-Dopa



tions. Two-way analyses of variance were done for each factor; the differences for factor 1 (anxiety-depression) were not reliable for either drug group or treatment period.

For factor 2 (anergia), the main effects were not reliable, but the interaction was ($F(2, 26)=3.58$, $p<.05$). This interaction may be due in part to the chlorpromazine group's score being somewhat higher initially, but it also was influenced by the tendency of this group to continue to improve after medication was cut.

For factor 3 (thought disturbance), there was a reliable group effect ($F(1, 13)=4.24$, $p<.05$) and period effect ($F(2,26)=3.59$, $p<.05$). The chlorpromazine group appeared to progress more quickly, but the L-dopa group also showed monotonic improvement.

None of the differences observed on factor 4 (activation) achieved statistical reliability, perhaps because of some tendency toward increased variance on these scales.

For factor 5 (hostile-suspicious), the main effects were not reliable, but the interaction was ($F(2, 26)=3.48$, $p<.05$). This finding was consistent with the clinical observation of increased anger in [illegible] L-dopa. However, our measurement [illegible] been past the peak of this effect, which [illegible] the failure to find a main effect for this [illegible]

DISCUSSION

The Brief Psychiatric Rating Scale [illegible] dopa-treated patients were only mode[illegible] worse than those of a comparison grou[illegible] treated with effective doses of chlorpr[illegible] there is little in the results to suggest that [illegible] treated group fared less well than a pla[illegible] group would have. On the other hand, [illegible] infrequency of statistically reliable group [illegible] it should be recalled that the groups we[illegible] that a lack of difference cannot be consid[illegible] similarity. (Because the use of L-dopa for [illegible] ic patients is controversial we felt that [illegible] with a small number of patients was neces[illegible] currently conducting a larger study.)

Another possible contributor to the pa[illegible] ferences between the groups on the [illegible] scores may relate to the point at which m[illegible] taken. Earlier in the course of treatment it [illegible] ier to identify the activated, labile, soci[illegible] patient as being on L-dopa. However, [illegible] nation of medication and 4 days afterwa[illegible] ferences were not discriminable and may [illegible] adaptation to the treatment.

It would appear that L-dopa does not [illegible] acerbate schizophrenic symptoms, since [illegible] schizophrenic BPRS factor, which incl[illegible] disorder and hallucinations, actually s[illegible] lowering in the scores of the patients who [illegible] 6 g of L-dopa from their baseline rat[illegible] schizophrenics may be vulnerable to the [illegible] dopa, these effects appear to manifest the[illegible] more diffuse toxic psychosis that is supe[illegible] the schizophrenic illness. We plan additio[illegible] study this issue.

It is not simple to place these findings in [illegible] of the dopamine hypothesis. Following [illegible] sults in a number of attempts to demonstr[illegible] turnover of central dopamine in schizoph[illegible] attention has shifted from presynaptic re[illegible] synaptic mechanisms, possibly including [illegible] in receptor sensitivity (21). An exacerbat[illegible] toms with L-dopa, followed by adaptatio[illegible] observed, is not inconsistent with such [illegible] are planning a study that will include a [illegible] ment period and a data-analytic design, wh[illegible] mit within-patient comparisons of the clin[illegible] with tremographic measures.

The worsening in the clinical condition [illegible] schizophrenic patients after several wee[illegible] doses of L-dopa was less than might be pred[illegible] the dopamine hypothesis. In addition, the [illegible] areas in which worsening occurred are diffi[illegible]

clearly confined to schizophrenic symptoms. Also, we noted some indications of adaptation to the effects of L-dopa; adaptation is not thought to be characteristic of the pathogenic process underlying schizophrenia. In fact, adaptation to L-dopa might have treatment potential considering the positive reports from a number of studies (7–14) in which L-dopa was given in conjunction with neuroleptics. Further study is indicated.

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The Social Context of Schizophrenia

BY J.K. WING, M.D., PH.D.

During the past 25 years psychiatry has increased its understanding of the social context of schizophrenia in four major areas. Reasonable reliability can now be achieved in describing and recognizing many of the acute and chronic syndromes, so that comparability can be achieved between different research teams. Much is now known about the proximate social causes of symptoms and disabilities. The relationship between social and pharmacological treatments is now better understood. A more rational approach to the planning and prescription of services and to the counseling of patients and relatives can be made. Each of these lines of advance promises to lead to further progress in the future.

THE PUBLICATION, 25 years ago, of the discovery of a really effective pharmacological method of suppressing the major symptoms of schizophrenia must rank among the great medical advances of this century. Not only did it prove of immense value in reducing the distress and disability suffered by patients but it led to biochemical studies that have already proved fruitful and promise, eventually, to deepen and broaden our understanding of the nature of the condition. No discussion of recent advances in our knowledge of the social causes of schizophrenia or of the social aspects of treatment is complete unless it gives due acknowledgment to the fact that the course and outcome of the disease are markedly altered by drug treatment.

Of course, it should be recalled that the prognosis for schizophrenia was not totally bleak in earlier times. Mayer-Gross (1) described a 16-year follow-up study of patients with schizophrenia admitted to the Heidelberg Clinic in 1912 and 1913. Just over one-third of these patients had made what Mayer-Gross called social recoveries. For 5 years Harris and his colleagues (2) followed patients admitted to the Maudsley Hospital in London for insulin coma treatment between 1945 and 1948. They found that 45% of these patients were socially independent and had few or no symptoms of schizophrenia at 5-year follow-up. In 1961 Brown and his colleagues (3) examined a group of patients first admitted for schizophrenia to 3 English [illegible] tals 5 years earlier, in 1956. The proport[illegible] dependent lives without marked sympto[illegible] phrenia was 56%. The figures cited from [illegible] are not strictly comparable because of [illegible] mortality and the numbers of patients lo[illegible] cause standard methods of defining and [illegible] groups of patients were not used. How[illegible] suggest a considerable improvement in c[illegible] of which seems to have taken place bef[illegible] pharmacological treatment and part sinc[illegible] possible that a further improvement [illegible] since 1961.

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THE IMPORTANCE OF DIAGNOSIS

Any historical perspective raises the p[illegible] comparability of clinical diagnosis. Thi[illegible] area in which we can claim considera[illegible] progress. Advances have been made in ac[illegible] munication between psychiatrists worki[illegible] times and in different parts of the worl[illegible] United States-United Kingdom Diagnos[illegible] and the World Health Organization Inte[illegible] Study of Schizophrenia (7) have demons[illegible] most characteristic central syndromes of [illegible] phrenia are universally recognized by ps[illegible] though there is less clinical agreemen[illegible] much more should be included under the [illegible] label. The adoption of standard techni[illegible] search purposes does not, of course, elim[illegible] tual differences, but it makes them muc[illegible] vestigate and provides a way of achievin[illegible] stricted comparability. In particular, [illegible] recognize syndromes reliably allows the [illegible] a wide range of biochemical, genetic, p[illegible] cal, and epidemiological work, and this [illegible] most promising ways to expand future [illegible]

Already, of course, we can be sure tha[illegible] of schizophrenia is not homogeneous. I[illegible] rank symptoms described by Schneider [illegible] temporal lobe epilepsy, amphetamine ps[illegible] coholic hallucinosis. Paranoid syndro[illegible] first-rank symptoms are very diverse, r[illegible] subcultural state characterized by exciter[illegible] secutory delusions colored by local be[illegible] tions such as morbid jealousy or "pari[illegible] lans." A wide variety of somatic and [illegible] factors may be involved. Conditions that [illegible] terized by catatonic symptoms but not by [illegible]

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hallucinations (rather rare in the acute form nowadays) may be due to encephalitis or other brain disease as well as to schizophrenia. Accurate description of the acute symptoms and signs is still extremely important and will pay dividends for the future.

Progress has also been made in defining and reliably measuring some of the more chronic symptoms that occur in schizophrenia. One set of such symptoms—the "chronic poverty syndrome"—comprises flatness of affect, poverty of speech, slowness, underactivity, social withdrawal, lack of motivation, and impaired ability to communicate using nonverbal language. These symptoms have a great effect on work output at simple industrial tasks and on social performance more generally (8). Another chronic syndrome includes incoherence of speech and unpredictability of associations; the individual does not seem to be able to think to a purpose but goes off on sidetracks, giving the impression of confusion and incompetence.

The relationship between the acute and the chronic syndromes has long been a matter of controversy. Some theorists have thought that specific kinds of delusional experience were primary, but others, like Eugen Bleuler, regarded flatness of affect and loosening of associations as "fundamental." A multigene theory, together with one taking into account the action of a variety of environmental precipitants (somatic, psychological, and social) could comprehend both points of view and link the dimensional with the categorical approach as well. The traits underlying both acute and chronic syndromes may be widely distributed. The main point about a diagnosis, however, is to suggest to the clinician theories that might be helpful when applied in practice, i.e., theories that will predict a useful form of treatment, prevention, or management or, at the very least, suggest a prognosis. None of the concepts of schizophrenia in vogue at present is firmly grounded in a set of linked and tested theories in the same way as diabetes mellitus, for example, although there are many analogies between the two concepts and we may hope that one day our knowledge of schizophrenia will equal our knowledge of diabetes. It is therefore necessary to proceed to some extent empirically.

We know that the acute syndromes usually respond to phenothiazine treatment, whereas the chronic syndromes respond less well. We know that the acute syndromes, particularly when precipitated by organic factors such as amphetamine or alcohol, are less likely to be preceded or followed by the chronic syndromes. We know that social withdrawal often accompanies chronic depression, mental retardation, chronic physical disability, Asperger's syndrome, and severe social deprivation. Moreover, mild degrees of affective blunting or of "thought disorder" cannot be recognized reliably. These are good empirical reasons why any theory linking acute and chronic schizophrenic syndromes should not be applied in practice, except with great circumspection. In particular, the supposed presence of chronic syndromes should not lead to a regime of treatment suitable only for the acute syndromes if the latter are absent, and especially if they have never been present. Terms such as "simple," "pseudoneurotic," "pseudopsychopathic," "latent," or "sluggish" schizophrenia should not be made to carry too much therapeutic meaning. Still less should the spectrum concept be used clinically. In my view, failure to understand this basic distinction has contributed to practices that can indeed be criticized (9).

SOCIAL CAUSES OF SCHIZOPHRENIA

The second area in which considerable progress has been made is that of the social causes of the characteristic acute and chronic syndromes. The simplest way to study these is to look for immediate effects of varying social conditions. It has been well documented, for example, that some patients living in a sheltered environment relapse very quickly when they are exposed to a program of rehabilitation that asks too much of them (10–13). In other words, social pressure, even when well intentioned, can be harmful. Brown and Birley (14) demonstrated an increase in the frequency of certain life events for schizophrenic patients compared with control groups during the few weeks immediately before the first onset or an acute relapse. Other studies (15, 16) have shown that patients who return home after discharge from the hospital to live with a relative who is hostile, critical, or emotionally overinvolved tend to have a very high relapse rate compared with those whose relatives are more accepting and emotionally neutral.

These three very different types of social precipitant have in common the fact that they occur in close contiguity to the onset or acute relapse of schizophrenia. There has long been a theory that the relatives of people with schizophrenia are in some way implicated in the original causation of the disease (17). Very few empirical studies have been carried out to test this hypothesis, and, in any case, the methodological problems are immense. Studies of identical twins brought up together or apart provide little convincing evidence (18). The best work methodologically is that by Wynne and his colleagues (19, 20) suggesting that communication deviances are common in the parents of patients with schizophrenia, although this has no necessary bearing on origins in childhood. However, this clear-cut result has not been replicated in a recent very careful study in England (21), and it may be that the reason, at least in part, is that the criteria for diagnosis in the two series were quite different. As for the really passionate advocates of various forms of the early environment model of causation, it is difficult to understand where the strong motivation to believe such theories springs from in the absence of any substantial supporting body of evidence.

Reverting to the three very different types of factors that seem to be clearly and immediately related to relapse or first onset, what they appear to have in com-

mon is a degree of social overstimulation. Other social causes of schizophrenic symptoms seem to work in a very different way. The clinical poverty syndrome, for example, tends to become much more pronounced under conditions of social understimulation. The classical example is the back ward of the old-fashioned mental hospital, where severely disabled schizophrenic patients with little capacity for spontaneous communication were left to their own devices and spent a very large proportion of their waking day doing absolutely nothing. They became very much more withdrawn and underactive and apathetic. Within limits, this effect can be reversed. An increase in the richness of the social environment leads to a decrease in the poverty syndrome (8, 13, 22). This result is not true only of the situation in the back wards. It has been found that schizophrenic patients are vulnerable to such effects wherever they are found—in hostels, hotels, group homes, day centers, sheltered communities, centers for the destitute, in lodgings, or in a patient's own home. In one 5-year follow-up study (3) it was found that, among patients who were unemployed and living at home, the length of time spent doing absolutely nothing was of much the same order as among long-stay patients in a hospital with a poor social environment.

If these studies are accepted as providing a basis for rational social treatment, there are two major problems: 1) the reduction of an excessive level of social withdrawal and other associated behaviors by enriching the social environment without provoking an acute relapse, and 2) the reduction of an excessive level of social stimulation without encouraging undue dependence.

To summarize the processes involved: many patients who experience an attack of acute schizophrenia remain vulnerable to social stresses of two rather different kinds. On the one hand, too much social stimulation, experienced by the patient as social intrusiveness, may lead to an acute relapse. On the other hand, too little stimulation will exacerbate any tendency already present toward social withdrawal, slowness, underactivity, and an apparent lack of motivation. Thus the patient has to walk a tightrope between two different types of danger, and it is easy to become decompensated either way. The difficulty in thinking and the inability to communicate verbally and nonverbally will be most evident in interactions with close kin, for example, and will be exacerbated in conditions of anxiety and high arousal.

Families might provide protective or provocative settings; there is no general rule. The same is true of nonfamily environments. However, the first onset usually occurs within a family setting, and there is a natural tendency on the part of relatives toward normalization, which can lead to further intrusion, particularly if the patient withdraws in natural reaction. The patient may try to explain the abnormal experiences in terms of hypnotism, thought transfer, witchcraft, or other subculturally acceptable ideas. The more intrusive the environment, the less the pati[illegible] withdraw and the more the symptoms ar[illegible] and provoked. Being admitted to a hospi[illegible] ing off alone may provide relief simply by [illegible] degree of social stimulation.

After several attacks, the patient may [illegible] with his or her experiences and find (whe[illegible] consciously formulates this or not) th[illegible] withdrawal is protective. Relatives, too, [illegible] themselves just what degree of stimul[illegible] missible, so that a working solution is a[illegible] tients usually do not wish to be entirely a[illegible] do like to remain in control of the inter[illegible] The role of the relative, however, is a v[illegible] because it is unnatural to acquire the det[illegible] ment and neutrality required; it is much [illegible] fessional people to adopt such a stance [illegible] of handicapped people may easily b[illegible] protective and overinvolved. In the ca[illegible] phrenia, this overinvolvement has spec[illegible] increasing the liability to further break[illegible] this shows itself in such symptoms as w[illegible] iness at night, refusal to eat, or delusion[illegible] relative, a circular effect is set up. The rel[illegible] such reactions on a future occasion.

The alternative temptation for the pat[illegible] draw altogether into contemplation of p[illegible] ences. When a patient is allowed to do so[illegible] stimulating conditions of some large hos[illegible] badly run hostels, in reception centers, [illegible] attic at home, the negative impairments [illegible] and more obvious.

INTERACTION BETWEEN DRUG AND SOCIAL TREATMENTS

Our improved understanding of the in[illegible] tween pharmacological and social treat[illegible] third major area I should like to discuss [illegible] long-stay schizophrenic patients there hav[illegible] indications that drug treatments and soci[illegible] are not alternatives but must be used to[illegible] each other. The better the environmenta[illegible] the less the need for medication; the poor[illegible] milieu, the greater the need (or at leas[illegible] drugs (8, 13, 16, 22). A similar generaliz[illegible] made, although the details are different, [illegible] methods of handling acute breakdown.

If phenothiazine drugs work, at least i[illegible] ducing a high level of physiological a[illegible] they should be most effective in condit[illegible] overstimulation; there is some evidence [illegible] (15, 16). It has been suggested (16, 25) [illegible] who live in "low-emotion" families can [illegible] (except for the operation of accidental si[illegible] without taking preventive medication. L[illegible] colleagues (26), for example, have recent[illegible] schizophrenic patients sitting quietly by t[illegible] their own homes have a high arousal level[illegible]

by skin conductance. If a "high-emotion" relative enters the room there is no change in arousal level. If a "low-emotion" relative enters, the high arousal habituates toward normal. This work needs to be repeated, but, if it holds true, it suggests that some relatives have a calming and supporting effect, similar to the protective effect of a good hospital, hostel, or therapist. On the whole, the need for medication is minimized when the patient is living in optimal social surroundings. The optimal environment is structured, with expectations geared precisely to the level of performance that the patient actually can achieve, or perhaps a little above, but with supervision that is not emotionally involved. Complex decision making is particularly difficult for patients and tends to rouse their anxiety or their withdrawal. Control of the level of social stimulation must be left to some extent in the patient's hands. A relationship of trust and confidence, whether with relatives or with professional staff, is most likely to develop when these conditions are optimal.

This kind of explanation may also be applied to observations to the effect that schizophrenic patients in some developing countries appear to have a better prognosis than elsewhere (27, 28). Factors such as the degree of social overstimulation (and the amount of face-to-face contact between close relatives that fosters such stimulation) may vary considerably in different cultural settings, as may the impact of more accidental social pressures. If so, it can be hypothesized that the lower the sum total of the environmental pressures, the less will be the need for pharmacological forms of treatment.

A great deal remains to be explained about the acute and chronic syndromes. The mechanisms whereby first-rank symptoms, such as thought echo or "thoughts spoken aloud," which are direct descriptions of basic experiences with very little elaboration, can be explained in terms of overarousal, phenothiazine action, or cerebral pathology remain obscure. Recent advances in pharmacology and biochemistry are so promising that it is clear that links will have to be made. Meanwhile, the formulation put forward here has obvious relevance to problems of management.

THE MANAGEMENT OF SCHIZOPHRENIA

The fourth area in which advances have been made and opportunities opened for further progress is that of "management," including the planning and prescription of services. So far, we have considered the reactivity of the acute and chronic syndromes and have not dealt with their effects on the individual's level of social functioning. Two more general factors need to be considered: extrinsic disadvantages and adverse secondary reactions.

Chronic impairments contain a component that is "intrinsic" and a component that is reactive. From a purely practical point of view, an intrinsic impairment is one that will not go away, no matter what biological, psychological, or social factors are brought to bear therapeutically. Of course, we can never be certain, in theory, that we have tested the limits of treatment, and circumstances sometimes change in a way that surprises even the most astute clinician, so that it is never wise to give up hope of improvement. However, to deny that severe and chronic intrinsic impairment can exist must do more harm than good. It is only by recognizing and studying disabilities that it is possible to find ways of compensating for them.

"Extrinsic" factors are those which would have been disadvantageous whether or not the individual had become ill. Someone born in a large family in a poor area with inadequate housing and high unemployment is socially disadvantaged. If this person has few assets in the way of vocational skills, intelligence, social attractiveness, family support, and so on, he or she is not well prepared to cope with chronic illness of any description, let alone schizophrenia. However, the socially disadvantaged are more at risk for developing all sorts of diseases, schizophrenia being one of them. There has been a long discussion of whether socially disadvantageous conditions are likely to cause schizophrenia, or whether the high rates observed under such conditions are due to a preexisting drift. Intrinsic impairments often precede the first acute attack. In such cases the prognosis is less likely to be favorable. The balance of the epidemiological evidence at the moment is in favor of the view that much of the association noted between the onset of acute schizophrenia and factors such as social class, marital status, migration, and social isolation is due to selection rather than to stress (29). On the other hand, it is also clear that a disadvantaged background has an adverse effect on the course of the illness because of the lack of social support. This is why an attempt to increase the number of assets—by providing social skills and vocational training, by creating a graded series of supportive social environments, by counseling in methods of self-help, and so on—is of such importance.

Adverse secondary reactions occur when the handicapped or disadvantaged individual's self-attitudes, confidence, and expectations suffer to an extent that is out of proportion to the cause. Such factors are particularly important in psychiatric disorders, and they depend a great deal on the reactions of important people in the affected person's social environment. Relatives, employers, workmates, friends, and professional people reflect to the patient an indication of personal worth and status. Severely handicapped people have to opt out of certain responsibilities, and others have to take them on. If the others are professionally trained for the job there can develop what Goffman (30) called a staff-client split, each side taking up stereotyped attitudes toward the other. The longer the client experiences dependence, the more he or she may come to prefer it. Staff, in turn, may exaggerate the severity of the intrinsic component in impairment, and thus a vicious circle is set up, with unnecessary

dependence developing as well as that which is unavoidable.

The acceptance by handicapped persons of limitations that are not actually necessary is the essence of secondary reaction. The most obvious example is institutionalism, at the heart of which is a gradually acquired contentment with life in the institution that culminates in the individual's no longer wishing to live anywhere else. Institutionalism is thus caused partly by a reflection back to the handicapped person of his or her own altered status as a human being. He or she is seen as a patient, rather than as an employee, a father, a mother, a customer, or a companion. The patient role is a constricted one, replacing many others that the patient might have been able to undertake. Partly, however, institutionalism results from forces in the patient—his or her own previous experience of illness, self-confidence, potential for developing alternative skills, and determination to achieve independence (8). Now that few people stay in the hospital for more than a few weeks, other types of adverse secondary reaction are more common—for example, attitudes of despair or apathy arising from experience of unemployment, solitary living, social rejection, poverty, and even destitution.

Perhaps the most striking change that has occurred in psychiatric practice during the past 25 years, as a result of the new methods of social and pharmacological treatment, is the decrease in the numbers of patients living in large mental hospitals. When it first became clear that this trend was firmly established, some psychiatrists began to think that most of the symptoms and the disability found in long-stay patients were actually caused by being in the institution. Thus, preventing people from entering the hospital at all would prevent much of this morbidity altogether. At the same time, sociologists were showing that, in some areas of the United States, people were being given a diagnosis of schizophrenia and admitted to a hospital even though their problems appeared to be social rather than medical and the functions of the hospital seemed custodial rather than therapeutic (31). The results were overgeneralized, but there followed a revulsion against the use of institutions.

In some parts of the United States it has become as hard to get into a hospital as, in former days, it had been hard to get out of one. An ideal of "community care" was set up instead, which in its most extreme form excluded the use of hospitals altogether. This period has been followed, in its turn, by a period of reappraisal. It is now clear that some patients continue to be severely disabled and vulnerable to relapse, even though they have spent only a brief time in the hospital or even no time at all. A small but important "new long-stay" group is accumulating (32). It is also clear that nonhospital services can suffer from deficiencies quite as great as those of hospitals (33). A good hospital is better than a poor hostel, nursing home, or hotel and is just as much a part of community care. Even the concept of community itself can be challenged, when the patient has no social roots, is markedly [illegible] and is unable to make spontaneous soc[illegible]

Three principles of service organizat[illegible] been accepted as being basic to a goo[illegible] service: that it should be responsible for [illegible] geographical district, comprehensive [illegible] with a very wide range of needs, and [illegible] tegrated to ensure adequate commun[illegible] agencies and not block or delay a patien[illegible] one part of the service to another. The a[illegible] organized in this way is to prevent, d[illegible] tain disease and disability by providing f[illegible] agnosis, therapy, rehabilitation, and s[illegible] calling the attention of other respons[illegible] effective means of prevention (34).

These considerations are particularly [illegible] schizophrenia. Earlier, I drew the anal[illegible] tes mellitus, and the concept of the "[illegible] a chronic physical disease is very appro[illegible] to be considered from three points of [illegible] patient; that of the patient's relatives an[illegible] immediate social environment on whe[illegible] well-being and livelihood may depend [illegible] professional people, administrators, [illegible] who provide, plan, and control acces[illegible] Each group sees a different set of prob[illegible] fails to appreciate the difficulties face[illegible]

The principles of management in [illegible] based on recently acquired knowledg[illegible] elaborated elsewhere (35). They requ[illegible] tion of skills that some psychiatrists, [illegible] tional therapists, and social workers co[illegible] sess. Above all, they require a willi[illegible] from patients and relatives, who are the [illegible] the essential information on which skil[illegible] must be based. Part of the peculiar diffe[illegible] ing schizophrenia is that it lies somew[illegible] conditions like blindness, which, alth[illegible] handicapping, do not interfere with an i[illegible] pacity to make independent judgments ab[illegible] own future, and conditions like severe m[illegible] tion, in which it is clear that the indiv[illegible] be able to make such independent judgm[illegible] frequently a fluctuating degree of insig[illegible] ty. In some cases the fluctuations appe[illegible] obvious pattern. They are then quite [illegible] and environmental circumstances seem[illegible] fact that the impairments are invisible [illegible] lems even more difficult, since neighb[illegible] and even, unfortunately, some profess[illegible] deny their existence altogether.

Nevertheless, a good deal is now k[illegible] social and pharmacological reactivity of [illegible] chronic syndromes and the way adverse [illegible] actions develop. This means that it is p[illegible] ence the course of schizophrenia for t[illegible] deed, in a great many areas, some par[illegible] already operates effectively to minimize [illegible] maximize assets, or relieve burden. If it [illegible] to put together a service that was effect[illegible]

parts, instead of only in some of them, a great deal of illness, handicap, and suffering would be prevented. Such a service would have to include a range of sheltered day and residential environments. The elements of a responsible, comprehensive, and integrated service are well-known on both sides of the Atlantic (34, 36).

One further point deserves emphasis because it has been insufficiently appreciated hitherto. This is that the relatives of schizophrenic patients are nowadays the real primary care agents. They and the patients deserve better counseling (37). The most important and most difficult requirement, for patient, relative, and therapist alike, is to be realistic. If the therapist expects too much (endeavoring, perhaps, to fit the patient into the straitjacket of some untested theory), he or she can very easily make the patient worse (10–13). It may seem reasonable to submit the patient to an intensive regimen of social skills training, rehabilitation, or social activation on the theory that invisible impairments need not be considered, but, although it will sometimes work, it often will not.

It should always be remembered that there is a suicide rate in schizophrenia. If the patient has severe intrinsic impairments they will usually be obvious, and it may be worthwhile, for example, to accept work in a sheltered setting where only part of the social role need be adopted. A neutral (not overemotional) expectation to perform up to *attainable* standards is the ideal. This rule, if difficult for the specialist to adopt, is a thousand times more difficult for relatives. Nevertheless, we should be humbled to recognize that a large proportion of relatives, by trial and error, do come to adopt it without any help from professionals. The patient will have the greatest chance of acquiring insight if those around him or her are realistic in spite of everything. This is why skilled counseling (which means much more willingness by professionals to learn from relatives and patients) is the essence of good management.

Our attitudes to cancer have changed fundamentally during the past decade. Opinion leaders are willing to say that they have been treated for it and that they can live with it. This is not yet true for schizophrenia. Doctors themselves are still frightened of the word. Because the most effective methods of management and the services to back them up are not yet universally available, known, or applied, it is rare for the condition to be frankly and realistically discussed. Public opinion remains ill-informed and prejudiced. But just as the results of treatment have dramatically improved during the past quarter of a century, so the simple application of our present knowledge could produce a further substantial step forward. We would find that public opinion moved forward with us.

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Foundations' Fund Prize for Research in Psychiatry

The American Psychiatric Association is inviting submissions for the second annual Foundations' Fund Prize for Research in Psychiatry.

Candidates for the prize should be citizens of the United States or Canada and should be nominated by a sponsor. Sponsors should be members of the American Psychiatric Association. Members of the prize board are excluded from submitting nominations.

The *sponsor* should submit a supporting letter justifying the nomination in detail and summarizing the nominee's research accomplishments.

The *nominee* should submit the following:

1) An up-to-date curriculum vitae,
2) An up-to-date bibliography,
3) A group of representative and thematically linked papers or books published (or accepted for publication) in English and dated within 10 years of the deadline for submission, and
4) A summary statement written by the nominee that emphasizes the principal theme or themes running through the work and its internal cohesiveness and consistency.

Six copies of each entry should be submitted to Joel J. Elkes, M.D., Chairperson, Foundations' Fund Prize Board for Research in Psychiatry, American Psychiatric Association, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but cannot be returned. The award will be presented at the Convocation of Fellows at the APA annual meeting in May 1979.

The deadline for submission is December 1, 1978. Any entry received after that date will automatically be entered for consideration for the 1980 award unless withdrawn by the nominator or candidate.

Automated Treatment for Flight Phobia: A 3½-Year Follow-up

BY MYRON S. DENHOLTZ, M.D., LAWRENCE A. HALL, PH.D., AND EDWARD MANN, PH.D.

The authors contacted 43 patients who had been given audiovisual treatment for flight phobia 3½–5½ years previously. Of the 26 subjects who had flown successfully by the end of treatment, 23 had maintained their ability to fly at follow-up. Eighteen said their attitude toward flying was more positive since treatment, and 21 rated their comfort level while flying as "much better" or "better"; however, only 3 said they were "cured." Eleven patients said the treatment had helped to alleviate other fears or anxieties.

ALTHOUGH BEHAVIOR THERAPISTS have been exemplary in empirically validating their clinical techniques, the long-term follow-up of behavioral treatment programs applied to clinical populations is basically nonexistent. Notable exceptions include work with speech anxiety (1, 2), stuttering (3), obesity (4, 5), and cigarette smoking (6).

The purpose of this study was to make personal contact with subjects who had been treated for flight phobia 3½–5½ years previously to check the stability of the assumed behavior changes and to look for any significant change in those who had failed to improve. In addition, we tested certain assumptions about posttreatment attitude, MMPI findings, and behavior.

ORIGINAL STUDIES

Using the subjectively reported anxiety responses and pleasure responses of 102 subjects, Denholtz and Mann (7) developed a 16-mm sound film and accompanying instructional tape. All of the subjects were shown 160 20-second scenes edited from TV commercials. Eighty scenes were designed to elicit anxiety in subjects with flight phobia, and 80 scenes were expected to elicit pleasurable responses. The 16,320 differential responses on a scale of 0 to 10 allowed hierarchical arrangement of all scenes by computer. We then arranged 27 paired scenes in a progression from low to high anxiety evocation. The pairs consisted of a 20-second anxiety scene followed immediately by a 20-second pleasurable scene and were separated by an identifying printed scene label.

Denholtz and Mann (8) then used this film as treatment. The "therapists" were housewives and college students who had been taught to use the projector, signal devices, and the tape player, which played the prerecorded instruction tape. The instruction tape first induced relaxation and then instructed subjects to press appropriate buttons, which registered the presence or absence of anxiety after the subjects viewed each scene pair. The tape was also used to induce relaxation between scenes. When a scene pair evoked anxiety, it was repeated until there was no reported anxiety, and then the next pair was shown. This procedure was continued until the film was completed, usually in 2–10 half-hour sessions.

There were 3 control groups. The first group, while relaxed, was shown a placebo film that was not related to flying. The second group, while relaxed, was shown the entire treatment film straight through in 6 separate sessions. The third group received treatment without relaxation. Subjects in the 3 control groups who failed to improve in 6 half-hour sessions were transferred to the treatment group.

The significance of this study was that it combined desensitization, modeling, and positive reinforcement in a totally automated audiovisual program and was administered by nonprofessionals who had no contact with the authors. All subjects were given pre- and posttreatment MMPIs. Any subject in whom psychoticism was suggested on the initial MMPI was eliminated from the study. Of the 51 subjects who previously had refused to fly and completed the program, 40 (78.4%) reported being able to fly after treatment. Significant positive changes were also noted on the Taylor Manifest Anxiety Scale of the MMPI for all subjects who overcame their fear of flying. In addition, the successful subjects demonstrated significant positive shifts on 14 of the 24 MMPI scales, whereas there was only 1 positive scale shift in the unsuccessful group. These results suggest that major personality changes can generalize from successful treatment in limited problem areas.[1] Although there was an overall success rate of 78.4% for the 51 subjects completing the study,

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

Dr. Denholtz is Clinical Associate Professor, Dr. Hall is Clinical Associate Professor, and Dr. Mann is Associate Professor, Department of Psychiatry and Mental Health Science, New Jersey Medical School, Newark, N.J. Dr. Denholtz is also Director, Department of Psychiatry, Elizabeth General Hospital, 925 E. Jersey St., Elizabeth, N.J. 07201.

[1]Mann ET, Denholtz MS: Behavior change and generalization in the treatment of phobias (unpublished paper).

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there were 30 dropouts from the control groups. The subjects in the control groups were not offered crossover to treatment until they had completed 6 sessions of control group "treatment." Many subjects who did not feel a reduction in anxiety or felt frustrated while watching placebo films dropped out before they could be transferred to actual treatment.

FOLLOW-UP PROCEDURE

We attempted to reach all of the 51 original participants for a semistructured telephone interview; we contacted 43. We asked them how many flights they had taken since treatment and the purpose and social circumstances of those flights and about their emotional comfort while flying, their use of security items, changes in their attitudes toward flying, other generalization effects, and their own assessments of success or relapse. We also recorded their spontaneous comments. The interviews lasted 5–20 minutes and were conducted by a clinician who had considerable experience with phobic patients and who was not part of the original project. All of the subjects contacted were very cooperative and appeared to respond honestly to the open-ended questions.

For this follow-up study, all of the previously treated subjects who were reported in the original papers were classified as behaviorally successful, i.e., had actually flown soon after the termination of treatment; attitudinally successful, i.e., were enthusiastic and had given every indication that they would fly but for financial reasons had not yet been able to purchase a ticket; and unsuccessful subjects.

RESULTS

Maintenance of improvement was determined by the subjects' responses to the questions "How soon after the fear of flying program did you fly again?", "Have you been unable to take any flights since the end of the treatment?", and follow-up questions about each flight taken since the end of the program. Flights were defined as round-trip flights including stopovers.

Twenty-three[2] of the 26 subjects (88.5%) in the behaviorally successful group were currently able to fly. This group had taken numerous flights (median=5, range=1–80). The 3 subjects from the behaviorally successful group who had stopped flying included 2 women who were in individual psychotherapy before entering the program and at the time of follow-up and a third woman who had taken 6 flights with comfort but stopped flying 2 years later after her husband was involved in an airplane crash.

In striking contrast to the behaviorally successful group, both the attitudinally successful and the unsuccessful groups showed little behavioral ga[illegible] measure of success used was subjects' ab[illegible] round-trip flight and their willingness to f[illegible] that flight. Six of the 7 attitudinally succes[illegible] had not flown, and 8 of the 10 unsucces[illegible] contacted had not flown. One of the 2 [illegible] group who were able to fly had subse[illegible] treated individually by a skilled behavio[illegible] ter his failure in the experimental progra[illegible] man's sister died suddenly, which necess[illegible] to a distant state. He described the fligh[illegible] despite preflight tranquilizers and much [illegible] lowing that flight, he would not retur[illegible] stated that he could not fly again. The d[illegible] havior outcomes of the behaviorally suc[illegible] compared to the attitudinally successful [illegible] cessful groups combined was statistical[illegible] (χ^2=21.79, p<.001).

Attitudinal data were elicited by the que[illegible] there been any changes in your attitude [illegible] since your participation in the fear of fly in[illegible] Of the behaviorally successful participa[illegible] tacted who were still flying, 81.8% (18 of 2[illegible] positive change in their attitude. As n[illegible] pected, the 3 relapses in the behaviora[illegible] group reported no change. Four of the [illegible] successful subjects, including the one w[illegible] indicated a positive change in their attitude[illegible] ing. Eight of the 10 unsuccessful subjects [illegible] change in attitude, 1 reported less fear, a[illegible] a more fearful attitude toward flying. Th[illegible] attitudinal changes for the behaviorally su[illegible] jects and the attitudinally successful and [illegible] subjects combined proved to be significa[illegible] p<.01).

Of the 22 behaviorally successful subje[illegible] flying, 15 rated their comfort level wh[illegible] "much better"; 6 as "better"; and 1 [illegible] "worse" although he continued to fly. [illegible] questioning, 3 of the behaviorally succes[illegible] reported themselves as "cured," i.e., they [illegible] of it anymore. The single attitudinally su[illegible] unsuccessful subjects currently flying [illegible] selves as "much better" and "cured," re[illegible]

For data relating to the generalizatio[illegible] treatment, all of the successful subjects [illegible] "Do you attribute any other changes in y[illegible] and behaviors to the fear of flying treatme[illegible] tion, subjects volunteered information th[illegible] terpreted as generalization. Many subje[illegible] regular use of antitension strategies, such [illegible] laxation techniques learned during treatm[illegible] viate other fears or anxieties during the d[illegible] ported a generally more relaxed feeling. [illegible] haviorally successful subjects currently al[illegible] described improvement in other areas, e[illegible] ing of claustrophobic feelings and improve[illegible] cross bridges. The 3 behaviorally succes[illegible] who had relapsed evidenced no generaliz[illegible] 7 attitudinally successful subjects, the o[illegible]

[2]One subject who was away on a flying trip was not contacted personally. Her neighbor reported that she regularly flies without fear.

flying and 2 of the nonflyers showed evidence of generalization. In the unsuccessful group, the currently flying subject who was treated subsequently and 2 of the failures had had some generalization.

To assess the use of security items, an indirect measure of relative insecurity and doubt about the permanence of the improvement, currently flying subjects were asked, "Do you use any security items such as tranquilizers or alcohol before flying?" Further queries determined if they purposefully carried items such as chewing gum, cough drops, mints, a small can of apple juice, etc. Of the 22 behaviorally successful participants who were flying, 10 (45.5%) continued to use security items. The attitudinally successful subjects currently flying relied on security items. The unsuccessful subject who had subsequent treatment and who now flies uses none.

SUBSEQUENT MODIFIED TREATMENT

There appeared to be a more rapid response to treatment and a greater success rate among subjects who had previously viewed the treatment film in its entirety a number of times, i.e., controls who crossed over for treatment after viewing the treatment film under other than optimum treatment conditions (8), and participants in the original evaluation of scenes in 1973 (7) who had returned for treatment. A modified treatment using the film hierarchy and treatment tape was developed and administered by one of us (M.S.D.) in a clinical setting to unselected patients who requested treatment of flight phobia. The criteria for inclusion in this group were the absence of elevated profiles on the psychotic tetrad scales of the MMPI and an unwillingness to fly even if accompanied by a family member. Each patient was taught relaxation by tape-recorded instructions that he or she used at home for a minimum of 8 sessions. Each patient then viewed the 25-minute film continuously on 4 occasions. After this preparation, the treatment was administered in the same manner as previously described. This modified treatment yielded a success rate of 83% (23 of 28 phobic individuals). All 23 successful patients flew after treatment, although most did so accompanied by a family member. Early follow-up data (3–18 months) on some patients suggest that they have maintained their behavioral gains.

DISCUSSION

We believe that this series of studies has demonstrated that audiovisual treatment programs designed to be administered by paraprofessionals were effective in the treatment of flight phobia. Not only was the initial outcome successful but behavioral improvement was maintained.

The major finding of the follow-up study was the differential outcomes among the behaviorally successful, attitudinally successful, and unsuccessful subjects. Eighty-two percent of the behaviorally successful subjects were able to fly $3\frac{1}{2}$–$5\frac{1}{2}$ years after treatment. The 3 cases of relapse seemed to be explained by an identifiable sensitizing experience or more general psychological factors. The unsuccessful subjects are still unable to fly, which is consistent with Marks' observation (9) that phobias are quite persistent and if untreated or unsuccessfully treated are unlikely to improve. Spontaneous recovery for this disorder is uncommon.

The inability of the attitudinally successful group to fly after treatment is difficult to interpret. It is possible that these subjects so wished to please that their self-report after treatment was inaccurate. Reliance on self-report, which could not be avoided in our program, is a common problem in outcome research. However, it is more conceivable that the subjects were accurate in their appraisal, but with the passage of time and without behavioral trial the therapeutic gains dissipated. It would seem essential, then, to include behavioral trial as part of treatment in every case.

The participants' ratings of their emotional comfort levels and the use of security items by many raises questions about the therapeutic ends achieved in this program. Although the majority of current fliers reported a positive change in their attitude toward flying and felt less anxious while flying, only a few reported themselves as "cured" and nearly half reported using security items when they fly. Thus, although the treatment helped the individuals to cope substantially with their fear of flying, flying-related anxiety did not entirely disappear. This finding parallels clinical experience suggesting that a large number of phobic patients, particularly those with multiple phobias, when treated are not "cured" even though they may be more capable of coping.

The data on generalization point to the development of coping skills and are consistent with a coping interpretation of fear reduction (e.g., Goldfried [10]); that is, subjects reported gains in areas not immediately related to flying. The subjects in our study reported feeling more relaxed in general and able to reduce tension in stressful situations by using relaxation techniques they learned during treatment and other antianxiety maneuvers. What seemed to occur for many subjects was the development of a "set" of being able to deal with anxiety that they applied in a variety of stressful situations. However, the marked positive shift on 14 of 24 experimental MMPI scales suggests generalization in other areas that we have not assessed behaviorally.

Experience gained in the use of the automated treatment by nonprofessionals allowed for a modest revision in the treatment procedure, which resulted in an improvement in treatment results compared with the procedure described in this paper. This modified treatment, which produced a more rapid and predictable result, involved more relaxation training at home and pre-exposure to the audiovisual program. However, this modified treatment was administered by experienced clinicians, and that unassessed variable must be

considered when the improved success rate is evaluated.

We are preparing another film to treat acrophobia. If the results of the study described here can be replicated, it may be possible to develop a library of audiovisual treatment programs. These might cover a wide variety of anxiety-provoking situations that can be classified under the broad umbrella of phobic and avoidance behavior and could lead to greater economy of therapist time.

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The Manfred S. Guttmacher Award

The American Psychiatric Association invites applications for its annual Manfred S. Guttmacher Award for 1979. The award is given for any outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or other work, including audiovisual presentations (films, etc.), submitted to or presented at any professional meeting or published during the period from November 31, 1977, to December 1, 1978. A plaque and an honorarium of $500 will be presented to the award recipient at the Convocation of Fellows at the 1979 APA annual meeting in Chicago, Ill. Please note that the award recipient is now required to give a lecture at the annual meeting.

Anyone who wishes may apply to receive the award by submitting five copies of the work as well as five abstracts to Carl P. Malmquist, M.D., Chairman, Guttmacher Award Board, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but will not be returned.

The deadline for submissions is December 1, 1978, and any entry to be considered must be in Washington by that date.

Am J Psychiatry 135:11, November 1978

Coping Styles of 34 Adolescents with Cerebral Palsy

BY KLAUS K. MINDE, M.D.

The author presents a follow-up study of 34 children with cerebral palsy. Eight years previously 23 of these children were attending a special school for handicapped children, and 11 were attending regular schools. The author evaluated the children and their families through formal and informal interviews and by administering parent and teacher rating scales of the children's behavior. He found that the development of these children between the ages of 10 and 14 was highlighted by their increased awareness and their parents' awareness of the permanence of their handicap and their consequent search for personal and, ultimately, occupational identity and their parents' emotional withdrawal.

THE INCIDENCE AND TYPES of psychological disorders associated with cerebral palsy have long been of interest (1–3). Although older studies (4–7) investigated specific difficulties encountered by handicapped individuals and their families in the course of their development, more recent work has pointed out the great complexity of social, organic, and psychological factors that influence the day-to-day functioning of the handicapped individual. For example, Richardson (8, 9) and Kleck and associates (10) measured the attitudes of different population groups toward various handicaps, and Rutter and associates (11, 12) and Shaffer (13) assessed the psychiatric morbidity of handicapped children with and without CNS disease. The latter group (11–13) consistently found physically handicapped children without brain damage to have a somewhat higher incidence of psychiatric disturbance. Cerebral palsy children with well-documented cerebral damage, however, had an even higher rate of psychiatric morbidity than healthy children.

These studies are important and may help us identify psychiatrically high-risk groups among the handicapped. They are of little use, however, for understanding the individual child and his or her family. Furthermore, most give a cross-sectional account of groups of children of various ages, ignoring the developmental aspects inherent in interpersonal relationships. For example, family conflicts, which may be the primary source of psychiatric symptomatology in primary-school-aged children, might become less important during adolescence, when children can begin to find other models of identification and have developed cognitive structures that may help them counterbalance some of the intrafamilial difficulties.

The methodological problems of measuring interactions between children and their caretakers or peers over time are obviously formidable and may account for the paucity of studies examining such processes. Kogan and associates (14) reported on 10 children with cerebral palsy under the age of 5 whom they had observed in interaction with their mothers for several years. The mothers showed a decreasing amount of warmth toward their children during the second and third year, especially if the child had not yet learned to walk. This indicated that the expectation of normalcy and the recognition that the child might not be normal was an important shaper of caretaker/child interaction. One other study (15), which did not measure actual parent/child interactions, reported on 210 children aged 4½–9 with congenital rubella 4 years after they had been initially investigated on various physiological, cognitive, and temperamental parameters. All of the children in this study had hearing defects, and 30% were also described as "neurologically damaged." The author reported that general parental concern had increased significantly between the time of initial contact and the time of follow-up (75% versus 90% of parents had an average of 2 versus 2.5 complaints) and that 79% of the children with neurological defects showed 3 or more severe behaviorally abnormal symptoms.

In summary, it appears that children with cerebral palsy and especially those with associated brain damage show a generally high incidence of behavioral difficulties. It is not clear, however, whether particular symptoms are related to specific ages of the children and to what extent general family functioning is influenced by the specific developmental tasks these children have to master. In addition, little is known about the long-term psychological development of children with cerebral palsy. In this paper I will attempt to answer some of the following questions: 1) How do children with cerebral palsy cope with the various hurdles inherent in the developmental process? 2) How do their families adjust to different developmental stages as they are modified by cerebral palsy? 3) Are children

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in special schools better off than those in regular classrooms? 4) Does contact with nonhandicapped children at specific times enhance or protect against psychiatric disorders? 5) How do the children come to terms with their handicap?

METHOD

Subjects

The present communication is a 1975 follow-up of 34 of the 41 children with cerebral palsy admitted in 1967 and 1968 to kindergarten and first grade at the school of the Mackay Center in Montreal. At that time the school provided daytime instruction only to children who had physical handicaps severe enough to prevent them from entering a normal school and intellectual ability that was at least in the educable range. Further details concerning the school and the study population have been published elsewhere (16, 17). The present sample comprises 83% of the initial group. Of the 7 children lost, 1 had died and 6 had moved away from the area. Twenty-three of the remaining 34 children were still attending the Mackay School in 1975; 11 had been discharged and were attending regular schools.

The handicaps of the children still attending the Mackay School were severe (i.e., the children needed substantial help and could not go anywhere unaccompanied) or very severe (children needed help in all daily activities). Fifteen of these children had quadriplegia with or without hydrocephalus, 5 had severe paraplegia, and 3 had other associated congenital abnormalities.

The children who attended regular schools were moderately handicapped (5 had hemiplegias, 4 had mixed cerebral disorders and congenital abnormalities, and 2 had poliomyelitis). All of these children lived at home The two subgroups did not differ on any of the other background variables shown in table 1.

TABLE 1
Background Data for 34 Children with Cerebral Palsy at Follow-Up (1975)

Item	Children in Special School (N=23)
Present age (years)	13.2
Age range (years)	12–14
Sex	
Male	12
Female	11
Socioeconomic class	
Upper	9
Middle	7
Lower	7
Present IQ	
101–115	6
81–100	12
60–80	5

Procedure

As reported previously (16, 17), from 1967–1971 I spent 3–4 hours a week in the Mackay School meeting with school personnel, the child, and/or his or her parent to discuss the emotional and cognitive development of each youngster. In addition, each parent and child had at least one formal interview every 2 years. In 1971 I left Montreal but returned in 1973 and 1975 to conduct follow-up interviews at home and in school.

In the formal interview I obtained data about the family's current social, economic, and psychological functioning, including conflicts among parents or siblings and the handicapped child. I also assessed the status and consequences of the handicap as perceived by the parents at the time of the interview. In addition, the parents were given a symptom checklist developed by Rutter and associates (18), and teachers were interviewed and asked to fill out a similar rating scale (19) for each child. Finally, the child was interviewed alone and assessed psychiatrically using the format described by Rutter and Graham (20). The c[illegible] asked about his or her handicap and futur[illegible]

RESULTS

In the course of our extended meetings, [illegible] provided considerably more data than w[illegible] during the formal interviews. Therefore [illegible] some clinical judgments to the statistic[illegible] square analyses) when this seems warrant[illegible]

All of the results were initially analyze[illegible] for sex, type of school (Mackay School [illegible] school), and three IQ levels (see table 1[illegible] were not different between these subgroup[illegible] ed for the total sample.

Question 1: How is family functioning [illegible] the developmental hurdles of a child with [illegible] sy over time? The families of these childre[illegible] rienced few psychologically dramatic occ[illegible] ing the 8-year follow-up period. Four cou[illegible] divorced; all of these had experience[illegible] throughout their marriages. None of the [illegible] institutionalized or physically harmed a[illegible] placed in foster care. However, the[illegible] subtle changes in the attitudes expresse[illegible] ents that reflected the chronic stress to w[illegible] the families were subjected.

In 1975 fewer parents (N=14) hope[illegible] provement in the physical condition of the[illegible] had 8 years previously (N=26); this differe[illegible] nificant ($p<.02$). This may simply reflect [illegible] the children had reached their optimal d[illegible] opment and their families had adjusted to [illegible] 8 of the 14 parents who still hoped fo[illegible] changes in their children's condition sin[illegible] stated that they had seen very little imp[illegible] their youngsters' physical and school [illegible] over the past 5 years. In addition, at [illegible] tellectual performance in the whole group [illegible] remained quite stable over time (see table [illegible]

TABLE 2
Intelligence Distribution of 26 Children Tested in 1968 and 34 Children Tested in 1975*

	1968		1975	
IQ	Number	Percent	Number	Percent
101–115	4	15	8	24
81–100	18	70	17	50
60–80	4	15	9	26

*Although 41 children were studied in 1968, only 26 had received reliable test scores.

findings suggest that the objective level of the children's physical and intellectual performance was not the main factor in the parents' judgment. This idea is further supported by the statistically significant shift in the outcome predicted for the individual youngster: far more parents were quite unsure of their child's future in 1975 (35%, compared with 12% in 1968; $p<.02$).

In terms of general family discord there was little gross change over time. Six families showed much, 10 showed moderate, and 16 showed little family discord, indicating that parents who managed to survive the first 5 years of living with a handicapped child appeared to weather the following 7–8 years as well. However, far fewer parents indicated that they discussed the difficulties they had with their child with each other or with anyone else than had before (50% versus 90%, $p<.01$). For example, 7 mothers mentioned the father's continued unwillingness to think about the emotional needs of their handicapped youngster and their own subsequent withdrawal from the child in an attempt to maintain their marital relationship. Twelve couples also told of having given up their attempts to receive personal guidance and support from their physicians. Their doctors had typically been very supportive during the early years of the child's life, when they talked freely about his or her physical and cognitive growth. Once changes in this area became less obvious, however, the parents reported that their doctors refused to talk about anything beyond the child's physical disability and thus seemed to have virtually nothing to say to these parents.

Fourteen of the 19 families who had other children claimed that the nonhandicapped siblings had suffered psychologically by being deprived of attention and care, especially in the early years. In 10 instances they also described a great deal of discord between their children, with the nonhandicapped older sibling forbidding the handicapped youngster to attend his or her parties or partake in other parts of their adolescent activities. These intersibling quarrels were restricted to families whose children still attended Mackay School and therefore always involved the more severely handicapped youngsters.

Question 2: What changes took place in the interactions between the parents and the handicapped child over time? As previously indicated, the parents became less hopeful about the future of their children. This was associated with a number of other developments in parental child-rearing practices. For example, children who went to regular school were "babied" less frequently than those still attending Mackay School (18% versus 35%). Parental discipline had also changed, with nearly 50% (compared with 8%) of all families in 1975 seeing no need to punish their children anymore. Although this may be an expected development reflecting the increased self-discipline of older children, parents often described it in such a way as if to say that they had given up trying to control their child's future.

Another area in which the possibly greater emotional distance between generations came up was in the scant knowledge parents generally had about their children's perception of their handicap. In 1975 60% of all the parents had no idea of what their youngster thought about his or her handicap and stated that they never mentioned it in front of the child. They also did not discuss their child's occupational or personal future with the child and assumed that he or she never thought of it as well. Even those parents who claimed that their child realized something about his or her physical and/or mental condition often did not know what the child's exact concept was. The unhappiness often found between the generations was confirmed by the substantial frequency of verbal arguments that occurred per week between the parents and their children (up from 1.6 in 1971 to 4.8 in 1973 and 3.8 in 1975). These arguments mainly centered around the child's responsibilities in the house. The arguments in 1975 were more frequent in homes in which the children still attended Mackay School (5.1 versus 3.2, $p<.01$), indicating that the less physically disabled children could satisfy parental expectations more easily.

The concerns parents had about their children also changed over time. In 1968 the most common concern (32%) was about the child's ability to get help for his or her physical disability, and only 3% of the parents showed concern for the child's learning ability. In 1971 parents were most concerned about their child's psychological functioning (21%) and their own role in assisting the child's development (15%). From 1973 onward this changed again; general retardation and academic deterioration became the main focus of parental apprehension (33% in 1973, 47% in 1975). Although a large proportion of the retarded children (78%) gave cause for concern in this area, 36% of those who scored within the normal range of intelligence also had worried parents. In addition, such practical issues as the difficulties in transporting an older and therefore much heavier child surfaced as a main parental concern in 1975 (13%), while the future capability of the child to cope emotionally ranked last (4%).

Question 3: How do the handicapped children see their development? Although each child reacted in his or her own unique style to the task of growing up with such a severe handicap, some general developmental sequences stood out. Starting in 1971, all of the nonretarded children talked openly about their handicap

TABLE 3
Symptom Checklist Ratings for Children with Cerebral Palsy, 1968–1975

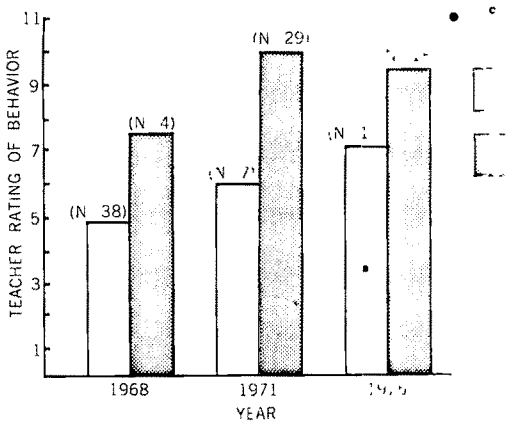
Year	Number of Children Rated	Parent Rating		Teacher Rating
		Mean Rating	Percent Deviant	Mean Rating
1968	40	—	—	5.1
1969	40	—	—	5.8
1971	37	9.8	29	9.1
1973	37	6.8	21	8.8
1975	34	9.2	32	8.3

TABLE 4
Global Mental Health Rating of Children with Cerebral Palsy, 1968 and 1975*

Year	Number of Children Rated	Rating		
		Good	Medium	Poor
1968	30	24	4	2
1975	32	12	14	6

*The difference between the ratings is significant ($p<.01$, $\chi^2=11.51$, 2 df).

FIGURE 1
Teacher Ratings of Behavior of Cerebral Palsy C
Without Nonhandicapped Friends



and seemed to conceptualize at least part of its consequences. Although some of them would initially say that they "had C.P.," all but 3 in 1975 could give a medical term for their handicap. The 9 retarded children also mentioned that they were not like other children, yet 6 of them still believed that they would grow out of their handicap within the next 5 years. The retarded children also either had highly unrealistic occupational expectations (e.g., a hydrocephalic quadriplegic child wanted to work for the local telephone company out in the country stripping overhead wires) or did not know what they wanted to do later (65%). Ninety-two percent of the intellectually normal group still attending Mackay School in 1975 talked about some occupational plans, although all of the children except 1 quickly added that they were "really not sure about it." The same apparent hesitation in regard to their future came out in the children's educational aims. Only 1 girl hoped to enter college, 4 retarded and 6 nonretarded youngsters thought they would finish grade 10, and the rest hoped for a high school diploma. None of the children had thought of a life beyond school.

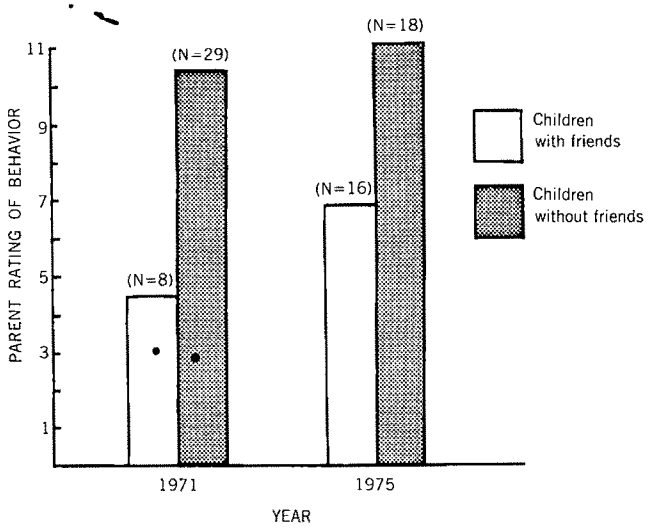
The children who went to a regular school thought similarly about their future schooling, but 70% had specific occupational plans (e.g., secretary, nursery school teacher for handicapped children, housekeeper) that they felt confident they could achieve. Six of these children who were not retarded also imagined that they would be married some day and generally thought about their life in a more long-range perspective. A further change was seen in the type of friendships these children were able to make. As discussed in a previous paper (17), only 15% of all the children had nonhandicapped friends when they were in third grade. This figure remained stable for 2 years but improved in 1975: 36% of all of the children had regular contacts with nonhandicapped youngsters in 1975. The friendships most commonly were with children 1 or 2 years older and generally conversations and some common outings.

Question 4: What is the incidence of turbance in children with cerebral palsy tects against it? The results of the psyc ments are shown in table 3. Parent ra deviance for all groups of children dec what from 1971 to 1973 and climbed a Teacher ratings were generally lower a variation between 1971 and 1975. The ings using an interview technique (20) s crease in poor mental health over time 1975 18% of all the children were assess definite psychopathology.

The patterns of disturbance the chil were not specific but covered both co rotic disorders. However, the persistenc in individual children was quite strikin youngsters who had all 5 teacher ratings and 1975, 4 had abnormally high ratings sion, 3 on 3 or more ratings, and 8 scor abnormal range less often. Parent rati equally high consistency: 5 children were abnormal on each occasion, 4 others on a 5 on 1 rating. Approximately 40% of the dren were identified by both teachers a each of the ratings.

When possible predictors of deviant be assessed, retardation correlated with symptomatology only until the child was

FIGURE 2
Parent Ratings of Behavior of Cerebral Palsy Children With and Without Nonhandicapped Friends



grade. A return to regular school did not change the rate of deviance. Severe family discord was not associated with a consistently high parent rating of the child but correlated significantly with teacher (r=.59) and psychiatric ratings (r=.68). A significant correlation was also found between having a nonhandicapped friend and low ratings on both teacher and parent measures of deviance (see figures 1 and 2). This relationship extended to the total sample and in practice meant that fewer than 10% of the children who had a nonhandicapped friend were likely to be labeled deviant at any time during the 8-year follow-up period, but just under 50% of those without nonhandicapped friends were described as showing psychiatric symptomatology.

DISCUSSION

The material presented must be seen in its proper context. The data deal only with a small group of severely handicapped children and were obtained through semistructured interviews, making statistical evaluations difficult. However, given the scarcity of any information in this area, the investigation revealed some themes that call for further attention.

It appears that the development of this group of children between ages 10 and 14 was highlighted by the following: 1) increased cognitive realization that their handicap was permanent and that there was very little hope for future improvement, and 2) the consequent tentative search for a more final personal and occupational identity, which, however, was still very precarious and in general did not extend beyond the hope to finish school. This can be seen as a manifestation of low self-esteem and the beginning of general passivity that so often characterizes older handicapped individuals. Although this behavior may be a reaction to the emotional withdrawal of many parents from their children at this time and a reflection of parental uncertainty as to their children's future, clinically there also appeared to be other sources of insecurity.

For example, many of the children had few meaningful relationships with nonhandicapped people; instead, they lived exclusively through their equally handicapped peer group. Their siblings, especially if they were older, frequently rejected them, possibly because earlier they had felt deprived because of their handicapped siblings' demands. It may also be that both the rejection by the older siblings and the parental withdrawal of emotional investment reflect attempts by these groups to retain their own stability. It is as if in the face of a doubtful future for handicapped youngsters, too much involvement of parents and siblings in them may become potentially damaging and must be avoided. The success of parental withdrawal and rejection by siblings may be evident in the low rate of family breakdown and major discord between the parents.

Although the incidence of psychiatric disturbance in the handicapped youngsters resembles that reported by others (12, 13), the majority of these children did not remain within the deviant category for many years. This supports the latest data of Thomas and Chess (21), who reported that 65% of the children who were seen as disturbed at age 5 were markedly improved in adolescence. In our group, all 7 children who were scored as having psychiatric problems on more than three ratings either came from a disturbed family or had parents who had insisted that they were "completely normal" or "forever crippled" (17). It appears as if the attempts of these parents to solve the conflicts brought about by having a child who was "deviant" (because he or she could not walk or speak) yet also "normal" (because he or she smiled or laughed like anyone else) (1) by attempting to mould the child into one extreme category have not been helpful. In fact, the data suggest that children who were able to bridge the gap between their "deviant" and "normal" parts by associating with both handicapped and physically healthy individuals had by far the best psychiatric outcome. This did not automatically happen, however, when a child left Mackay School and went to a regular school. Although all but 1 of the children attending regular schools had nonhandicapped friends, a number of them were trying very hard to compete with their nonhandicapped peers on all possible levels. This caused a great deal of tension, was often associated with psychological difficulties, and may be similar to the competition phenomena observed by Seidel and associates (22).

In conclusion, this study shows that children with cerebral palsy continue to exhibit an increased amount of psychological difficulties in early adolescence. As others have pointed out before (23), individuals such as physicians who are traditionally seen as providers of care and advice often do not live up to the ex-

pectation that they transgress their more traditional role of assessing and treating medical conditions. This leaves many children and their families trying to work things out alone—an often difficult task. The young adolescents of this study are now entering a phase that may determine their future personal and occupational identity. Many appear to slip into an increasingly passive role toward themselves and others, possibly a reaction to their parents' emotional distancing. The consistency of this development must be determined through future studies.

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TOPICAL PAPERS: Future Mental Health Care Delivery Systems

Mental Health Care Delivery Systems: Introduction

BY BENJAMIN LIPTZIN, M.D.

THE PAPERS in this section were presented at a special session of the 130th annual meeting of the American Psychiatric Association in 1977. The major purpose of the session was to bring to the attention of practitioners and policy makers the distinction between a health care financing system and a health care delivery system. The former refers to how services will be paid for, an issue that has received a great deal of publicity because of the interest in national health insurance (1–3). The latter refers to how services are organized and delivered, issues relevant to discussions of the relative merits of community mental health centers, health maintenance organizations, private practice, etc.

It is important to understand the nature of the delivery system because changes in the financing system will have intended or unintended consequences for what services are delivered to which patients in what settings. An understanding of the different components of the delivery system will put policymakers in a better position to encourage or discourage the delivery of particular services to particular populations. For example, one proposal for national health insurance would limit the number of office visits to a private practitioner but would pay for an unlimited number of visits to a community mental health center (CMHC). Although such a proposal would capitalize on the cost containment features of an organized care setting operating on a limited budget, it ignores the question of whether a community mental health center could provide high-quality intensive psychotherapy or whether the patient population treated at the CMHC would use such services.

The papers presented here were written by leading spokesmen for four major components of the system for providing services to individuals with mental disorders. These are primary health care programs (Morrill) and, within the specialty mental health sector, private psychiatric practice (Lebensohn), state mental health programs (Okin), and community mental health centers (Sharfstein). They provide a policy perspective of where we are today and where we may be in the next several years.

Policy issues that are not addressed in depth in any of the individual papers include the need to coordinate the care received in different mental health settings as well as the need to integrate mental health services with other health and human services. The patchwork of funding and organizational arrangements has made coordination and integration difficult.

From a policy analytic perspective, there is also a need for more detailed information on which patients use what services in what settings and to what effect. A first step has been taken by Regier and associates (4), who have estimated the number of individuals in treatment in different settings. Weissman and Klerman (5) have recently urged the expansion of research efforts in psychiatric epidemiology. This should include studies designed to learn what agencies or persons "untreated" individuals rely on when they experience emotional distress. The knowledge gained from health services research and psychiatric epidemiology will be essential to future planning of mental health delivery systems.

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

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The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

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The Future for Mental Health in Primary Health Care Progr

BY RICHARD G. MORRILL, M.D.

Our largely separate mental health system has developed in relationship to a health care system oriented toward specialization and solo practice. Now the health care system is moving in the direction of primary care and group and organizational practice. New forms of mental health delivery are needed to maximize the potential of these new health care programs for mental health services. The author describes these new integrated programs which bring mental health providers into the primary health care programs for direct services as well as consultation. Issues discussed include mutual roles, changes in services, the referral process, and provider relationships. The advantages of such integrated programs include decreased stigma, increased prevention through earlier detection and referral, increased family orientation, greater coordination of care, and less duplication.

PRIMARY HEALTH CARE is currently undergoing a significant revitalization in this country as a result of past overspecialization, patient dissatisfaction, rising health care costs, and new government funding priorities. At the same time there has been a continuing trend toward increasing organization in health care delivery, e.g., group practice and health maintenance organizations. The intersection of these trends is primary health care or comprehensive health care through organized ambulatory health care delivery settings. This presents important new opportunities to reverse some of the isolation and fragmentati
mental health programs have undergo
the rest of health care. This paper wi
and findings gathered from the literatu
work in integrated health-mental hea
Roxbury Comprehensive Community
and Upham's Corner Neighborhood He
Boston.

A number of potent forces leading
tegration of health and mental health s
veloped in recent years. Psychiatry
considerable role diffusion since the s
of the 60s and is generally rediscove
with medicine. Some primary care tra
are trying to give higher priority to th
side of health care. Mental health se
likely to be reimbursed by third-party
formerly, and health care programs are
about including them. The much herald
mental health centers have not fulfilled
because of lack of financing and perhaps
attempted too much too soon. Deinstit
usually not followed by the promised
community care. Mental health servi
progress, albeit with difficulty, in quali
containment, and utilization review– t
trends present in health care general
government is withdrawing support fr
of mental health care providers in favo
of primary health care providers with
skills.

The traditional areas of health-men
action have been several. Consultat
units from departments of psychiatr
within general and teaching hospitals.
often quite peripheral to their health c
which they had little influence. In the a
hospitals have run separate medical, pe
cological, and psychiatric clinics, with li

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2-6, 1977.

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cation and an exceedingly high "no show" rate on cross-referral. Attempts at postgraduate psychiatric education for general practitioners reached very few—usually only those who were already committed. There has been little interest in the general health of patients in psychiatric clinics and mental health centers. In addition, the expressed attitudes of psychiatrists and their medical colleagues toward each other have often been derogatory.

Given the above situation, it seems that there has developed not only a fragmented system but *two* mental health care systems—one through the primary health care provider and the other through the separate private psychiatrists, CMHCs, and mental hospitals.

Part of the answer lies in working out new joint health-mental health programs by taking advantage of these new forms of ambulatory health care delivery. These include group practices, health maintenance organizations, neighborhood health centers, hospital primary care clinics, and rural health centers. The purpose of this paper is to explore joint health-mental health programming in these expanding organized forms of primary health care. Areas I will discuss include roles, types of disorders, new forms of services, the referral process, and provider relationships. Primary health care is defined as general health care that 1) exists at the point of first contact by the patient, 2) is continuing to ensure the patient's overall health, and 3) coordinates the delivery of other specialized health and social services.

THE ROLE OF MENTAL HEALTH PROVIDERS IN PRIMARY CARE SETTINGS

There seems to be a broad consensus that there is much mental health care to be done in primary health care programs and that some mental health input is thus needed. Two general approaches have developed. In the first (1) the mental health provider carries out direct or indirect consultation only. In the second (2–6) he provides consultation *and* a variety of direct mental health treatment services. The principle embodied in the first approach is that one should facilitate mental health work by the primary health care provider but not risk taking it over. The second approach is based on the supposition that many patients will never leave the primary health care program to be diagnosed or treated in a separate mental health system and therefore the two should be integrated in the ambulatory care area.

There seem to be additional benefits for mental health providers carrying out the latter approach—full service in the primary health care program. Several authors (2, 3, 5, 6) have pointed out that, given the mistrust and poor communication between the mental health provider and health providers in general, carrying out direct service in the primary health care program serves to educate, sensitize, and create a feeling of "colleagueship." This often serves to sharpen criteria for referral and remove the "mystery" of just what the mental health provider does. As the communication and mutual respect improve, the primary health care provider becomes freer to ask for consultation for some of his problem patients. Thus a patient seen as a "crock" might be perceived instead as a patient who sets up recurrent hostile dependent relationships. The semantic difference is not important. The new perception of patient needs and feelings that might be amenable to change is important.

If the mental health provider plays a full role in the primary health care program, including consultation, evaluation, and treatment, is the primary health care provider encouraged to "dump" all the patients he finds difficult, "bad," uncooperative, or "crazy"? In general, we have not found this to be the case. There are too many other factors impinging on this referral process, which will be further elaborated below.

WHO TREATS WHAT? "PROBLEMS IN LIVING" VERSUS "MENTAL DISORDER"

Certainly, there is not yet a widely recognized conceptual framework in which to apportion mental health treatment tasks, although there does seem to be a broad emerging consensus in the literature. The general turfs seem to be the "vicissitudes of everyday life" for the primary health care provider, "self realization" for religion and to a growing extent the new "human potential movement," and "pathological symptom and behavior patterns" for the mental health provider. Of course there is broad overlap between these and the problem areas listed below.

The primary care literature (7, 8) suggests that "problems in living" might better be cared for by the primary health care provider. A partial list would include helping the patient with life crises (birth, adolescent identity, marriage and divorce, loss, death, etc.), alcoholism, normal development issues, encouraging strengths, dealing with normal feeling states in health and illness (anger, guilt, depression, low self-esteem), anticipatory guidance for mothers, changing roles of women, adjustment to chronic illness, and others. This list would also include working with stable or compensated chronic psychotics. (More primary health care providers might be willing to do this if mental health backup were more readily accessible.)

The mental health provider working in an integrated setting naturally begins to see the patients with whom the primary health care provider is not trained to work or who do not respond to his intervention. This includes adults and children with serious or life-threatening disorders, the clearly psychotic, those who require long-term or specialized treatments (e.g., group or behavioral), chronic patients who need a complex variety of backup supportive services, diagnostic problems, patients who require complicated drug treatment (e.g., lithium), and others.

It might be said that if primary health care providers miss too much mental illness, mental health providers find too much. Coleman and associates (9) noted, "Pediatricians start out from the assumption of health, psychiatrists from the assumption of illness, but both may err." What then, is preferable—a false negative (labeling a chronic depression in a young woman that affects her parenting as "You're tired—take a vacation") or a false positive (labeling a culturally determined time-limited hysterical grief reaction as schizophrenia, "You're sick")? Both practitioners take the perspective of their experience. One provider has to assume the best, which serves to support patient assets, and the other has to find the illness if he is not to miss that which may be treatable. The conclusion seems to be that each approach has its purpose and can inform, educate, and improve the other if they work together.

THE SHAPE OF MENTAL HEALTH IN THE PRIMARY HEALTH CARE SETTING

What forms of mental health service are appropriate to the primary health care program? A number of programs have successfully provided evaluation and therapies, including crisis, short-term, group, family, couples, behavioral, and drug treatments. Long-term psychotherapy has depended on the resources available. There are, however, some unique aspects of a primary health care program that the mental health services can use to become more effective.

The primary health care provider typically follows patients in brief episodes over long spans of time in contrast to a separate mental health clinic, where patients are usually seen for prolonged episodes over a relatively brief span of time. Thus the separate mental health program may try to turn out a finished product because most patients are not expected to return. However, the primary health care program assumes patients *will* continue to return for health care for themselves or their families. This makes possible new treatment formats. Long-term observation and less intensive therapeutic input for a chronic problem may be more acceptable to many patients, more effective, and more efficient.

Alternatively, one might employ a "string of beads" approach. The patient may work on the same theme or conflict over a succession of interrupted crisis episodes for which he returns to the same provider to take up where he left off—a type of interrupted continuity more possible in a primary health care setting. This allows us to reach patients who cannot easily accept the traditional time scales of psychotherapy.

Other unique aspects of this setting include a wider variety of patients and problems, which tests the adaptability and flexibility of mental health staff; the diversity of "helping figures" (doctors, nurses, dietitians, and others), which allows us to structure a wider variety of therapeutic plans to reach more patients; and the ready availability of physical ... cal diagnosis and treatment, which imp... with the psychosomatic disorders.

THE REFERRAL PROCESS—A COMPL... TRANSACTION

Referrals of ambivalent patients ... health care program to a separate me... gram have always been marked by ... pliance. Important factors in understa... ess of referral include the primary ca... ception of the need for referral, fear of ... or having to share a professional rel... ing that all mental health services are ... in some way.

Education of primary health care ... course one way to reduce these resis... response, which seems to be more eff... has been put into full operation, is to br... health care and mental health provi... physical proximity in the same prog... study[1] showed that referral to a ment... sional within the primary health ca... duced 23% first appointment failures. w... to a separate outside mental health c... 75% first appointment failures.

In the view of the primary health car... mental health resource is thus changed ... clinic or hospital to "Dr. Jones" or "M... in the office down the hall—a profes... shared work with your difficult patien... titudes and effectiveness you have co... for. This relation increases the numb... ness, and timeliness of referrals by ... and respect to develop between the staf... the informal communication, which ... break down some of the stereotypes ab... ness and treatment (11), and 3) creating ... envelope or a formal or informal ... team, which can reduce the issue of ... "giving up" the patient.

IDENTITY, STATUS, AND THE PROV... RELATIONSHIP

Historically, the relationship betwee... and their medical colleagues has been p... not based on mutual respect. These is... be dealt with in any joint endeavor.

Potent factors in this relationship ar... status. In joining a health care speci... selected to share certain traits and is ... ized into shared assumptions about pr...

[1]Data from an unpublished paper by J.K. Carpe... entitled "The Psychiatric Social Worker as a ... Group Medical Setting" (1974).

care and the limitations on one's role to act on these. The concept of the reference group is useful in clarifying apparently contradictory changes in attitude and behavior. A physician may in the course of a day play out different roles by virtue of belonging to several different reference groups. The "we" might be "pediatricians" at 9 a.m. in the pediatrics clinic, "doctors" at 1 p.m. when negotiating with the director of nursing, or "team member" at 4 p.m. when reviewing the day's patient load with the team. His receptiveness to mental health roles and issues may vary accordingly.

In the past few years there has been tremendous interest in a new breed of doctor—the primary care physician (a specially trained internist, pediatrician, or family practitioner). Training programs are in the process of shaping new generalist identities and show a tendency to want their own mental health teachers—at times shutting out an associated department of psychiatry. The basic issues, often unspoken, are as follows: Does the presence of a psychiatrist in a primary care program undermine the generalist "yes, we can do it all" approach? Does the psychiatrist with his associated status denigrate the developing identity and pride of the young generalist trainee and his teachers? Does he represent a punitive "big brother" function? These are factors that should be worked with so that they will not subvert the promise of a new health-mental health collaboration.

One promising vehicle for productive interaction between health and mental health practitioners is an interdisciplinary team approach. These have existed mainly in training programs thus far and have been difficult to establish in the real world because of lack of real administrative autonomy or support for the team in most programs, the costs of team meeting time in terms of measurable benefits, and the relative absence of training in true settings in most health care professions.

DISCUSSION

This paper has described mental health practice within a primary health care program—the roles, services, provider relationships, and cost issues. In the primary health care programs of the future, there is an advantage to be gained in incorporating mental health programs that go beyond the traditional consultation and liaison model, which is more suitable for mental health input into medical inpatient wards. The consultant or liaison person often functions in these settings as an emissary between separate departments or clinics.

If the new primary health care settings are to maximize an integrated approach to health care, they should bring mental health providers into the primary health care programs for direct services as well as consultation (12).

The advantages of such an integrated health-mental health endeavor include 1) decreased stigma associated with mental health services and less patient resistance to referral; 2) greater family orientation of mental health services, which are shaped by the family orientation of the setting they are in; 3) increased coordination of care; 4) more effective treatment of psychosomatic illness; 5) earlier contact with the patient and family, resulting in more opportunity for preventive services through earlier detection and education; and 6) less duplication of effort and greater manpower efficiency.

To maximize these advantages, an integrated program should incorporate the following general principles, based on existing mental health programs in primary health care settings: a broad spectrum of ambulatory mental health services on site, mental health staff who work at least 60% of the time (preferably full time), delivery of mental health and health care in the same contiguous space to promote communication, some form of interdisciplinary team activity, and accountability by the mental health care program both to the primary health care program and to any sponsoring mental health organization. One might ask at this point whether increasing the primary health care provider's knowledge of and readiness to refer patients to the mental health provider is in fact desirable if it increases demand and thus reduces the apparent insurability of such services.

Increasing the availability of mental health services will necessitate increased attention to the initial evaluation of the patient and to more appropriate use of a greater variety of resources and treatment modalities. This can meet the increased demand for services and better match the service to the need, thus helping keep costs in bounds.

The mental health staff presence can also facilitate higher quality mental health services by a larger group of health care providers, thus enlarging the effective mental health manpower pool. Two studies (13, 14) have shown that mental health services may decrease utilization of health services, but further studies are needed to validate this finding.

These integrated programs can originate from different sources. One possibility is linkage with a CMHC which can deliver much of its ambulatory care through primary health care programs to expand its availability and effectiveness. If the primary care program is neighborhood-based, the decentralization enhances accessibility. The CMHC can in turn provide backup subspecialized services that are not appropriate for the primary care site, such as hospitalization, partial hospitalization, drug abuse treatment, community residences, and special services for the mentally retarded.

Integrated health-mental health programs have been slow to form, in part due to lack of leadership. The existing separate health and mental health systems are preoccupied with developing their own programs and often find integrated services not among their short-term interests or priorities. There must develop both consumer and professional leadership that sees the field of health as a whole and provides the stimulus and

accountability to integrate our fragmented systems of care.

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The Future of State Mental Health Programs for the Chronic Psychiatric Patient in the Community

BY ROBERT L. OKIN, M.D.

The author explores the problems facing the chronic psychiatric patient in the community and discusses the response of federal and state governments to these problems. Errors of the past and present are delineated and corrective actions are suggested for the future. Finally, it is argued that if society is able to meet the challenge posed by the chronic psychiatric patient in the community it will simultaneously have set up mechanisms to solve the problems of other disabled and disadvantaged groups of people as well.

THE PROBLEMS of the chronic psychiatric patient in the community and the future role of the states in dealing with these problems are of critical importance for several reasons. First, the problems faced b[illegible] psychiatric patient in the community cons[illegible] lic health issue of the most enormous[illegible] Moreover, the way in which a state gra[illegible] responsibility for this group of patients [illegible] nificant implications for the way it deals [illegible] lems of other disadvantaged groups. Fi[illegible] few years have witnessed profound change[illegible] of the chronic psychiatric patient. These [illegible] continue in the future and must be unders[illegible] fessionals and citizens alike so that their [illegible] be shaped by the needs of our patients a[illegible] by extraneous, although powerful, politi[illegible] nomic considerations.

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THE SITUATION OF DEINSTITUTIONA[illegible] PATIENTS

Over the last 20 years, states have dis[illegible] sands of chronic patients from their institu[illegible] is no doubt that many of these patients ha[illegible] greatly from life outside the institution. M[illegible]

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however, continue to be discharged into communities that are unprepared to accept them, are actively hostile toward them, and are certainly unable to provide them with the comprehensive range of services they require.

Many patients who had been institutionalized for years now find themselves living in low-cost rooming houses, rocking in front of television sets, and wandering the streets. The level of welfare payments, on which many chronic patients depend for their livelihood, is set so low by both the federal government and the states that they are often consigned to a life of abject poverty. In no way am I implying, however, that community life is their first brush with poverty. Poverty characterized their lives in institutions as well, but it was both less visible and, unfortunately, more readily tolerated by society because it did not directly affect the lives or property values of the ordinary citizen. Moreover, its most pernicious consequences were often masked by the pseudoprotective characteristics of institutional life. Outside the institution, however, the poverty of the disabled patient is extremely visible and often manifested in poor nutrition, substandard housing, and inadequate access to transportation, work, and medical services. In fact, it is often the effects of the patient's poverty, rather than his mental illness per se, that has become the salient feature of deinstitutionalization in those cases in which the process has miscarried. For many patients, then, the burden of poverty has been added to the burden of mental illness, resulting in multiple episodes of decompensation, reinstitutionalization, and discharge. Such is the shame of the states—individually and collectively.

Poverty is not the only problem faced by the mentally ill in the community. Resources necessary to provide aggressive outreach, aftercare, vocational rehabilitation, and extended employment have never been sufficiently forthcoming to serve the many patients being released into the community. While patients are being released from the institution at geometrically increasing rates, resources for community services are being appropriated at a rate that is barely arithmetic. Far too little money has been provided by state and federal governments to undertake the kind of radical transformation of the mental health system that the deinstitutionalization process requires if it is to amount to anything more than a mere change in the locus of care.

ECONOMIC AND BUDGETARY CONSIDERATIONS

Initially, it had been thought that massive reallocation of resources would inevitably accompany decreased state hospital utilization, thereby reducing the mental health budget or at least permitting level funding. In certain cases this was almost promised by mental health advocates in return for legislative and executive support of the deinstitutionalization process. There developed, in consequence, a strange alliance between mental health advocates and state budget directors in which both advocated a reduction in state hospital utilization, although clearly for different reasons. As with most unholy alliances, this one ultimately became unglued when it became clear that vast amounts of money were not to be immediately saved through the deinstitutionalization process. This was true for a number of reasons, outlined below.

1. State hospitals initially lost their least disabled patients to the community. These were the patients who had worked at the institution, usually without pay, in the dietary service, laundry, on the grounds, and with the nursing staff, taking care of the more disabled patients. In short, the first patients to leave the institution in large number had essentially served as an unpaid labor force, which the institution then had to replace with paid staff in order to keep its operations going. The institution was then faced with the embarrassing paradox of requiring more staff at the very time its patient census was decreasing.

2. The problems cited in item 1 were compounded by a wave of right to treatment litigation, increasingly rigorous standards of hospital certification accompanying federal financial support, and a change in the orientation of the institution from mere custody to active treatment.

The combined result of these factors was an increased need for institutional resources, not a decrease. For many patients who had been discharged into the community in the expectation that resources and services would follow them from the institution, the results of this miscalculation have been tragic.

Given the difficulties of achieving significant reallocation from a reduction in institutional census, many states have sought to close certain of their institutions. In this way, it was hoped, the monies historically tied up in the maintenance of the physical plant could be released for community services. This strategy may have made sense programmatically but has been difficult to implement politically, for the institution over the years had become the source of major revenues to the surrounding towns and an important source of employment. Legislators and town officials have opposed and will always oppose the commissioner of mental health when he attempts to close a particular institution. This opposition will be joined by employees who depend on the institution for work. The commissioner will then be forced to choose between the most disadvantaged of his patients and the most disadvantaged of his employees. Assuming he does succeed in actually closing an institution, the commissioner will find his plan for reallocation opposed by yet another force: the state budget director, who will often attempt to divert for other purposes the savings that accrue from institutional consolidation. A prior commitment from the governor and the ways and means committees must be obtained if these hard-earned savings are to be kept within the mental health system.

Although the federal government is also under financial pressure, this is in no way comparable to that of

the states. When one considers that the cost of one B-1 bomber exceeds $93 million, it becomes clear that the issue before us is not an absolute lack of resources but rather the ways in which our national resources are being spent. The future of state mental health programs will be grim indeed unless the states are able to force a radical shift in our national priorities.

CMHCS AND THE CHRONIC PATIENT

At a minimum, this will require a significant change in the direction of the CMHC program. Despite the findings that prompted the CMHC legislation in the first place, the community mental health statute has not until recently adequately focused on the needs of the chronically ill population. For example, residential services and vocational rehabilitation, both essential for the disabled patient, had not been considered until last year "essential services" within the legislation. Influenced by this, and compounded by a lack of interest in the very disabled patient among many professionals, CMHCs have often allocated only a small percentage of their resources to the chronic psychiatric patient. Although the most recent version of the CMHC statute has been modified to include a requirement that CMHCs provide services to the chronic patient, no new monies were appropriated for this purpose. In addition, the programs of many CMHCs have been organized and delivered at the level of the federally defined catchment area rather than neighborhood and town levels, so that many centers have been unable to bring to bear on their disabled population the critically needed services of natural community caregivers such as the YMCA, the clergy, and the local housing authority.

Aggravating the problem further has been the relative isolation of the state hospital system from the community mental health system. The two systems, one operated by the state and the other influenced by the federal government, have not only been virtually divorced from one another but have competed actively for resources. The future requires the integration of these systems so that the CMHC screens those patients at high risk for institutionalization, links them with their families during institutionalization, and provides or arranges a comprehensive range of human services for those who require such services after discharge.

THE FEDERAL ROLE

A discussion of federal financing and its effects on the future of state mental health programs must go beyond delineating the problems of the CMHC program, for the issue is much more complicated, as was noted by the Comptroller General of the United States in a recent report to the Congress on the subject of deinstitutionalization (1). In this very significant document, the General Accounting Office c[illegible] lack of commitment by the federal gov[illegible] needs of the chronically disabled popu[illegible] has it been difficult for disabled patients [illegible] to the resources of certain mainstream [illegible] such as the Department of Housing an[illegible] opment and the Social and Rehabilit[illegible] they have been discriminated against [illegible] the Medicare and Medicaid programs [illegible] working together are successful in re[illegible] this situation, their ability to provide [illegible] ices to their disabled patients will con[illegible] tremely limited.

No discussion of financing would be [illegible] out some reference to national health [illegible] Although often viewed as a panacea fo[illegible] problems, most NHI proposals disc[illegible] the mentally ill in general and the chron[illegible] ill in particular. In addition, national h[illegible] proposals are not complemented by a fi[illegible] nism for those social services which are [illegible] if the insured health services are to [illegible] there is no financing mechanism to ge[illegible] sues, NHI is unlikely to have a signifi[illegible] the chronic psychiatric population. M[illegible] NHI proposals are based on an individ[illegible] model. This model by itself will do little [illegible] development of the integrated system [illegible] by the most disadvantaged of our patie[illegible]

ORGANIZATION AND INTEGRATION OF SERVICES AT THE STATE LEVEL

Financing is and will continue to be [illegible] ence on the future of state mental hea[illegible] the disabled patient in the community [illegible] that go beyond financing and relate to the [illegible] of services. Indeed, it is remarkable tha[illegible] failed to appreciate the extent to which [illegible] was, for many patients, an integrated [illegible] system that provided medical, nutrition[illegible] residential, legal, and economic service[illegible] inadequately. Many states did not rec[illegible] portance of replacing this system of ser[illegible] patient had left the institution. Those st[illegible] not recognize this either felt it was not t[illegible] bility to provide or arrange for these se[illegible] not in a position to do so because of fis[illegible] regulatory obstacles, or lack of power [illegible] availability of other human services.

The provision of a range of human s[illegible] disabled client has been and will contin[illegible] difficult in the community than it is in t[illegible] In the institution the department of men[illegible] total operational control of these servi[illegible] did not have the resources to use this c[illegible] vide its clients with a decent quality of lif[illegible] munity, the department of mental health [illegible] the resources but the power, for it clear[illegible]

ational authority for the medical system, the employment situation, and the housing market. And yet some organizational entity at the state level must assume the responsibility for trying to influence these systems, even if it does not control them. In this sense, the role of the state mental health authority must shift from one of direct service to one of planning, integrating, sanctioning, monitoring, priority setting, and funding. It is the integrative role I am focusing on here, for unless the state mental health authority can influence those systems that it does not directly control, the disabled patient will not have access to the range of human services he requires.

Finally, it must be said that many states did not adequately understand the radical nature of the changes that would be necessary for community care of the disabled citizen to succeed. The problems inherent in long-term institutionalization in large, antiquated, understaffed state hospitals and state schools were so compelling that many states failed to appreciate the potential for problems in the community. It has become abundantly clear that an improvement in the quality of life for many of our clients does not occur automatically as a natural consequence of their discharge from the institution. Social isolation is possible in cities and towns as well as in institutions. Indeed, in many places such isolation, as well as outright hostility, tends to be the rule rather than the exception unless aggressive measures are taken to prevent or deal with it. A fundamental transformation of community attitudes must be accomplished by the mental health system if the lives of our clients are to be enriched by discharge from the institution. Tremendous effort must be expended if the age-old ambivalence of our society toward the mentally ill and mentally retarded is not to reassert itself. Many of the same anxieties and fears that prompted exclusion from the community in the first place remain powerful today and must be altered if the real goal of community services is to be realized. Such a change in attitudes cannot be effected primarily by staff. Only citizens and legislators providing a kind of moral leadership in their own communities can really accomplish this mission through education, persuasion, and active struggle. Unless this alteration of community attitudes is successful, our clients will feel more isolated and alone in their new neighborhoods than they did in their old institutions, despite the services which may be afforded them. If this occurs we will have failed our disabled citizens miserably. Only the form and not the substance of their problems will have been changed.

CONCLUSIONS

Clearly, the states and their legislators are facing an enormous challenge. Success in meeting this challenge will necessitate 1) an organized effort on the part of the states to influence the federal government's responsiveness to the chronic psychiatric patient, 2) an infusion of new resources, 3) a radical transformation in the pattern of financing to permit funding of the least restrictive and most normalizing types of settings and services, 4) an integration of the mental health system with other human service systems, 5) a decentralization of services to the level of the neighborhood and town, 6) a more vigorous utilization of the natural caregivers which exist only at these levels, and 7) an increased emphasis on the role of citizens and legislators, who ultimately have the greatest ability to alter community attitudes and thereby reduce the extent of the hostility which often greets the disabled patient when he returns to the community.

The nation's success in solving these problems will have vast implications for the solution of similar problems faced by other disadvantaged groups—certain elderly people, the mentally retarded, people with severe physical handicaps, and people who have been deprived by virtue of extreme poverty. If the states, their legislators, and commissioners, as well as the federal government, are able to meet the challenge posed by the chronic psychiatric patient, it is likely that they will simultaneously have set up mechanisms to solve the problems of these other groups as well.

It is not an exaggeration to say that the chronic psychiatric patient challenges not only the mental health system and the human service system of the states but the very structure of our society—its behavior, values, and beliefs. It has been said that a society can be judged by the way it treats its least fortunate members. Will the states' response to the chronic psychiatric patient stand up to such judgment?

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Private Practice of Psychiatry: Future Roles

BY ZIGMOND M. LEBENSOHN, M.D.

Psychiatrists in private practice have contributed more to treating the mentally ill in America than is generally known. The private practice sector may be viewed as a massive national outpatient and inpatient service for the mentally ill. The impact of national health insurance on the future character of the private practice of psychiatry and the thorny problems to be resolved are discussed and compared with the experiences of England, the Soviet Union, and China. Future trends will most likely include increased emphasis on adjuvant techniques, greater emphasis on resident training in general psychiatry, briefer forms of therapy, and psychiatry's return to the medical model.

Alas for those that never sing
But die with all their music in them!

—Oliver Wendell Holmes, "The Voiceless"

PSYCHIATRISTS in private practice have had a singular ineptitude for telling their story. In spite of their major contributions to the efficient delivery of mental health care to a substantial segment of the population, their achievements have gone largely unsung. The reasons for this are multiple and complex. Most psychiatrists in the private sector are so swamped by their clinical responsibilities to their patients that they have little time or energy to compile statistics, write about what they are doing, or mount a campaign to quantify their results. They are characteristically a hard-working, dedicated group who devote 90% of their time to treating their patients, a task that is often exceedingly demanding. Thus they tend to be ineffective spokesmen for their cause. Possibly as a result of all these factors, psychiatrists in private practice have been called "an endangered species" by critics from both within and outside the profession, and they well may "die with all their music in them."

It was not until 1968 that the American Psychiatric Association finally recognized the need for a study of private psychiatric practice in the United States and appointed a task force to study the
years, 2 chairmen, and various co
was finally published in June 1973
and Future Importance of Patterns o
ric Practice in the Delivery of Me
ices" (1). This report, in spite of
weaknesses, is well worth reading.
derived primarily from the America
ation, the 1972 survey of mental he
tucky, and various other National
Health, American Medical Assoc
sources. This report concluded that

> the private practice sector of psy
> most essential element in the deliv
> services in our country, and one whic
> at this time. . . . In general, the aver
> he is sick prefers to see a physician
> willing to pay for his services in the
> chosen, the physician is *his* doctor.
> revealed in their relationship will
> fidential, that the doctor will be avail
> and that the doctor will have acces
> hospital facilities . . . adding up to a
> system that provides for continuity of
> agement. . . . Admittedly the private
> not meet all of the health needs of the
> must be supplemented by a wide rang
> *But the end product should be a he
> each reinforcing the other.* Our po
> system and our present day culture c
> velopment of a single unitary model
> health services. (1, p. vi)

It is quite probable that the immin
of national health insurance inspire
tion Service in 1973 to launch a nati
vate office practice. This study, pre
with a text by Dr. Judd Marmor, wa
with commentaries by experts in va
general, the methods used in this st
phisticated and comprehensive than
APA task force but still depended la
to a lengthy questionnaire. Psychiat
notoriously loath to answer question
ly the lengthy ones needed in this
Hence, the extraordinarily high res
not only an eloquent testimony to
the questionnaire but also suggests
in private practice have finally be
concerned about their own future
species to take the time to answer such

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HIGHLIGHTS OF THE KENTUCKY SURVEY

According to the survey of mental health needs in Kentucky conducted in 1970, the private practicing psychiatrists of that state treated approximately 12,300 people. The APA task force report concluded that private psychiatrists treated nearly one-half of all persons who received psychiatric treatment that year. The report went on to say,

> The private practice segment of the profession in Kentucky is the largest component of the state's mental health delivery system. This finding quite possibly may be applicable to the nation as a whole. . . . It is useful to picture the private practice sector of psychiatry as a massive national outpatient and inpatient service for the mentally ill, and one that meets the service needs of about half of all emotionally disturbed persons who seek professional help. (1, pp. 15, 27)

Sharfstein and associates (3) point to "the statistical hazards inherent in projecting findings from a single state to the nation as a whole . . ." and show that "more modest claims regarding the role of the private practice sector of psychiatry might be more appropriate even in the state of Kentucky. . . ." Statements in the report and the critical rejoinder by Sharfstein and coworkers point out the futility of such comparisons between the private and public sector: the situation is not an "either-or" one, and it seems clear that we must end with a viable mix of the best of both systems.

HIGHLIGHTS OF THE JIS SURVEY ON PRIVATE PRACTICE

It will not come as a surprise to most psychiatrists that the JIS survey reported that one-to-one psychotherapy is the dominant modality used by the vast majority of psychiatrists in private practice, although a substantial number occasionally combine it with adjuvant techniques such as group, conjoint, or family therapy. The use of these other techniques tends to be more widespread among child psychiatrists and general psychiatrists than it is among psychoanalysts. Marmor, in his commentary, regretted this tendency among analysts and urged greater flexibility in the psychoanalytic sector for the greater benefit of patients.

As might be expected, the added use of small-system modalities, with chemotherapy when indicated, seemed to shorten the course of treatment. With the imminence of some type of national health insurance and its understandable emphasis on the cost-benefit ratio, pressures will be exerted on psychiatry to use these less expensive methods.

The number of patient visits per year in the JIS survey appears to be significantly higher than in the Kentucky survey. The JIS survey reported an average number of psychotherapy visits per year for nonanalytic patients of only 38, and the figure was even lower for those receiving adjunctive chemotherapy. The average number of visits per year for analytic patients was 140. Although Senator Kennedy in his commentary said he was "personally convinced that this therapy (psychoanalysis) is the best medical science can offer to many patients who are in deep need of treatment," he made the shrewd observation that

> the average cost of a year's analysis (at 140 visits) today would be over $6,000. In face of this very high cost, it is distressing to note the relatively limited attention being given in private psychiatric practice to group and other treatment modalities that hold apparent promises for more efficient, less costly treatment. . . . Finally, grave problems remain with respect to equity. If we were to implement a comprehensive National Health Insurance Program tomorrow . . . we would be asking the 80% of American families whose earnings are under $20,000 a year to pay the lion's share of the cost of a health care service which is rendered by and large to individuals in families whose incomes are over $20,000 a year. Moreover, we would be asking black families to pay taxes for services only 2% of which go to blacks. And, of course, we would be asking residents of areas of the country which have few psychiatrists available to help pay the bill for other areas of our nation where help can be more easily obtained. (2, pp. 150–152)

Marmor is quite properly concerned by the double-edged nature of overfrequent sessions. Most general psychiatrists would agree that most patients treated on the average of once a week do every bit as well as those seen 3 or 4 times per week. He emphasized that it is not the frequency of therapy that is the crucial determinant but rather its *duration*. Franz Alexander's observation (4) that high frequency of sessions may be antitherapeutic and even lead to excessive dependency on the therapist has been made many times (before and since) by seasoned psychiatrists who sense this danger and take steps to wean the patient from this unrealistic type of support before it is too late.

Whether analysts would see their patients more frequently if all visits were covered by a health insurance scheme may be open to question. If they did so it might raise very serious ethical, technical, and fiscal problems. In this connection Marmor refers to some recent data collected by the late Dr. Louis Reed, compiled from the Blue Cross/Blue Shield plan for federal employees, which pays 80% of psychiatric fees above a $100 annual deductible. Reed found that in 1973 in the Washington, D.C., metropolitan area, psychiatric patients with charges of $5,000 or more for that year constituted only 3% of all persons with supplemental mental health charges but incurred 19% of the total charges for mental illness. For the United States as a whole it was found that 2% of persons (all or most of whom were presumably in analysis) accounted for 17% of the charges.

COST OF PRIVATE AND PUBLIC SERVICES

Because of the time-consuming nature of one-to-one therapy, the cost per unit of service is high, and the

patient faces a serious financial burden, especially if he is not covered by some form of insurance. This gives rise to much loose talk about high fees and fat incomes enjoyed by psychiatrists. However, the AMA reported that psychiatry ranks next to last among 8 specialties in yearly net income. Only pediatrics ranks lower.

The difficulties of making a meaningful comparison between costs of one hour's therapy by a private psychiatrist and by a psychiatrist in a CMHC were clearly presented by Sharfstein and associates (5). The JIS median cost figure for therapy by a psychiatrist was $35 an hour in 1973. Data from 53 CMHCs in 1974 indicated that the cost of one hour's psychotherapy by a psychiatrist in a CMHC "could be approximated at $45 per hour" (5). The CMHC estimates ranged from $20 to $70 an hour. Again, we see an example of the futility of comparisons between apples and oranges, which benefit no one.

ATTITUDES OF PRIVATE PSYCHIATRISTS TOWARD NATIONAL HEALTH INSURANCE

American psychiatrists have traditionally been more liberal politically than doctors in most other branches of medicine. Although no individual poll can be cited, it is my impression that most psychiatrists feel that some form of national health insurance is inevitable; most of them hope, as does their parent organization, the APA, that mental illness will have equal coverage with other illnesses under any form of NHI. Most psychiatrists of my acquaintance in private practice have a rather fatalistic attitude toward the possible advent of national health insurance. Perhaps they are lulled by the fact that most of them have full practices and their patients seem to be satisfied with their services. This may explain the results of a 1973 Harris poll which showed that even though confidence in all institutions was declining, the American public still had a higher degree of confidence in medicine as a profession than in such institutions as higher education and the military. All of this suggests that the American public trusts its own doctors and may not be as dissatisfied with the present system of medical care as is so often reported.

It is also my impression that most private practitioners would tend to agree with Eli Ginzberg's observation that

> the shortcomings and defects of the health care system are not likely to be cured by national action alone. The problems of access, availability, and quality of care are not likely to be solved through national legislation and administrative action because of the tremendous variability and the distribution of resources among and within regions and small areas of the country. The only way a national solution might work is if the entire system were under total government control—doctors, hospitals, patients. Yet even in Yugoslavia and Bulgaria, which have such systems, I have found that access, availability, and quality are a long way from being assured to the entire population. (6, p. 18)

Unless there is a radical and unforesee[illegible] democratic form of government, with i[illegible] the free enterprise system, it is unlike[illegible] care will be placed under "total gove[illegible] trol" in America. However, a quick lo[illegible] happened in other countries may give [illegible] to what the future may hold for those [illegible] tice.

EXPERIENCE IN OTHER COUNTRI[illegible]

The British National Health Service [illegible] istence since 1948. Although there hav[illegible] from time to time, most Britons wou[illegible] there to stay. Under the British syste[illegible] tice is not outlawed, and many psychi[illegible] part-time private practice in additio[illegible] tients assigned to them under the natio[illegible] Citizens who have the means to con[illegible] chiatrist are able to obtain treatment [illegible] have greater latitude in choosing a p[illegible] though this practice is periodically at[illegible] bour Party and unions on the grounds [illegible] festly unfair and that the right to good [illegible] never be affected by the power of one [illegible] private practice in Britain has continue[illegible]

In the Soviet Union I expected to f[illegible] medical care under total governmental [illegible] Ginzberg described it. However, I was [illegible] prises. On my last trip to Leningrad, [illegible] schoolteacher in her 40s who worked f[illegible] ing school vacations. Toward the end [illegible] after visiting several psychiatric inst tu[illegible] fided to me that she had consulted a ps[illegible] had benefited greatly from his help. [illegible] seen the psychiatrist at the "Dispe[illegible] trict. She replied emphatically and [illegible] "Oh, no! I see him privately at his hom[illegible]

One is reminded of an episode in [illegible] *Cancer Ward*. Dr. Lyudmila Dontsova [illegible] believed somewhat naively but fanatic[illegible] viet medical system of total governmen[illegible] when she developed symptoms of cance[illegible] became terror-stricken at the possible [illegible] and, instead of consulting her colleagues [illegible] went as a private patient to her former [illegible] Oreshchenkov, an older man, now ret[illegible] still permitted to conduct a small privat[illegible] dialogue between Dr. Dontsova, the y[illegible] state-controlled medicine, and Dr. Ores[illegible] saddened but wise old exponent of p[illegible] and personalized service, brings out the [illegible] weaknesses of both systems, with the g[illegible] model emerging as the superior and m[illegible] system of ministering to the sick.

The few reports available concerning [illegible] psychiatry in the People's Republic o[illegible] dicate that their entire health system [illegible] more rigid and total governmental contr[illegible]

case in the Soviet Union. It is doubtful, for example, that any private practice exists there today. Some observers have commented astutely that the reduction or elimination of prostitution, drug addiction, unemployment, begging, abject poverty, starvation, venereal disease, and crime, and the general improvement in public health that has occurred since the ascendancy of Chairman Mao have done more for the mental health of 800 million Chinese than the efforts of all the world's psychiatrists and mental hygiene movements put together. Perhaps so, but most of us who live in Western democracies would consider the absolute and total conformity to a monolithic political system too high a price to pay. The example of China, however, points up the truism that shapes the future form of the practice of medicine in any country—namely, that any system of medicine exists at the pleasure of the society it serves. Society giveth and society taketh away. Political systems such as ours, which permit a diversity of medical practice, may be more inefficient at times, but their great strength lies in the freedom given to the human mind to develop new and better methods to serve the sick. In this freedom lie both the warning and the challenge to the private practice of any profession, including psychiatry.

FUTURE TRENDS

It is my firm conviction that the private practice of psychiatry in the United States will not only survive but will continue to flourish. Fewer psychiatrists will be going into solo practice, probably because of its special stresses, and more psychiatrists will be entering some form of group practice. Third-party coverage for psychiatry will continue to increase both in the numbers of those covered and in the psychiatric benefits provided. This is especially true of outpatient visits to a psychiatrist's office. Most health plans, as well as NHI (when it arrives), will have limits on the number of visits per year, which number may well correspond to the average of 46 found in the JIS survey for all patients (analytic and nonanalytic). Whether the limitation is placed on the total number of visits or on the dollar value of these visits, such a step is sure to influence mightily the character of psychotherapy in America. My own feeling is that this influence, when it comes, will put psychiatrists on their mettle to develop more efficient and briefer forms of psychotherapy, which will most likely involve the greater use of adjunctive methods such as chemotherapy and group therapy. People with emotional problems will continue to seek out private psychiatrists when that is the most appropriate step to take. Peer review will exert its influence on all aspects of practice, and ways will be found to circumvent one of the serious problems attending such review, namely, the erosion of patient-doctor confidentiality. The second problem that has already emerged from peer review will be more difficult to counter. I refer to the tendency to conformity. This tendency could well stifle development of innovative treatment methods and encourage mediocrity, to the detriment of the patient's health. In spite of all these problems, psychiatrists' schedules will continue to be filled even though their income may be somewhat less; they will still be able to live comfortably while pursuing their chosen field.

Changes in Psychiatric Education

Medical schools and psychiatric residency training programs will slowly but surely begin to pay more and more attention to training residents for the general practice of psychiatry. This means placing a greater emphasis on the medical model and the biological roots of psychiatry. Gerald Klerman (8) recently listed the current hierarchy of psychiatric therapies in descending order as follows: 1) insight therapy, 2) supportive therapy, and 3) pharmacotherapy. He then added that "the degree of involvement of the profession in these three therapies is in inverse proportion to the proven evidence of their effectiveness." It is now high time that the profession, and particularly psychiatric residency training programs, recognize this important fact and revise the curriculum accordingly. I believe it is inevitable that they will do so.

Group Therapy

I anticipate a sharp increase in the use of group therapy in private practice. The indications and contraindications for this specialized approach will be sharpened and new techniques will be developed. The group therapy movement will become more solidly entrenched in American psychiatry, and some of the "far-out" methods will fade into oblivion. The reasons for the phenomenal growth of group therapy in the last decade are complex, but certainly the economics of reaching larger numbers with limited personnel is one of them. Aside from that, group therapy will continue to fill a very real need in the lives of thousands of unhappy, lonely people living in our overcrowded urban centers. For these maladjusted people who have had so much difficulty in establishing satisfying interpersonal relationships, group therapy will continue to be the treatment of choice.

Social Psychiatry Versus the Medical Model

Psychiatry, frustrated by its flirtation with sociology, will return firmly to the medical model, from which it should never have strayed. Some will choose to call it the "biopsychosocial" model, a term coined by Engel (9), which includes the biomedical, psychological, and sociological aspects of all illness. Engel points out the dangerous dogmatisms of the biomedical model when it is adhered to with fanatic singlemindedness. However, the medical model, as I use that term, is broad enough to include the psychological, social, and biological factors that may be present in any illness, and the future will see its reemergence not only in the medical school but in the private practice of general psychiatry.

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Will Community Mental Health Survive in the 1980s?

BY STEVEN S. SHARFSTEIN, M.D.

There are now 675 funded community mental health centers (CMHCs), covering almost half the country. Many of these programs were funded in the social optimism of the 1960s and now face a crisis of purpose and funding. Additional requirements imposed by the 1975 amendments to the CMHC act are not matched by additional fiscal resources. Programs are graduating from the federal grant to find that other sources of funds, especially third-party insurance funds, are not replacing the lost federal dollars. There is evidence that CMHCs are changing from clinical/medical programs to social programs; the numbers of persons seeking care who have diagnosable mental illness and of psychiatrists and nurses relative to other staff are decreasing. The issue is whether CMHCs as a national program are headed for extinction or whether there will be new vitality for this program into the 1980s.

IN THE GENUS of social programs called human services, a species known as the federally funded community mental health center (CMHC) is [illegible] tinction. This is the story of that pro[illegible] and nurtured in the optimism and ab[illegible] the 1960s but coping with a precarious [illegible] the 70s, increasingly burdened by fi[illegible] and multiple levels of accountability. T[illegible] story is still in doubt. Whether the p[illegible] with a whimper, collapsing under the w[illegible] problems and changing times, or whet[illegible] ate a new vitality, producing a secon[illegible] service programs fitted for survival in [illegible] become evident in the next two years.

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

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Views expressed herein are those of the author and do not necessarily reflect the opinions, official policies, or positions of the Alcohol, Drug Abuse, and Mental Health Administration or the National Institute of Mental Health.

SURVIVAL FROM 1963 TO 1977

At the time of its inception in 1963 [illegible] mental health center represented a [illegible] proach" toward meeting the mental [illegible] the community. The scope of its mis[illegible] erately writ large—a minimum of 5 c[illegible] to be made readily accessible to all [illegible] of ability to pay, a commitment to ser[illegible] munity at large as well as to troubled [illegible] the same time it was assumed tha[illegible] period of declining federal financial su[illegible] ters would generate alternative source[illegible] become self-sufficient. After the firs[illegible] significant nonfederal sources did not [illegible] period of federal support was extended [illegible] CMHC program continues to rest on [illegible] after federal "seed money" initiates a[illegible] be replaced over time with increasing [illegible]

federal support. Much of this support has come from state and local funds and service receipts for older centers, with state, local, and third-party funding being a limited source of funding for centers in the initial years of existence.

The initial expectations of the new program were very high. Its success, although variable, was on the whole evaluated as satisfactory (1, 2) It managed to survive the dark ages of the Nixon-Ford years, which attempted to phase out the program on the basis that it was "a successful demonstration" and should therefore be picked up entirely by state, local, and private resources without any federal "seed money." It was the Congress that provided the impetus for its continued survival through Public Law 94-63, which clearly stated in its preamble to Title III that

> the Congress finds that community mental health care is the most effective and humane form of care for a majority of mentally ill individuals; the federally funded community mental health centers have had a major impact on the improvement of mental health care by (a) fostering coordination and cooperation between various agencies responsible for mental health care, which in turn has resulted in a decrease in overlapping services and more efficient utilization of available resources, (b) bringing comprehensive community mental health care to all who need care within a specific geographic area regardless of ability to pay, and (c) developing a system of care which insures continuity of care for all patients and thus our national resource to which all Americans should enjoy access.

Through P.L. 94-63, Congress provided for the survival of a "Great Society" program while many others perished. However, in its enthusiasm for this species of social and medical programming Congress may have created a modern day dinosaur, a huge and complex organism that requires nutrients which, from a Darwinian perspective, far exceed the capacity of the environment to sustain its existence.

From 5 essential services the comprehensive concept was expanded to 12 essential services. In addition to the original 5 services, P.L. 94-63 now requires services for children and the elderly, for persons with alcohol and drug abuse problems, and for the "deinstitutionalized" populations through the triad of screening, follow-up, and transitional living arrangements. Congress also added a number of compliance or accountability features designed to promote the accessibility and acceptability of the services provided. Requirements regarding the governing body, quality assurance, meeting certain cultural sensitivities, using at least 2% of the budget for evaluation, complying with the provisions of the law in 1 year and converting to the 12 services in 2 years, and implementing multiple reporting systems required by the federal government impose a significant and continuing management burden on CMHCs.

Additional pressures from state and local governments and from the private sector to provide data and to meet certain standards create a situation in which the CMHC is not only expected to be all things to all people but to document that happy status regularly and in great detail to a variety of audiences.

BUDGETARY SHORTFALLS

The idealism and competence of the program-oriented "authorizing" Congressional committees is not matched by the budget-oriented "appropriating" committees. Funds were appropriated at lower levels than authorized. For 5 of the 6 grant mechanisms under P.L. 94-63, funds made available by Congress were 60% below the authorized amounts; for a sixth grant mechanism, there was no appropriation. In each funding category in 1976, the first year of experience with the new law, there was a shortfall of funds leading to an impressive backlog of approved but not funded grants. The high number of projects recommended for approval but unfunded (123) amounted to $37.3 million in fiscal year 1976. At the end of FY 1977 the approved but unpaid account amounted to $37.8 million. If General Motors had to face the same backlog of orders for automobiles it would rapidly be replaced by a competitor, or a new mode of transportation would be invented! Those centers that have gotten funds have received less than the amount necessary to sustain costly clinical care—a full range of emergency, diagnostic, treatment, and follow-up services.

As of June 1978 it is estimated that there are 675 funded CMHCs. Sixty of these are alumnae of the federal CMHC programs, i.e., programs that have graduated from all forms of federal support and have not received any additional support from P.L. 94-63. An additional 329 are still being funded through the old limited staffing program, with no P.L. 94-63 funds involved, but they were expected to comply by August 1978 with the requirement of expanding to 12 essential services and the other P.L. 94-63 requirements for accountability. Without additional funds they might become "dropouts" from the federal program. Further, there remain over 800 unfunded catchment areas in the country. Today it is no wonder that there is cynicism based on the gap between the grand expectations of the federally funded community mental health center and the reality in the community, with massive unmet needs and budget shortfalls.

SOCIAL OR MEDICAL PROGRAMS?

One potential major source of funds for CMHC survival is health insurance. However, health insurance is based on an "illness" or "medical" model and the trend in CMHCs seems to indicate a shift away from the clinical or medical approach toward a "social" model. Trend data show a shift in diagnosis from 1970 to 1974. Almost 20% of all additions to CMHCs in 1970 carried a diagnosis of depressive disorders. This category dropped to 13% in 1974. For schizophrenia the

shift from 1970 to 1974 was from 19% to 11%. During this same 5-year period diagnoses of "social maladjustments" increased from 4.6% to 20%.

Complementing this diagnostic shift is a shift away from medical practitioner staff. In 1970 there were 6.8 average full-time equivalent psychiatrists per community mental health center; in 1975, there were 4.3. The number of registered nurses decreased from 9.7 full-time equivalents per CMHC in 1970 to 8.9 in 1975. During the same period the numbers of psychologists increased from 4.9 full-time equivalents per CMHC to 8.5 and social workers from 9.1 full-time equivalents to 12.2 (3).

Even for diagnosed mental illness, there is great difficulty in obtaining third-party coverage. Extra restrictions are enforced on reimbursement for psychiatric services in the Medicare and Medicaid programs and in most private health insurance programs. There are four types of restrictions in Medicare and Medicaid. First, no CMHCs are certified as providers under Medicare, and many states exclude CMHCs from provider status under Medicaid. Second, there is great variation in the eligibility of patients even when CMHC services are covered under Medicaid. For example, many poor patients seen in a CMHC are ineligible for Medicaid because Medicaid eligibility is tied to certain welfare categories rather than income. Third, benefits are limited and are often discriminatory toward the mentally ill, e.g., the limitation on the amount Medicare will pay for outpatient psychiatric care. Fourth, reimbursement, even when available, often does not fully cover the cost of services to Medicare and Medicaid eligible patients. Therefore, CMHCs remain on the periphery of third-party financing.

Although CMHCs improve their third-party status as they get into their sixth, seventh, and eighth years, this source still represents a relatively small source of support and cannot be expected to generate the funding necessary for comprehensive services that are closely coordinated with other social and medical agencies in the community. Health insurance does not pay for the time spent on the telephone, traveling between appointments, or consulting, i.e., meeting with school personnel, the police, social agencies, and dealing effectively with multi-problem families and highly disturbed individuals. It then is up to the state to provide the necessary funds, and state mental health authorities themselves are experiencing [illegible] getary shortfalls in attempting humane and sound programs for the chronic patient.

CONCLUSIONS

If the evidence bears out the notion that the CMHC program is mutating from a clinical-model program toward a social program, what does this portend for survival? If national health insurance will be only a small portion of the total program, can the companion social insurance program [illegible] ing pieces? Given the concern today about health costs—health care approached 8% of the gross national product this year—can the federal Treasury bear the costs of the CMHC program over the next 10 to 20 years?

These are difficult questions, posed [illegible] help the CMHC program cope, adapt, and [illegible] ing federal dollars, health dollars, social dollars, and private funds. It is clear that the goals and expectations of the CMHC program must be brought into line with fiscal realities. Too much has been expected of so few with so little, and priority-setting are critical, but planning cannot substitute for adequate support and resources for the community. The CMHC program, as an integral system, publicly financed and accountable in a treatment area, oriented toward quality clinical care and prevention, is a system worth preserving. The alternative is a return to the "economics" of the isolated large state hospitals and institutions for the many and the private mental hospital and practitioner for the few.

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Mental Health Care Delivery Systems: Discussion

BY BORIS M. ASTRACHAN, M.D.

THE PAPERS by Lebensohn, Okin, Sharfstein, and Morrill taken together present an overview of the treatment systems in which the work of our profession is performed. Much has been made of claims of fragmented or nonexistent delivery systems, but these papers demonstrate that within a number of different sectors, patients are receiving concerned clinical care. In all of the papers, the medical roots of psychiatry are evident and the goals of practice are emphasized: to prevent disease and disability when the knowledge base is adequate; to treat or to consult as to treatment in the face of disease or disorder; to cure when possible, to care always; and to identify disability and through the use of psychosocial interventions to restore functioning.

Psychiatry is represented as a bridging discipline, with important concerns about physical as well as mental health, and with required interaction with segments of the human services and social welfare systems. The four treatment systems clearly differ in the extent to which they encompass and/or interact with social welfare systems. To some extent, this reflects the nature of the patient population with whom each system is most concerned.

The private mental health care system revolves about the efforts of the individual practitioner. To a significant extent, changes in insurance practices have democratized access to private care. Currently services in this sector are available and used by members of the working class as well as the more affluent. An organized treatment system that integrates inpatient and outpatient activities is not required; as Lebensohn notes, the individual practitioner can provide for continuity of care. In this sector the greatest proportion of the practitioner's time is spent in providing direct clinical service.

The great strength of this system is paradoxically its most serious weakness. The usual form of practice is that of the isolated individual clinician responsible for and to his patients. Individuals with complex medical-psychiatric-social problems demand much time and assistance just to provide them with adequate entry into care. This is particularly true for those who are both fiscally deprived and psychologically disabled. These individuals are not well represented among patients in the private sector.

Okin speaks clearly for the needs of the destitute severely mentally ill. The state hospital system and those receiving services from it have often been devalued by society and their requirements neglected. Okin clearly points to our responsibility to provide for the chronically mentally disabled. He identifies not only the importance of medical and rehabilitative forms of care but also the social welfare needs of this patient population.

Within community mental health centers, clinical and social welfare goals have at times become confused. Sharfstein articulates the differing goals and processes in human services and in clinical settings and raises questions about the direction in which the CMHC movement will progress. At one point it had been hoped that combining clinical and social welfare services would in fact provide for more comprehensive care for individuals in greatest need. As Sharfstein notes, this usually has not happened within community mental health centers and too frequently the chronically ill have not been served.

Morrill identifies an important role for psychiatry in multidisciplinary group practices or in HMOs. The psychiatrist consults to other practitioners (particularly to generalists) and to other health professionals, assisting them in the management of relatively routine emotional problems and with some of the emotional complications of physical illness. The practitioner is also available to deal with the more difficult clinical cases directly. The health center serves as a locus in which medical care is emphasized but in which connections are directly available to mental health, rehabilitative, and social welfare systems.

In speaking to the future of health delivery systems, all of the authors identify the importance of fiscal considerations in defining organizational structures. As Okin notes, our patients have been discriminated against in provisions of Medicare and Medicaid and if such discrimination persists and is reified in whatever form of national health insurance is enacted, our patients will suffer and our treatment systems will be incompetent to make up for that suffering.

We do not anticipate that out of these differing systems any single monolithic one will be formed. However, legislated regulatory forces probably will increasingly insist upon better coordination of governmentally funded services. In particular, voluntary hospital, state hospital, and community mental health center services can be expected to be required to complement rather than compete with one another. To some extent, we already see general hospital services interacting with government-sponsored ones, either

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directly as part of CMHCs or, increasingly, as a preferred setting for the initial hospital treatment of those with insurance coverage.

Currently, each of the described treatment systems has its own unique advantages and limitations. Each performs some aspects of patient care most efficiently. The problem for psychiatry as well as for the rest of medicine is how to stimulate organizational systems to provide excellent care for individual patients without doing injury to the necessary pluralism in our health care giving treatment system. A major dilemma that is still only poorly addressed, even in theory, is how best to coordinate multiple service needs for emotionally, financially, and educationally disadvantaged patients. The failures of total institutions are well known. They are, however, matched by our inability to assist the severely disabled with medical, rehabilitative, housing, and financial resources.

BRIEF COMMUNICATIONS

A Comparison of Depression and Physical Illness in Men and Women

BY MELVIN L. SELZER, M.D., MARIA PALUSZNY, M.D., AND ROBERT CARROLL, M.D.

A limited self-rating depression scale given to male and female factory workers indicated that subclinical depressive symptoms were correlated with visits to the factory health service for females and with absenteeism from work for males. In general, the females acknowledged more depression symptoms.

IN RECENT YEARS there has been much research on various aspects of depression. Studies on the psychology, epidemiology, sociology, and neurobiology of depression are prominent in the psychiatric and related literature. Studies of subclinical depression are less frequent. We were interested in assessing a "normal" population to determine 1) the extent of depression in such a sample, 2) the possible correlation between depression and physical illness, and 3) possible sex differences in respect to these and other variables.

METHOD

Sample

The sample consisted of blue collar workers in a small rural factory where the males and females had identical occupations and similar backgrounds. Of 200 workers asked to participate, 159 responded to the first questionnaire (84 females and 75 males). Of these, 114 also took the second test (61 females and 53 males). There was a wide age range, 16–52 years, with a mean age of 28 years. Educational levels of the subjects ranged from seventh grade to 1 year of college.

When this study was done, Dr. Selzer was Professor, Department of Psychiatry, University of Michigan Medical School, Ann Arbor, Mich., where Dr. Paluszny is Associate Professor and Dr. Carroll was a medical student. Dr. Paluszny is also Clinical Chief, Institute for the Study of Mental Retardation and Related Disabilities, University of Michigan. Dr. Selzer is now in private practice, 6967 Paseo Laredo, La Jolla, Calif. 92037.

Measurement—the SDS

The Zung Self-Rating Depression Scale (SDS) (1) was used to assess the degree of depression. The SDS has been criticized by some researchers (2), but with the following minor modifications it served our purpose. First, before the test, we gave the following specific instructions to our subjects. "Answer all questions as they apply to you. If you feel an answer should be 'none of the time,' check the space 'a little of the time'; if you feel an answer should be 'all of the time,' check 'most of the time.' " We felt these instructions would allow us to compare our data with other studies using the SDS while reducing the number of questions left unanswered. Second, we added 4 questions (items 21–24): 1) I feel sad, 2) Things I try seem to go right, 3) I usually get what I deserve, and 4) I feel I get a raw deal out of life. These items were added to elicit additional data on depressed affect and self-esteem (numbers 1 and 2, respectively) and resentment (numbers 3 and 4).

Procedure

In order to assess the relationship between depression and physical illness, we correlated the SDS with absenteeism and visits to the plant health service (categorized as visits for injury or for illness).

The SDS was administered at the factory by a medical student. There was a 38-day interval between the two testing periods. The health records for the 38-day interval and for the 38 days before the first test were checked for health-related absenteeism for visits to the plant health service. The workers were told that all responses were confidential. The testing procedure took 15 minutes and subjects were paid $1.50 for filling out the questionnaire.

RESULTS

We found that females showed more depressive symptomatology than males at both SDS rating peri-

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TABLE 1
Comparisons of Illness Behavior by Sex*

Illness Behavior	Females (N=61) Mean	SD	Males (N=53) Mean	SD	Comparison Difference	
Absences						
Period 1	2.14	3.73	0.90	1.76	1.24	.05
Period 2	1.88	3.13	1.14	3.47	0.74	ns
Visits to health service for illness						
Period 1	2.20	2.74	0.20	0.60	2.00	.05
Period 2	2.05	2.54	0.36	0.76	1.69	.05
Visits to health service for injury						
Period 1	0.66	1.25	0.44	1.75	0.22	ns
Period 2	0.82	1.40	0.54	1.19	0.28	ns

Period 1=38 days immediately preceding the first SDS, and period 2=38 days between the first and second SDS.

TABLE 2
Correlation Matrix of Illness Behavior with Depressive Symptoms

Illness Behavior	Males Test 1	Test 2	Females Test 1	Test 2	Test 1	Test 2
Absences						
Period 1	.39*	.39*	.02	.12	.16	
Period 2	.01	.00	.12	−.02	.09	
Visits to health service for illness						
Period 1	.06	.02	.24*	.17	.26	
Period 2	.10	−.05	.24	.28*	.18	
Visits to health service for injury						
Period 1	−.08	.05	.01	−.05	.03	
Period 2	.19	.10	.02	−.02	.11	

Significant correlations ($p<.05$).

ods. On an item-by-item basis, females reported significantly more symptomatology on 9 items, whereas the males did not score significantly higher on any item.[1]

The females also showed more illness behavior, as indicated by work absences and by visits to the health service (see table 1). However, not all of these differences were significant. The most clearly significant finding was that females had more illness-related visits to the health service. Visits to the health service for injuries were slightly but nonsignificantly higher for females. Absenteeism was higher for females but the difference was significant only in the first 38-day period (before the first SDS administration).

There were interesting sex differences in correlations between depressive symptoms and illness behavior. In males, depressive symptomatology was positively correlated with absences from work in the first period. No such correlation was found for females in either period. Females did show a positive correlation between depressive symptomatology and visits to the health service for illness in both periods (see table 2).

[1]A table comparing individual test scores is available from the authors on request.

DISCUSSION

In this study the female subjects reported significantly more depressive symptoms than the male subjects in terms of total scores and scores for 9 individual items. Our data do not enable us to determine whether this represents a quantitative difference in the expression of symptoms, a greater willingness on the part of females to report their symptoms, or a difference in the ways in which the sexes view their symptoms. Some researchers cite sex as a source of heterogeneity in a meta-analysis. In a survey, Blumenthal (3) stated, "Men are expected to bear their troubles silently while women are encouraged to complain. When a woman weeps or has a headache or says she isn't feeling well, she is behaving within a culturally accepted framework, while a man who engages in the same behavior is not. It seems logical, then, that when a man makes similar complaints on a symptom checklist, such complaints are indicative of more serious problems than those of women." Blumenthal argued that it was therefore "unreasonable to compare normative scores for the two sexes." This is an important consideration in studies like ours, where the primary measures are based primarily on the individual's willingness to recognize or report his/her symptoms.

Our findings that females consistently exhibited more illness behavior than males is in accord with the conclusions of other investigators (4–6) and represents, in part, a greater readiness for self-referral. The importance of this factor has been reported elsewhere (7) and is alluded to by Blumenthal in the above quotation.

When the relationship between reported depressive symptoms and illness behavior was examined, females showed significant correlations only for one test administration and visits to the health service in the previous 38 days. Males, on the other hand, showed significant correlations for depressive symptoms and absenteeism for the same period. In all cases the manifest form of illness behavior was exhibited before the symptoms were reported on the questionnaires. Therefore, it is possible that illness behavior reflected symptomatology that had been present for several weeks or longer.

The ways in which males and females exhibit illness behavior related to their symptoms deserve special consideration. Females tended to visit the plant health service and complained of functional symptoms ("headache," "don't feel good," "feel tired"). Rarely did they complain of illness with objective findings. Social pressures in the form of taboos deny men this form of illness behavior. Therefore, it is possible that absenteeism is a form of illness behavior in men. This may account for the fact that absenteeism was the only manifestation of illness behavior significantly related to depressive symptoms in men.

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Blind Ratings of Mother-Infant Interaction in Home Movies of Prepsychotic and Normal Infants

BY HENRY N. MASSIE, M.D.

Home movies of infants later diagnosed as having early childhood psychoses were compared with movies of normal control infants. A scale of attachment indicators measured mother-infant dyadic avoidance and reciprocity in the first 6 months of the infant's life. Three judges blind to diagnosis saw a general trend to dampened interaction for prepsychotic infants and their mothers. Prepsychotic and normal infants were not significantly different in touching and eye gaze behaviors, whereas mothers of prepsychotic infants exhibited less adequate eye gaze and touching than mothers of normal infants.

A SERIES OF family-made home movies of the infancy of children subsequently diagnosed as suffering from psychosis, collected by researchers studying the early natural history of childhood psychosis, serve as prospective-like data for the study of mother-infant interaction, the initial signs of illness and their development, and the constitutional qualities and neurologic symptoms of the children who later become sick. Investigators aware of the children's diagnoses have reported on 3 early cases in the series (1, 2). The intrapsychic functioning of infants and their parents drawn from their psychotherapies was correlated with the observed behavior of parents with their infants in the movies. Infancy patterns of social interaction appeared to be longitudinally incorporated into and to characterize the child's later psychotic symptomatology. A third report (3) documents the appearance of symptoms for each of the first 10 cases studied. Key findings included the relative absence of symptoms in the first 6 months and the frequency with which abnormality was heralded by the failure of the children to develop mature communication of meaning, intent, and affect through facial expression at about [illegible] Many of the prepsychotic 1-year-olds [illegible] oped an aberrant plasticity or lability of [illegible] which moods and meaning seemed to [illegible] seconds.

This report presents the final phase [illegible] ratings of 13 subject and 15 control fa[illegible] three judges, blind to diagnosis and su[illegible] of the child's life, studying the fil[illegible] months of life of the two populations. [illegible] dren, diagnosed before 6 years of age, [illegible] range of childhood psychotic diagnoses [illegible] biotic psychosis, childhood psycho[illegible] schizophrenia, and mixed forms [illegible] clusiveness results from the vague and [illegible] syncratic distinctions therapists mak[illegible] noses. Additionally, it remains to [illegible] whether the different diagnoses repres[illegible] nesses, different syndromes of one i[illegible] uum in degree of severity of one ill[illegible] movies were obtained largely through [illegible] tutions and parent group newsletters. [illegible] were obtained "for a study of child[illegible] largely through nonpsychiatric instit[illegible] letters. Groups were matched for num[illegible] second-born children.

METHOD

The study of index and control cases [illegible] key modalities of mother-infant recipr[illegible] in the first 6 months of the infant's life—[illegible] ing, eye gaze, and touching (vocaliz[illegible] since the family movies were silent). [illegible] analysis these modalities were broken [illegible] ponent behaviors that I incorporated [illegible] attachment indicators used by the jud[illegible] grades the intensity of the mother's or [illegible] ment to the other from a low value of [illegible] maternal or infant aversion and isolatio[illegible] ue of 5 (indicative of intense responsive[illegible] ing). The purpose of the scale was to [illegible] tifying and verifying my impression [illegible] more maternal avoidance of attachme[illegible] and more infant apathy in the prepsy[illegible] one or more modalities than in the co[illegible]

Three judges, who were educati[illegible]

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health professionals, were trained for approximately 5 hours in the use of the scale. The judge was asked to study each scene in which mother and child engaged in holding, eye gaze, touching, or feeding and was encouraged to view the scene with single frame and slow motion speeds as often as necessary to select the numbered scale description that most closely reflected the behavior occurring in the scene.

Although the judges knew about the research hypotheses and that they would be given unedited movies of the infancies of normal and psychotic children, they did not know from which group a given film came. Films were coded and chosen randomly for each judge, who then viewed the films independently. All three judges saw all of the films. After they rated all the scenes in a case, the judges were asked to make an overall assessment of mother and child. They were asked to report their general clinical impressions, i.e., whether the infant and mother seemed "unusual," "healthy," "attached or avoiding," "content," or "distressed." Fathers generally appeared too infrequently in films to permit rating. The cutoff point of 6 months was chosen because no recognizable symptoms of psychosis or autism appeared in any case before 6 months of age and also because of the fundamental importance of the first 6 months for later development.

RESULTS

Differences in Intensity of Reciprocal Responsiveness

Nonblind study led to the hypothesis that normal mothers and infants should show higher mean ratings on the scale of attachment indicators and for the judges' general clinical impressions than the prepsychotic cases. T tests were performed to determine whether the observed differences between means were significant. One-tailed tests of significance were used with t values significant at $p<.05$.

Mean ratings were determined by averaging ratings made by each judge after he or she looked at all the scenes of a given attachment behavior (e.g., feeding, holding, etc.) for each index and control case. Thus, there were three average ratings (one per judge) for each case, from which a mean score was determined. In some cases, the judges felt that they did not have sufficient footage to make a clinical rating, so the actual number of cases used to determine mean scores was sometimes different from the total number of control and index cases.

As table 1 indicates, the overall ratings show that in each of the categories—feeding, holding, eye gaze, and touching—the trend went in the predicted direction. The normal groups of infants and mothers achieved higher values for attachment than the mothers or infants in the prepsychotic groups. When t tests were used to compare normal infants with prepsychotic infants, there was no significant difference between groups in feeding behavior, eye gaze, or touching. However, infants in the normal group showed significantly higher ratings for holding ($p<.05$) than infants in the prepsychotic group.

For the two groups of mothers, there was no significant difference in ratings for feeding. However, mothers of normal infants achieved significantly higher ratings for holding ($p=.05$), touching ($p<.05$), and eye gaze ($p<.01$). Thus, in two categories—eye gaze and touching—the two groups of infants were not distinguished, whereas the mothers of normal infants were distinguished by higher ratings than mothers of prepsychotic infants, and in one category—feeding—neither mothers nor children in the two groups were distinguished.

With respect to interrater reliability, one judge had to be replaced in the middle of the project; however, each case still received three ratings. The average of interrater correlations across attachment modalities ranged from .54 to .39. When the ratings of the two judges who rated the most cases ($r=.49$) were used to complete the same t tests separately for each of these two judges' ratings, both the trends and significance levels of the overall results of the pooled data were replicated.

General Impressions of Attachment

For the global impression of each mother and child, the three judges responded to a series of questions (e.g., "Does this mother/child seem healthy . . . strongly attached . . . avoiding . . . content . . . distressed?"). Without knowing the identity of the cases I sorted the responses into three categories that were weighted numerically: 1=negative responses (e.g., avoiding, not healthy); 2=neutral responses (e.g., healthy, content); and 3=strongly positive responses (e.g., strongly attached). The values for each case were averaged. A mean of 2 to 3 was considered indicative of normal attachment, a mean of 1 to 1.667 was considered poor attachment. Each mother and child in the normal and prepsychotic groups was assigned to cells representing poor attachment and healthy attachment. Of the normal infants 4 appeared poorly attached and 11 normally attached; of the prepsychotic infants 7 appeared poorly attached and 6 normally attached. A chi-square test with Yates correction for small numbers applied to these figures indicated no significant difference between the overall impressions of the two groups of infants. With regard to the mothers, however, 10 mothers of prepsychotic infants appeared poorly attached to their infants and 3 normally attached; in the control group only 2 mothers appeared poorly attached and 13 normally attached. This distribution was significantly different from chance ($p<.01$). The correlations for the general clinical impressions for pairs of judges ranged from .67 (the largest number of cases seen by a pair of judges) to .31 (the fewest number of cases seen by a pair of judges). The overall average correlation for the pairs of judges was .47.

TABLE 1
Ratings of Attachment Behaviors in Normal (Control) and Prepsychotic (Index) Infants and Their Mothers*

	Feeding				Holding				Eye Gaze				Touch			
Group	N	Mean	SD	t	N	Mean	SD	t	N	Mean	SD	t	N	Mean		
Infant	18				27				23				18			
Control	11	3.44	.637	1.362	15	2.67	.529	1.876**	12	2.79	.862	.420	10	2.84		
Index	7	2.97	.816		12	2.22	.729		11	2.65	.723		8	2.63		
Mother	18				28				21				20			
Control	11	3.87	.646	1.296	15	3.05	.604	1.70***	12	3.84	.414	2.279†	11	3.59		
Index	7	3.34	1.09		13	2.60	.797		9	3.40	.462		9	3.11		

Higher ratings indicate greater intensity of responsiveness.
p<.05.
p=.05.
†p<.01.

DISCUSSION

Unlike many investigations, this study does not use a standardized sample to which a standardized test can be applied. Rather, the home movies that families took during the infancies of their children for their own use are a naturalistic sample of family life. The typical movie scenes (bathing, feeding, holding, etc.) reappear often, although the length and detail of segments vary. There was no attempt to standardize the film segments for the judges in order to avoid biasing the samples through investigation selectivity. I also decided to limit their training to avoid influencing the judges too strongly, which would have created an experimental bias. Consequently, one judge might begin examining a given scene at an earlier point of interaction than another judge, or one judge might omit a scene because he felt it lasted too few seconds to provide an impression. These contingencies explain the relatively low interjudge correlations. If training were extended, if raters were more homogeneous, or if the film samples were more standardized, higher correlations would be anticipated.

Several important findings emerge. For all modalities of infant and maternal behavior there is a trend to lower ratings in the psychotic groups compared with the normal groups. This achieves statistical significance for infants and mothers for holding and also for the mothers for eye gaze and touching. These lower ratings suggest several phenomena in the first 6 months of the infant's life: a dampening of mother-infant interaction in dyads where psychosis eventuates in the child; less attachment of mother to infant and infant to mother in the prepsychotic group; and more avoidance and less reciprocity in interactions in the prepsychotic group when compared with normal mothers and infants. In the series of cases as a whole, the ratings do not distinguish between dampening, decreased attachment, reciprocity and synchronicity, and avoidance and isolation. However, whenever any of these dampening phenomena occur, clinical experience and psychoanalytic and attachment theory predict that the infant is likely to develop pathology.

The most striking finding is that for eye gaze and touching there was no significant differ[illegible] psychotic infants when compared to [illegible] However, the mothers of the prepsyc[illegible] ther initiated less activity with or respo[illegible] avoided more) their infants in these [illegible] suggests that as a group the prepsycho[illegible] ceived a different and less positive 'sin[illegible] than the normal infants, but does not ne[illegible] that this different mothering caused the [illegible] data are consistent with the possibili[illegible] ferent parental behavior may have facil[illegible] gence of psychosis in a constitutional [illegible] infant. Further, one cannot distinguish w[illegible] tial dampened maternal responsiveness [illegible] arisen because a primary apathy in an inf[illegible] mother's responsiveness or whether [illegible] mother profoundly affected a neonate's [illegible]

The results of the general impression[illegible] size the findings of the scale of attachm[illegible] by showing even more notably inadequa[illegible] in the mothers of prepsychotic infants [illegible] mothers ($p<.01$) and no significant differ[illegible] groups of infants.

It is interesting that feeding differ[illegible] emerge for the two groups, since psych[illegible] ry has traditionally focused on the ora[illegible] nal development. These data point to [illegible] gaze and bodily touching as two modal[illegible] aversion and unresponsiveness may oc[illegible] quently and be more crucially traumatic [illegible] feeding interferences. This, of course, d[illegible] the developmental principle that under[illegible] bidinal phase (5, 6)—that the infant's he[illegible] of organismic tension, which is initially e[illegible] a primitive life and death anxiety, must [illegible] that the infant can gradually incorporat[illegible] and ultimately identify with a mother w[illegible] enced as primarily gratifying. Rather, [illegible] phasis, as Bowlby (7) has also done in [illegible] attachment, away from a more unitary co[illegible] ity to a group of behaviors—babbling, [illegible] closeness), eating, and especially eye ga[illegible] ing. Reciprocally, the mother can frustra[illegible] mate the infant's attempts for gratificat[illegible]

these attachment modalities that are instinctually determined for both mother and baby.

The limitation of statistics is that many of the particular findings become "washed out" in looking at the whole series of cases. Individual cases (1, 2) showed how in one family a mother primarily avoided eye gaze, in another family a mother avoided ventral-ventral holding, in several cases parents initiated intense overstimulation across several modalities, interfering with the infant's ability to consummate any attachment behavior, and in other cases the infants were unresponsive to their mothers across several modalities. Patterns emerged in which children were isolated (by dint of parents avoiding them in specific ways such as gaze aversion or of the infant's own apathy) or overstimulated. The overstimulated children were effectively isolated emotionally since their parents were not able to perceive their affective distress and empathize with them. Individual case studies showed clearly that infants in the first 6 months of life had a range of affect that occurred in response to interactions with people and events in their lives, especially their mothers. The babies were not just organisms devoid of an emotional life. The mothers and fathers stimulated babies repeatedly in the first 6 months to expressions of pleasure, distress, depression, and dejection with positive parental actions or parental withdrawal and prohibitions.

Finally, the standard deviations of the ratings of both the scale of attachment indicators and the general clinical impressions reveal that there is considerable overlap between both groups of infants and both groups of mothers. This is consistent with the nonblind examination of the movies. There were gray areas in which some of the control and index group mothers and children looked similar and some in which control group mothers and children looked worse than index families. The extremes, however, were very dissimilar; i.e., the worst parental isolation, overstimulation, and avoidance and the most atonic infants appeared in the index group while the most empathic parenting and the most lively and responsive infants appeared in the control group.

In sum, the use of home movies cannot discern specific patterns of mother-infant coupling in particular cases as can be done in intensive prospective and retrospective individual case studies (1, 2, 8, 9). On the other hand, such case studies lack adequate blind controls. The controlled data from this research support the results of others and point to multiple and multidetermined etiologies for childhood psychosis.

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Psychological Factors in Susceptibility to Drug-Induced Extrapyramidal Symptoms

BY PERITZ LEVINSON, M.D., RICHARD MALEN, M.D., GEORGE HOGBEN, M.D., AND HARRY SMITH, PH.D.

In an effort to identify nonneurobiologic factors in the susceptibility to neuroleptic-induced extrapyramidal symptoms (EPS), the EPS response in 20 patients was correlated with field dependence, self-object differentiation, and premorbid social competence. Only the last proved to be a good predictor, which suggests that psychosocial factors play a role in a patient's EPS response.

AT THE SAME time that use of neuroleptic drugs has greatly increased, there has been a recent and intense interest in extrapyramidal symptoms (EPS), which include parkinsonism, acute dyskinesia, akathisia, and tardive dyskinesia. Clinicians have noted differences in individual susceptibility to extrapyramidal effects of antipsychotic drugs (1, 2). Because it has been noted that the greater the extrapyramidal response to a drug the greater the antipsychotic effect (3), researchers are looking for efficacious drugs without these motoric side effects (4). The minimum research done on individual susceptibility to EPS has been almost entirely neurobiologic (5). In this paper, we will report on our investigation of the possible nonneurobiologic variables in the susceptibility to neuroleptic-induced EPS.

Psychosocial factors contributing to the extrapyramidal response have been suggested by Klein (6), who found that chlorpromazine had a consistent positive effect in reducing psychopathology among premorbidly non-asocial subjects. For the premorbid asocial schizophrenics the results were not as good, possibly because of an extrapyramidal effect. Two psychological factors that have been found to correlate with premorbid social status are field dependence (7) and degree of self-object differentiation (8). Premorbid asocial subjects have been found to be field dependent and to perceive others in a less differentiated way. We decided to investigate the correlation of the extrapyramidal response with these three personality variables—premorbid social competence, field dependence, and self-object differentiation.

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METHOD

Twenty-six consecutively hospita[illegible] treated with chlorpromazine were stud[illegible] hered to the treatment regime. They w[illegible] 55 years of age (mean=30.5±1.89), sch[illegible] cording to the New Haven Checklist (9[illegible] antipsychotic drugs on admission to th[illegible] formed consent was obtained after the [illegible] procedure had been fully explained to [illegible] Chlorpromazine blood levels at the time [illegible] ruled out the presence of drug. At the t[illegible] sion to the project a score on the Adj[illegible] Scale (ARS) (10) for self-object differe[illegible] Embedded Figures Test (EFT) (11) fo[illegible] dence, and the Phillips Scale (PS) (12) i[illegible] social competence was obtained for each [illegible] ing a state of sufficient lucidity. The patie[illegible] started on a schedule of gradually increa[illegible] chlorpromazine. Although the dosage sc[illegible] not be absolutely fixed because of indiv[illegible] needs, there was a strong consistency in [illegible] administration from individual to individ[illegible] ment of drug side effects as well as the [illegible] quirements for sedation on an as-needed [illegible] done through the use of medications tha[illegible] crease the amounts of tranquilizer or supp[illegible] log of the daily dose of chlorpromazine [illegible] each patient throughout the project (r[illegible] mg/day).

The EPS were measured from the onse[illegible] tion via the Bordeleau EPS scale (13) [illegible] week at regular times in the day by the s[illegible] er, who was blind to medication dosage, [illegible] ophrenia, etc. At the end of the project (s[illegible] 4 weeks) the ARS and EFT were repeated [illegible] value (Δ=change in the score) for each [illegible] Greater differentiation is manifested on t[illegible] higher score and on the delta ARS by a g[illegible] value. Greater differentiation on the E[illegible] smaller score and on the delta EFT a l[illegible] value. On the Phillips Scale, the higher t[illegible] poorer the premorbid status.

The data were used for the following pr[illegible] tives: 1) Can psychosocial or psychologic[illegible] data be used to predict the number of EP[illegible] tion of increasing dosage levels during th[illegible] chlorpromazine regimen? If so, which ad[illegible]

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able is most useful in prediction? 2) Is the relative rate of increase in the number of EPS through this treatment regimen predictable by one or more of the admission variables? If so, which admission variable is most correlated with this rate of increase?

The response of each individual to chlorpromazine was assessed using two main statistics for each patient—the average number of EPS and the relative rate of increase in EPS per unit dosage of chlorpromazine. These values were determined by plotting dosage against EPS, fitting a straight line, and deriving a value for this line.

RESULTS

Of the three principal variables, premorbid social competence as measured by the Phillips Scale (12.4±1.94) turned out to be a good predictor of the relative increase in EPS per unit dosage of chlorpromazine ($r=.6481$, $p<.01$). In other words, premorbid asocial schizophrenics had a greater value increase in EPS per unit dosage of chlorpromazine and thereby demonstrated increased susceptibility to these drug-induced motor phenomena.

The negative findings indicated that age, sex, severity of illness (as measured by the New Haven Checklist) and chronicity of illness (as measured by previous hospital admissions) did not correlate well with EPS response in these subjects. Field dependence as determined by the Embedded Figures Test (pretrial=113.02±12.83; posttrial (Δ)=5.73±6.15) also showed little relationship to intensity of EPS. The degree of self-object differentiation as measured by the Adjective Rating Scale (pretrial=10.05±2.02; posttrial (Δ)=−1.85±0.96) showed no demonstrable relationship to the EPS response.

DISCUSSION

Premorbid social status was found to correlate with EPS susceptibility in our group of subjects. However, the fact that field dependence and self-object differentiation did not correlate with EPS response weakens the support for psychological causology and suggests that premorbid adjustment consists of diverse dimensions, which have been recently conceptualized as "bio-psycho-social" in nature (14).

Many physiologic as well as other psychological factors have been advanced to differentiate the good-poor premorbid dimension, but the results have been ambiguous and inconsistent (15). Central nervous system organicity has also been studied, but to date no clear relationship between this factor and premorbid adjustment has emerged (16). A possible exception may be a recent study that found "a group of schizophrenics with premorbid asociality" to have neurologic soft signs (17), but this needs confirmation. Even if the EPS-premorbid status correlation turns out to be due to the psychosocial factor, the epiphenomenal nature of this category must be dealt with. At this point, further research into the nature of the premorbid process is needed. Crucial to success in this area is the development of a standard basic premorbid scale that could be readily "plugged in" to all kinds of biological as well as psychosocial clinical research.

Psychological mechanisms have long been used to explain disorders of movement (e.g., catatonia and conversion hysteria). Muscle tone, body type, and personality type have been correlated with specific EPS responses (18). Nonetheless, at this point in our knowledge, the positive findings in this study may be taken simply as a suggestion that psychological and psychosocial factors may play a significant role in a patient's EPS response.

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Psychological Observations of Bank Robbery

BY DONALD A. JOHNSTON, M.D.

Bank robbery has been only partially examined in the psychiatric literature. It has been publicly considered an act of men of strong will to obtain money. According to the author's observations in a federal penitentiary, bank robbery more often is a symptomatic act with psychological meaning. The author describes several of the unconscious motives and defense aspects of bank robbery in the hope that this will assist in demythologizing the bank robber.

BANK ROBBERIES receive a great deal of publicity. The focus of attention of this publicity is rarely on the criminal's motivation. We hear many details of how the robbery was committed, but the why is often left to "Sutton's law." (When asked why he robbed banks, Willie Sutton replied, "That's where the money is.") The assumption is that bank robbery is an attempt to get rich, or at least to get enough to live on for a while.

I shared these assumptions until 1968, when I was assigned to a federal penitentiary for men in Springfield, Mo., and interviewed many bank robbers. My earlier assumptions and the general public's fantasies about bank robbers were echoed by the inmate population. On the inside, much of the inmate's status is gained from his "criminal occupation." In federal prison bank robbers are up near the top; they are perceived as powerful and daring operators. This occurs despite the fact that in closer relationships within the walls many individual bank robbers are regarded as "punks" or "dummies." A curious split exists—a man might be regarded from a distance as impressive because he has robbed a bank, but on examination he often turns out to be passive and dependent, sometimes ignorant, often physically unattractive, and not infrequently grossly psychotic.

Certain ideas or myths that exist about the bank robber's character and actions came down to us from the 1930s, the era of John Dillinger, Alvin Karpas, Ma Barker, and Bonnie Parker and Clyde Barrow (1). These ideas are promulgated today by movie and television scripts and the drama of news accounts—a shrewd protagonist who fails to remember a significant detail of his plan is captured under fr[illegible] stances, or a vicious criminal blazes his [illegible] of a bank. There are many subplots a[illegible] most of these scripts involve a cunni[illegible] character who wants the money and is [illegible] the known hazards of security devices. [illegible] a high arrest-and-conviction rate, and [illegible] prison sentences (usually 25 years).

As my understanding of bank robb[illegible] crimes became clearer over the two yea[illegible] at the penitentiary I saw that the act of [illegible] bank robbery often had very little, if an[illegible] to the theft of money for personal profit [illegible] lieve that the bank can become an are[illegible] chological pressures are expressed a[illegible] densed action. At times the direct acting [illegible] occurs, while in other instances the robb[illegible] defense against other more overwhelmin[illegible] bing a bank becomes the lesser of two i[illegible]

Homosexuality and narcissistic issue[illegible] tence and inadequacy are particularly [illegible] bank robbery. In some instances the re[illegible] rect attempt to be killed or a self-destruc[illegible] venge. It can be the acting out of tremen[illegible] placed from earlier experiences or the gr[illegible] the wish for sadistic excitement. The imp[illegible] bank robbery is remarkable. In 1967, fe[illegible] 2,200 bank robberies, only 453 were c[illegible] men who knew anything at all about the [illegible] tion of their target bank (2).

I would like to present some of the case[illegible] these observations have been drawn. I [illegible] them into the two broad categories of the [illegible] drives and the defense against drives, [illegible] certainly many overlapping themes.

ACTED-OUT IMPULSES

Case 1. The most vivid of the cases involv[illegible] the impulse to suicide was that of Mr. A, a 44-[illegible] erately successful, self-employed attorney. A[illegible] Catholic, Mr. A considered himself very de[illegible] wife and nine children. He had been having a [illegible] ual affair: he no longer loved his wife, could n[illegible] and feared his affair was about to be found ou[illegible] came depressed and then later feared that Co[illegible] infiltrating the government. Bent on exposing [illegible] wrote long, vague, paranoid letters to newspap[illegible]

On the morning of the bank robbery Mr. A [illegible]

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that something drastic was about to happen. He went to a bank where he was well known and robbed it with a threat backed by an unloaded 45-caliber automatic pistol. After getting the money he lingered several minutes, allowing for the arrival of the police. He aimed his gun at the police and was shot in the arm. He again threatened to fire and was once more wounded in the arm. He then ran and was finally shot in the leg and captured.

After arrest and medical treatment for his wounds Mr. A was sent to the Springfield facility for a sanity evaluation. He was significantly depressed. Of the bank robbery he said, "It seemed like I wanted to get myself bumped off." He stated that suicide was impossible for him because it would nullify his insurance. Further, as a devout Catholic he said he could not commit such an act.

Case 2. In this case there was a strong need to defend against feelings of inadequacy as well as the wish to be killed. Mr. B was the son of a successful physician father and a doting, protective mother. He felt that his father considered him a weakling. He had obtained a degree in journalism, married a greedy and demeaning woman, and was divorced by her after 7 years. He said her tombstone should read, "She wanted more." He felt he could not satisfy her in any way. Following the divorce he became depressed and felt that if he could write a book he would feel better. He was unsuccessful in this desire to write and attempted suicide by ingesting pills.

After a brief hospitalization Mr. B took a bus trip to San Francisco. He had no money with him. In San Francisco he walked the streets looking at the impressive hotels and restaurants where he had been in the past, using his father's money. He walked to the ocean, looked at Alcatraz, and considered suicide by drowning. However, he said, he "did not have the guts."

In front of a bank he met a hippie who asked him for a quarter. This stimulated a resurgence of his sense of failure because he realized he had no money. He told the hippie, "Wait a minute, I'll get some" and immediately headed into the bank. The hippie realized what was about to happen and ran away.

Mr. B saw an armed guard inside the bank, but this did not serve as a deterrent. He got in line at the teller's window and waited. When it was his turn, he attempted to rob the teller with what he described as a "Humphrey Bogart sneer" and his finger in his pocket suggesting that he had a weapon. He heard a click, turned around, and saw the guard's cocked pistol next to his face. He remembered feeling that it was "the biggest thing I had ever seen in my life." At that point, he said, "the fight went out of me," and he nearly fainted. He was convicted of bank robbery and sentenced to a federal penitentiary. He was a very quiet, well-mannered inmate who was aloof to other prisoners.

Case 3. In this case the wish for revenge was prominent. Although the robber exposed himself to risk, the real target of his aggression was his wife. His actions illustrate the dynamics of "victory through defeat." Mr. C was 51 years old, obese, and had ill-fitting dentures. He felt he was a failure financially, socially, and sexually. He had had two marriages to the same woman and had never had any children. Mr. C's wife controlled him and ordered him about constantly. Since it was difficult for him to be directly angry at her, he said he would "fiddle with chemicals in the basement making small explosions which would frighten her." When he became angry enough to threaten divorce, she would put a stop on his checking account. In 1957, in a fit of rage at her, he robbed a neighborhood branch bank with a jar of Wesson Oil. He told the teller it was nitroglycerin. He was well known at the bank and was readily identified, captured, and convicted. His wife was ashamed of him, lost community status by his action, and divorced him.

Eight years later he was released from prison, remarried his wife, and, in 18 months, did it again—the same bank, the same teller's cage, and another jar of Wesson Oil. This time he wore a dashing Tyrolean hat and a very loud plaid sports coat. He was easily recognized on the second try as "the guy who did it before." He was arrested the next day and claimed total amnesia. He kept protesting, "How could I have done it; only a fool would have done it again." He was captured wearing the same clothes he wore in the bank, where he had been clearly photographed by the automatic cameras. In prison he spoke of his act with real pleasure, knowing that he had made his wife miserable again.

Case 4. This man felt driven to avenge his childhood. His robberies were a repetition of displaced anger from early loss of a parent and probably also represented an identification with his father. Mr. D's mother had died in an Army hospital during his childhood, and he blamed the government doctors for "murdering her." By the time he reached his 20s he had evolved a delusional system in which he saw himself as a god. He committed five bank robberies in 2 years, all in the Robin Hood tradition. He would rob the bank and then go immediately to the slums of the city and distribute all the money to skid-row derelicts. He said, "It was terrific to be able to do that." To him it represented giving something important to someone in need. He also believed that the money was really "government money" and that by reclaiming and redistributing it he was doing good deeds for the poor and downtrodden. Perhaps this represented his wish to undo the loss of his mother. The giving away of money to the unfortunate was also a link to his father, who had always given small change to bums, saying, "It's a goddamned shame there are guys like that."

After disposing of all the robbery money Mr. D washed dishes until he saved enough money for an airplane ticket to the next city in Ohio, where he repeated the same acts. The cities of his robberies formed a circle around his father's residence. He never used a weapon, although he threatened bank tellers with the implication that he had one; he believed he would never be shot or harmed during the robberies because "you shouldn't be hurt while doing good deeds." He felt that bystanders in the street were cheering him on while he escaped from the banks.

Case 5. Mr. E's case illustrates how bank robbery discharged sadistic excitement. He was a small, plump, belligerent, 28-year-old from rural Georgia who had been raised in an orphanage. He was a sadomasochistic homosexual. "I like," he said, "to screw boys in the ass and bite them." He also liked "for boys to bite me before sex. If you don't have pain, you don't get anything out of it." He disliked women and enjoyed frightening them. A bank with women tellers and customers became a setting for his acting out. On the day of the robbery he took one of his young male lovers with him into the bank and screamed, "All right, this is it!" and emptied an automatic pistol across the ceiling. The noise, chaos, and terror of the women was transiently gratifying. He was apprehended before leaving the bank. In prison he said of his crime, "I was going to be somebody. It was fun and they would know my name."

DEFENSES AGAINST DRIVES

The following three cases fall under the general heading of defenses against drives. They are typical of many similar instances in which deviant sexuality or feelings of failure and inadequacy were significant features of bank robbery motivation.

Case 6. At age 31, Mr. F, a single man, robbed two banks in 3 days. He was employed as a machinist and had no previous criminal history. Several days before the robberies, Mr. F underwent an acute psychotic decompensation with referential thinking and feeling sexually stimulated. He had the delusion that his brother's posture in the machine shop where they both worked indicated they should have homosexual intercourse. He began to receive messages from television sets, restaurant waiters, and police officers that he should rob a bank.

Feeling it was "something I must do," Mr. F walked into a suburban branch bank with a note stating he had a weapon. He had never seen the bank before; he selected it because it was on a street that began with an O, and a woman he knew had a name that began with O. He hid the $1,300 proceeds of the robbery in his apartment. Two days afterward he felt driven to commit a second robbery and was arrested in the bank.

The youngest of six children, Mr. F had been raised in a middle-class family in South America. He was close to his religious mother and was treated as the baby of the family. At age 10 he began hearing voices that intermittently told him how to act. His sexual identity was tenuous. As an adolescent he compulsively masturbated and left his ejaculate in the hope it would be discovered by his mother or sisters so "they would know I was a man." His rare heterosexual experiences had been with prostitutes in meetings arranged by men who felt he should lose his virginity. Adult sexual gratification was through occasional but extremely conflicted homosexual encounters. Mr. F said that his bank robberies were "the most masculine thing in my life" and was very pleased with himself for having committed them.

Case 7. Mr. G exemplifies a situation of narcissistic inadequacy defended by impulsive, grandiose action. He was 30 years old when he, like Mr. B, robbed a bank in San Francisco to give a beggar a quarter. The fact that he had to admit that he had no money when he was panhandled was such an internal defeat that he felt compelled to walk into the nearest bank and rob it. As he confronted the teller, he remembered thinking, "I could do anything; it was my moment of destiny. I really felt bad, seeing the teller so scared. I didn't want to hurt him or scare him too much, but I told him that I had a bomb in my pocket—nitroglycerin." He was given $3,000 and, frightened by what he had done, caught a plane to Hawaii. Once there, he immediately turned himself in and returned the remaining money.

As a child Mr. G had been deserted by his mother and raised in an orphanage called the New England Home for Little Wanderers. He had had a close relationship with his grandfather, a kindly Italian barber who died at about the same time he was abandoned. He felt that no matter how hard he tried, he could never be as good as his grandfather. When he left the orphanage he wandered, living out the orphanage's name. He was never without his teddy bear and transistor radio. He shoplifted candy and s... animals. He said these were "presents to my ...

Case 8. Mr. H impulsively robbed a bank ... ter he heard the news of Neil Armstrong's ... moon. Mr. H felt he had never accomplish... lived by odd jobs. While passing out handbi... radio commentator describing the moon la... phrase "a giant step for mankind." Mr. H w... by how small and insignificant he felt. ... robbed a nearby bank with a note. He felt ... tence was worth it: "I really did something bi...

DISCUSSION

These eight men and their acts pro... understanding of bank robbery. It is imp... several points about the selection of the... cases. Each category was represented by ... custody; in fact, after a time, I found my... for the "normal" bank robbers, those so ... by Sutton's law. I found few. My sam... came from a federal evaluation and tre... and, obviously, did not include any su... robbers who had avoided conviction. I w... that I came in contact with more than 200 ... in a 2-year period.

I do not want to underestimate the se... and inadequacy of this population of ... fenders. Many of the men had chaotic and ... riences in childhood and adolescence. Fe... vere psychosis rendered the accused ... competent to stand trial. Of the men con... had little real sense of the number of yea... serving a bank robbery sentence. For so ... structure of prison life seemed to be a ca...

Above all, it became clear that bank r... act that is more complicated than a sim... getting money. It is decidedly a part o... life and an attempt to resolve conflict. C... bank robbers are dangerous men who re... tion from society. However, many others ... sentences for acts that were meant to ... selves. These men exchange 20 to 25 ... lives for behavior intended to restore ... psychic equilibrium.

It is remarkable that the mythology of ... continues to persist in the face of o... tradictions. Bank robbery is easy. Banks ... accessible, and can be robbed on impulse ... in a coat pocket. The myths prevent a ca... tion of these criminal offenders and ... flexible decisions on treatment and incarc...

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A Marketplace Model of Consultation Psychiatry in the General Hospital

BY FREDERICK G. GUGGENHEIM, M.D.

The manner in which internists and surgeons view potential psychiatric consultations is analogous to the way in which consumers evaluate a service or product in the marketplace. Factors that determine a purchase or a consultation request include 1) perception of need, 2) prior attitude and experience, 3) projected image, 4) availability, and 5) cost/benefit ratio. The author presents a model of psychiatric consultation, with similarities between research and development, manufacturing and merchandising phases on the one hand, and negotiation, investigation, and implementation phases on the other.

OUR UNDERSTANDING of the interactions of the psychiatric consultant in the general hospital has evolved considerably over the past two decades. At first, the consultation process was conceptualized as primarily a dyadic relationship (1, 2). Then it was seen as a pluralistic one (3–5). More recently, the consultation process has been represented by a general systems model (6, 7). The general systems approach, currently used in teaching residents about the complexities of consultation work, has been helpful in explaining the multifaceted interactions of the consultant with patients and their families and employers, members of the health care team, and the physician who has requested the consultation (termed the consultee in this paper).

General systems theory has permitted an understanding of the consultant's direct and indirect interactions with the patient and his milieu. Unfortunately, the theory as presented to date has not taken into account complexities of the general hospital as a sociological structure with powerful opinion-swinging constituencies that can accept or reject the consultant. It has also failed to focus on the consultant's roles as good will ambassador and salesman (in addition to his usual roles as physician, psychiatrist, and psychotherapist). Finally, general systems theory does not present forcefully the concept that psychiatric consultations can provide a substantial benefit at a definable cost.

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Expanding on a general systems approach, I would like to present a marketplace model as an aid in explaining certain aspects of the consultant's interactions in the general hospital. Since the selling of a service is involved, the general hospital can be seen as the consultant's marketplace, the consultation as the consultant's service or product, and the consultee as the purchaser. Viewed in this way, certain rules and roles can be carefully examined.

For purposes of comparison, one can conceptualize the introduction of a new product to the marketplace or a service in the general hospital as having three separate but coordinate phases: 1) research and development, 2) manufacturing, and 3) merchandizing, with purchasing, consumption, and renewed demand. In merchandizing, advertising and good will are important factors. Image is a central issue; the appearance of substantial worth is as important a determinant of the business venture as the reality of its worth.

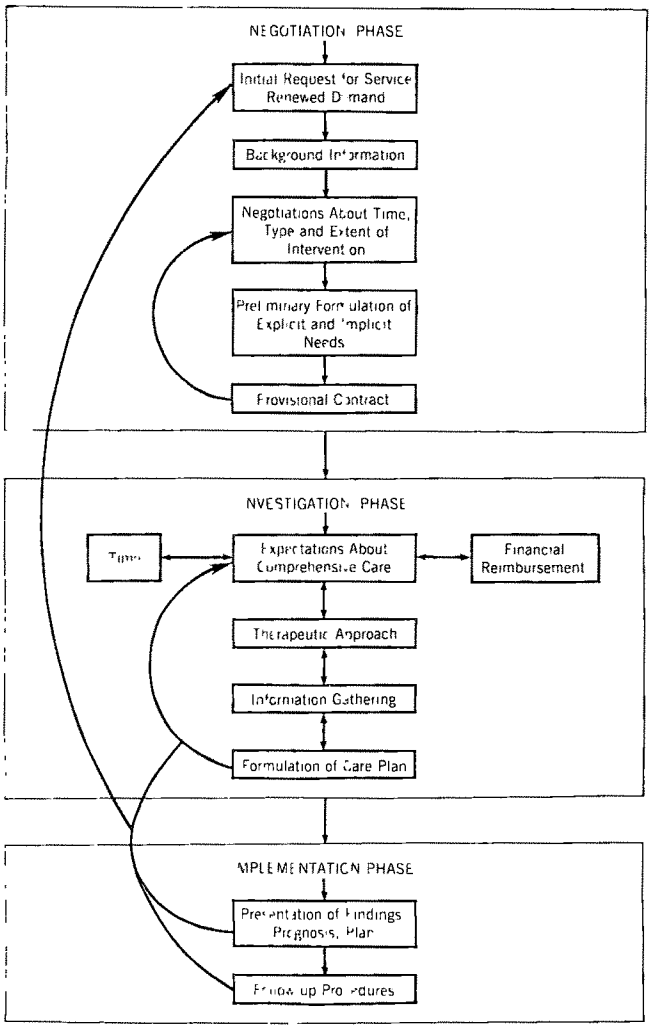
In consultation work, the three separate but coordinate phases are negotiation, investigation, and implementation. Figure 1 depicts the marketplace model as it applies to these three phases of the consultation process. In the negotiation phase, the initial request for service leads to a marketing survey and a provisional contractual agreement: Before agreeing to the consultation, the consultant needs to know how emergent the clinical situation is, what the problem is, and how the patient feels about being seen. Finding out that a patient is agitated, threatening others in the hall with her cane, limits the need to ask how she feels about being seen by a psychiatrist. If she needs to be seen instantly and the consultant has no available time for the next three hours, the consultee's expectations are a moot point. After negotiations about specifics of the intervention, a preliminary formulation of explicit and implicit needs leads to a provisional contract.

The investigation phase involves dynamic interaction among consultant, consultee, patient, family, nursing staff, and, occasionally, employer or sponsoring agency. The consultant's enthusiasm, perceptiveness, skill in interpersonal relationships, and technical skills, manifested by a positive therapeutic approach, greatly influence the "yield." Reactions to the consultant will influence staff availability in information gathering and care plan formulation.

Two other factors that influence the success of this phase are time and money. Although they are rarely discussed in the literature, they are of crucial impor-

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FIGURE 1
Marketplace Analogy for Consultation Psychiatry



tance. The effectiveness of the best consultant will be diminished if his available time is severely limited. In practice, those consultants who squeeze consulting time out of a packed schedule rarely perform consultation that is up to their level of capability. Financial reimbursement also influences the quality and volume of work produced by the consultant. The consultant retained on a salary often engages in staff education, mediation of staff-staff conflicts, and patient-oriented preventive mental health measures. However, if the consultant is merely called in on specific cases and his anticipated payment is scanty and inconsistent, he may not deal with certain relevant, evident problems that directly or indirectly impinge on care. Factors like time and money determine whether the consultation process involves secondary and tertiary preventive measures or primary prevention.

Following the information-gathering phase, the consultant formulates with the staff a realistic care plan that is oriented toward the pathological [illegible] ing the patient, a diagnostic/descriptive [illegible] the patient's illness, i.e., his psychosoci[illegible] that disease, a dynamic/interpretive pr[illegible] consultant's understanding of the patie[illegible] and behavior allows for a comprehe[illegible] that includes concrete suggestions abo[illegible] behaviors for the staff and medication[illegible] for the patient (9).

The implementation phase of the co[illegible] ess is analogous to the merchandising [illegible] ness. Manufacturing is important, and [illegible] tant's gathering of information and form[illegible] namics. The crucial matter, however [illegible] tee's accepting and then implementing [illegible] recommendations. Far too often the [illegible] consultant underestimates the impo[illegible] plementation phase. Thus, he places litt[illegible] selling his suggestions to the consultee [illegible] try to overcome the mild suspicion wit[illegible] tees often regard consultations of all s[illegible]

In addition to selling his suggestion[illegible] needs to be certain that his goods are [illegible] packaged. New facts, and old ones re[illegible] be woven together into a product that is [illegible] the consultee. The consultant presents [illegible] pathology of the patient in light of the [illegible] experiences of the consultee. Some [illegible] open and curious consultees want a c[illegible] dynamic exposition of the family co[illegible] how it has played a role in the product[illegible] tional behavior. Other consultees, for [illegible] sonal and professional reasons, merely [illegible] list of suggestions about what to do wi[illegible] patient over the next few days.

The care plan must be adapted to the [illegible] providers in order to permit useful [illegible] rather than exasperated frustration. [illegible] follow-up support and prognosis should [illegible] vided. Proper attention to the implem[illegible] leads to mediation of disputes, easier co[illegible] better cooperation (10), and renewed [illegible] service.

Image is central to these phases. As [illegible] place, the projected capacity for a th[illegible] proach is as important to the process of [illegible] is the consultant's actual capacity for [illegible] gagement. Visibility, dependability, cred[illegible] cality, and accountability are factors tha[illegible] consultant's image. Also referred to as [illegible] demeanor" and "bedside manner," image [illegible] tant element in clinical practice and can [illegible] tential consultee's decision whether or [illegible] consultation. Reactions to the consultan[illegible] al demeanor influence staff availability [illegible] gathering and staff participation in the fe[illegible] implementation of a care plan. Staff [illegible] work enthusiastically to implement the s[illegible] a consultant who smoothes patient-staf[illegible] contrast, they often fail to follow the adv[illegible]

tants who interact with only the patient and chart or who use psychoanalytic jargon.

THE PURCHASE

The consumer's decision about buying the service or product is the crucial part of the complex chain of events in the marketplace. The buyer may have heard good things about the product and may have used it. He may have obtained funds for the purchase. Is the product available when the buyer wants it? To translate this to consultation work, Is the consultant both physically and emotionally receptive to the consultee's request? The consultant can choose the rules for his role, limiting or facilitating access, erecting dense or subtle barriers. These rules often arise from countertransference, the consultant's feelings regarding patients, staff, consultees, and institutions. Inexperienced consultants often are unaware of the ways they "turn off" staff.

A variety of factors influence consumer resistance and attraction toward the purchase. Neither the consultant nor the manufacturer has control over certain determining factors in a potential consumer or consultee, such as prior attitudes and experiences. Moreover, leaders' opinions can influence a potential purchase. If the chief of a medical service belittles the value of consultation, the proportion of staff seeking consultations for anything but "psychiatric disasters" may be rather small.

Cost of the consultation is another factor that influences the purchase decision. Generally, the cost to the consultee is small. He mentions to the patient that a psychiatrist will be stopping by to talk about problems, coping, pain, grief, nerves. However, if the referral is poorly timed or clumsily executed by the consultee, the cost may be an angry attack from a patient who feels punished or rejected. Making matters worse, some inexperienced consultants unfortunately do antagonize their patients with stressful interviews, seemingly irrelevant questions, and premature interpretations.

Some habitual nonconsultees never purchase psychiatric consultation because "it only stirs the patient up." Here the nonconsultees' fears of cost or of the unknown (e.g., dismissal from the case or displacement in important managerial decisions) (11) are important. Other habitual nonconsultees never even perceive a bona fide need. Surgical specialists, for example, often see their care as segmental, not comprehensive. They tend to seek psychiatric consultations infrequently (10); when they do, the patient is often a psychiatric disaster, with full-blown delirium tremens, homosexual panic, or severe suicidal ideation.

Although the consultant cannot control the effects of prior attitudes, experiences, and perceptions, he does have considerable input into his image. Public relations based on visibility, dependability, accountability, interest, and practicality can do much to swing potential users toward requesting a consultant's services at appropriate times.

OUTCOME EVALUATIONS

How can the vendor find out about the quality of his work? Industry typically spends 5% of its budget on research and development, including polls of consumer trends and satisfaction. The presence of objective measures and quantifiable outcomes aids both the precision and accuracy of the survey and permits perception of "blind spots."

In industry and intensive individual psychotherapy paid for by the patient, there is but one knowledgeable critic: the individual consumer. In consultation psychiatry, there are a number of critical consumers: the patient, the consultee, the family, the staff, and the institution.

Measurable outcome for the patient, in addition to satisfaction, might include decreases in anguish, overutilization of medical facilities for psychogenic problems, psychogenic invalidism, and noncompliance with medical regimen (12).

For the consultee, the immediate clinical impact of the consultation outcome might be an increased level of awareness and understanding of relevant psychosocial factors. This in itself is difficult to quantitate. However, a derivative behavior might also ensue—increased facilitation in the identification of problems amenable to specific intervention. This transformation might then lead to such measurable change as an increase in the referral rate, along with earlier detection of problems and greater implementation of the consultant's suggestions.

For the staff, effective consultation work might lead to a decrease in staff turnover and perhaps to improved morale and higher levels of comprehensive care.

Another important outcome question is how much profit is being generated. In an era of scarce funds and a need for continual examination of cost-benefit ratios, this is a crucial question. Improvement of comprehensive care is the goal of consultation work. In this respect, it differs from industry, where the goal is monetary profit. In the case of a university consultation service, "profits" can be examined in terms of 1) improved comprehensive care, which is hard to quantitate; 2) teaching, which is easier to assess in terms of number and types of conferences plus student critiques and performance; and 3) research, which is also easier to measure in terms of quantity and quality of original and scholarly contributions.

THE BOTTOM LINE

Examination of the marketing process entails a step-by-step review of each phase, with objective descriptions of corporate strengths and weaknesses. The sim-

plest way to do a "quick and easy" quantitative, quasi-qualitative overview of any business is to inspect the bottom line—how much profit is being generated, how much work is being done?

The referral rates of consultation and liaison services from various medical centers have been published. Lipowski (10) reported a range of 4%–13% of all admissions, with an average of 9%, but some figures from academic settings are as low as 2.2% (13). British studies cite referral rates of 0.7% and 1.34% (14, 15). Although Lipowski (10) stated that American consultation services have higher rates of referral than European services, he is unclear on the reason for this. Since some of these figures group wards with liaison activity, they need to be looked at with a critical eye. The referral rate on a liaison service may be 35% (16), while that for a private psychiatric consultation service in the same hospital without a liaison service runs about 4% (17). Hospital-wide psychiatric consultation rates are a complex matter, reflecting, among other things, availability and adaptability of manpower, image, and departmental-institutional goals and priorities.

Although it plays a pivotal role in the commercial marketplace, formal quality control inspection is often lacking in consultation work. The consultant's lack of meaningful feedback deprives him of an important stimulus to learning. Therefore, a quantitative measure (the number of consultations requested) is usually the only quality control measure built into consultation work, although qualitative criteria could possibly be applied. For example, is the consultant called in appropriately early? Are his opinions valued and suggestions carried out? Does his intervention appear to improve comprehensive care?

CONCLUSIONS

A marketplace model points out the interactive and interdependent nature of consultation work. Insufficient attention has been paid to the implementation phase of consultation work. Inexperienced consultants often attend to information-gathering r[illegible] total consultation process.

Despite its demonstrated benefit to c[illegible] care and its relatively minuscule cost [illegible] other health and mental health care acti[illegible] tation psychiatry continues to be under[illegible] dizing the activities of a viable and use[illegible] patient care.

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A Look at Psychiatric Decision Making

BY BRUCE ROBERTS, M.D.

The author reviews the efforts of medicine and psychiatry to understand their decision-making processes and notes the lack of psychiatric literature in this area. The decision table, a sophisticated form of algorithm, is introduced as a structure to help explain the decision-making process. This tool is applied to the evaluation and biological treatment of depression. Decision tables can be used as an educational tool, a method to facilitate the delivery of mental health care, a method to monitor the quality of treatment, and as a research tool.

IN POSTGRADUATE training and particularly medical school, psychiatry and the other medical specialties have typically emphasized the acquisition of bodies of knowledge as opposed to an understanding of the decision-making process. Specific factual data obtained from lectures, books, and journals are important, but what seems to be at least as important, or more important, is the ability of the psychiatrist (physician) to use these diverse data for making appropriate decisions. A systematic explanation of logic for good decision making is presently not being taught. I propose that decision tables may help us to better understand how we make decisions.

In the past decade there has been an increasing interest in objectifying clinical judgment. Gauron and Dickinson (1, 2) investigated how 12 psychiatrists arrived at clinical conclusions in 40 hospital cases. They found 5 basically different diagnostic approaches.

1. Some psychiatrists committed themselves to a diagnosis on an intuitive basis early in the process and then challenged the rest of the information to disprove this diagnosis.
2. Some psychiatrists could not narrow the number of possible diagnoses and made an impulsive, arbitrary decision to choose one diagnosis.
3. Some psychiatrists considered every possible diagnosis with equal weight and ruled out each one systematically.
4. Some psychiatrists rigidly followed a formal structure, i.e., a textbook, and requested a tremendous amount of data before diagnoses were ruled out.
5. Some psychiatrists went where the information led them and had no preconceived notions of the diagnosis.

Some work in statistical decision theory (3–5) and the use of algorithms (4, 6) has further expanded the application of decision analysis to medicine and psychiatry. With this work has come a tremendous interest in the organization (7) and computerization of psychiatric and medical data (4, 7–18). In the past five years, particularly in the past two years, there has been a great deal written in medical journals on the application of decision analysis to medical problems (19–31) and relatively little written about this in the psychiatric literature.

A new technique, decision tables, has recently beeen introduced in the medical literature by Holland (32). The application of this technique to psychiatry is the substance of this paper.

DECISION TABLES

Decision tables were developed in the late 1950s by the General Electric and Sutherland companies to replace algorithms that could not handle the complexities of their manufacturing processes. Since their development, the literature has been full of stories of how decision tables succeeded where older techniques, i.e., algorithms, failed (32, 33). Decision tables compactly represent a great deal of data, logically linking the variables (conditions) and outputs (actions), and they are commonly used in business and government.

To illustrate the application of decision tables, I shall transfer the logic contained in the following ancient Persian poem (32) into a decision table:

He who knows not and knows not that he knows not is a fool—shun him.
He who knows not and knows that he knows not can be taught—teach him.
He who knows and knows not that he knows is asleep—wake him.
He who knows and knows that he knows is a prophet—follow him.

This poem can be written in the decision table

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FIGURE 1
Decision Table for Ancient Persian Poem*

CONDITION	1	He knows	Y		N	
	2	He knows that he knows not	—		Y	N
	3	He knows that he knows	Y	N	—	—
ACTION	1	Shun him				X
	2	Teach him			X	
	3	Wake him		X		
	4	Follow him	X			

*All items above the double lines are the conditions. Y indicates that the condition is present, and N indicates that the condition is not present. A condition that is not needed or is irrelevant to the particular pathway is represented by a horizontal line. All of the items below the double line are the actions (outputs). X indicates that the action to the left of the X is to be initiated. This illustration is based on material from Holland (32).

shown in figure 1. In figure 1 all of the items above the double line are the conditions. If the condition is present it is represented by a "Y" to the right of the condition. If the condition is not present "N" is the correct choice. A condition that is not needed or is irrelevant to the particular pathway is represented by a "—" to the right of the condition. All of the items below the horizontal double line are the actions (outputs). In the area below the double line "X" indicates that the action to the left of the "X" is to be initiated.

As an illustration, let us assume that we want to know the outcome of "He who knows and knows not that he knows." Choose "Y" for condition 1, "He knows." Condition 2 is immaterial under "Y." Proceed to condition 3, "He knows that he knows." For condition 3 we want the negative of "He knows that he knows," and we therefore choose "N." Drop a vertical line under "N" in condition 3, and the "X" under the horizontal double line is the output, "Wake him," action 3.

DECISION TABLE FOR THE EVALUATION OF THE CHIEF COMPLAINT OF "FEELING BAD"

To illustrate the use of decision tables to facilitate objectifying the diagnostic process, let us look at the problem of a patient with the chief complaint of "feeling bad." The novice may feel overwhelmed by the various options he/she may pursue for this chief complaint. The novice must learn what decision to make and assign priorities to these decisions. The experienced psychiatrist will more efficiently recognize (it may not be conscious) the priority of decisions he or she must make without prematurely closing off hypotheses. The decision table shown in figure 2 attempts to objectify this process. I do not propose that it represents the only or best way to evaluate the chief complaint "feeling bad." However, it will facilitate understanding the *process* (not the conte[illegible] think.

The decision table in figure 2 propose[illegible] decision which should be made is to de[illegible] er the chief complaint of "feeling bad" [illegible] tation of depressed mood. If the patien[illegible] is anxious when he says he is "feeling [illegible] 1 is "N." The appropriate action for [illegible] tient is determined by dropping a line [illegible] condition 1. Conditions 2–7 are imma[illegible] vertical line would intercept "X" at ac[illegible] not the manifestation 'depressed mood[illegible] festation, i.e., anxiety, fear, etc., nee[illegible] uated in another decision table." Thus [illegible] sumption that this is a depressed mood[illegible] one is directed to another decision table [illegible] fined in this text, i.e., in actions 1–8, on[illegible] be directed to reenter this decision table[illegible]

Assuming that we are dealing with the [illegible] depressed mood, we see that condit[illegible] lowing the vertical line in figure 2 we pro[illegible] tion 2 and need to make a decision to de[illegible] er the patient is an immedite danger to h[illegible] ers. It seems judicious that this deci[illegible] before considering the possibility of a bi[illegible] ment. Let us also assume that the pati[illegible] ideas but is not an immediate danger to i[illegible] ers—thus condition 2 is "N"; that there [illegible] loss of a major love object—thus condit[illegible] making condition 4 immaterial; that th[illegible] dence of psychotic behavior or diagno[illegible] phrenia—thus condition 5 is "N"; that a[illegible] organic etiologies have been ruled ou[illegible] nist—thus condition 6 is "Y"; and that t[illegible] never been successfully treated for this p[illegible] past—thus condition 7 is "N." This pro[illegible] in figure 2 by the vertical line. This line [illegible] "X" at actions 7 and 8.

The reader may ask, "How do you esta[illegible] patient is an immediate danger to himse[illegible] or "How do you determine if the 'depre[illegible] part of a normal grief reaction?" Deci[illegible] facilitate asking these questions. Organi[illegible] tion around relevant decisions gives a n[illegible] to factual data. Each condition (1–8) an[illegible] may require a decision table of its ow[illegible] beyond the scope of this paper.[1]

Despite what may appear to be a rigid [illegible] decision table should be used as a tool fo[illegible] trist and should not interfere with the ps[illegible] tient relationship. The use of a mechan[illegible] psychiatrist's thinking does not mean th[illegible] ous verbal information from the patient i[illegible]

Because the psychiatrist has used the [illegible] in figure 2, he or she is assured that he has [illegible]

[1]A decision table is available from the author on req[illegible] in figure 2. This decision table defines a model to de[illegible] er a biological intervention is indicated, and if it is [illegible] the intervention should be.

FIGURE 2
Decision Table for the Evaluation of the Chief Complaint "Feeling Bad"*

CONDITION	1	Verify that the chief complaint "feeling bad" is really the manifestation "depressed mood."	Y									N
	2	Is patient an immediate danger to himself or others?	N								Y	—
	3	Is there a recent loss of a *major* love object?	Y				N				—	—
	4	Is this part of a normal grief reaction?	N			Y	—				—	—
	5	Is there evidence of psychotic behavior or diagnosis of schizophrenia?	N		Y	—	N			Y	—	—
	6	Have all appropriate organic etiologies been ruled out?	Y		N	—	—	Y	N	—	—	—
	7	Has the patient been successfully treated for this problem in the past?	Y	N	—	—	—	Y	N	—	—	—	—
ACTION	1	This is not the manifestation "depressed mood." This manifestation, i.e., anxiety, fear, etc., needs to be evaluated in another decision table.										X
	2	Make a management decision to prevent an immediate suicide or homicide attempt.									X	
	3	This patient may need support, but does not need any major psychiatric intervention.					X					
	4	The patient's depressed mood may be a manifestation of a psychotic illness. This requires more information about the nature of the psychotic illness before a treatment decision can be made.				X				X		
	5	The patient's depressed mood may, in whole or part, be a manifestation of an organic illness. Rule out all appropriate organic etiologies for this age group.			X				X			
	6	Integrate this information into the evaluation. You may reenter this table at action 7 if you are not sure of treatment plan.	X					X				
	7	Now determine if patient will benefit from a biological treatment for "depressed mood." Go to biological treatment of depression decision table.		X					X			
	8	Evaluate for appropriate psychotherapy.		X					X			

*See figure 1 for an explanation of the symbols used here.

responsibilities. He knows that he has a patient with a depressed mood, whom he has evaluated for immediate suicide and homicide potential, for a normal grief reaction, psychotic behavior, and possible contributing organic factors. If the psychiatrist has used the decision table and has kept good records, he can justify his logic both to the patient and the peer review committee.

CONCLUSIONS

Decision tables may be used as an educational tool, a method to facilitate the delivery of mental health care, a method to monitor the quality of treatment, and as a research tool.

Decision tables focus on the process of integrating diverse information for making appropriate decisions. This is in contrast to the present method of trying to teach medical students all of the data and leaving it to them to integrate these data to solve problems. In our present system, it is hoped that when a medical student sees a patient and is confronted with the patient's problems, he or she will somehow remember the relevant data and know how to apply it to evaluate, define, and solve the presenting problem. We know that this does not happen; what is closer to real life is that medical students learn clinical judgment by their own devices. Some learn it better than others. Time is constant (4 years), and quality becomes the variable. Since the core curriculum focuses on the regurgitation of facts, this attitude still pervades in the clinics. The emphasis on regurgitation of facts is out of proportion to the emphasis on decision making.

When used appropriately and not overzealously, decision tables can facilitate mental health care. They are efficient in defining a relevant data base. The process of obtaining the data is a dynamic one, the variable being the answer to the previous question. Decision tables facilitate logical thinking and help us define what we know and do not know. They minimize the pretentious illusion of knowledge when we do not have the data.

Keeping a good record of the decision-making process enables the psychiatrist to document his or her logic. This creates a mechanism for the psychiatrist to be accountable to the patient, the peer review committee, and himself.

Decision tables can be useful tools in psychiatry as a mechanism to expose and explore how we think.

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Videotape Recording of Dynamic Psychotherapy: Supervisory Tool or Hindrance?

BY CLAUDE T. H. FRIEDMANN, M.D., JOE YAMAMOTO, M.D., GEORGE H. WOLKON, PH.D., AND LYNDA DAVIS, M.A.

The authors questioned residents and supervisors at a large clinic about their attitudes and experiences regarding clinical supervision. Half of the residents then videotaped one of their psychotherapy sessions and presented the tape in their next supervisory session. The procedure had little effect on patients, was anxiety-provoking for the residents, and had some diagnostic benefit for the supervisors. After the taping, the residents' views about taping were less positive than before the taping, but the supervisors' views did not change significantly.

THE TEACHING of dynamic psychotherapy in the United States relies heavily on secondhand reporting of clinical material by a resident to a supervisor. This methodology, devised by Eitington (1) for the Berlin Psychoanalytic Institute, is considered a cornerstone of psychiatric education. It departs in several ways from the medical model of direct patient examination by the attending physician: 1) it assumes that the reporter will give a reliable account and that the supervisor will be able to recognize gaps and errors, 2) the therapeutic process as reported over time is assumed to be more important than a detailed presentation of each hour (2), and 3) the resident's relationship with the supervisor is considered very important and the patient is not per se the focus of the discussion. Fleming and Benedek (3) consider supervision to be parallel to psychoanalysis.

The nature and goals of supervision are in debate. Tarachow (4) disagrees with Fleming, stating that supervision should be patient centered. Sloane (5) would not deal with countertransference issues, but Goin and Kline (6) encourage it. Some would concentrate on supervision as a growth experience and stress residents' own intrapsychic dynamics, whereas others prefer to focus on diagnosis, therapeutic techniques, and the patient.

Gruenberg and associates (7) point out that traditional supervision, regardless of goals, is subject to conscious and unconscious editing and misperceptions and that the therapist is often unaware of his or her nonverbal communications. Gruenberg proposes videotaping sessions to improve residency education. Grotjahn (8) also voices misgivings about traditional supervision, as do Schuster and associates (9). When Schuster saw puzzling patients "live," he often received a very different impression than previous reporting had given him. Stein and associates (10) related bias in the initial interviews of patients after comparing secondhand supervision with live observation. Buehler (11) also found discrepancies between written reports and direct observation.

In the 1950s the Conference on Psychiatric Education (12) held that audiovisual media have little place in psychiatric education, and Eckstein and Wallerstein (2) agreed regarding dynamic psychotherapy. When Kubie (13) suggested tape recording analytic sessions he met great resistance, including a feeling that the study of supervision was inappropriate. Questions have been raised about the effects of videotapes, one-way mirrors, audiotapes, and direct observation of the therapist and patient, as well as fear of artifactual interference with the therapeutic process. Others see these tools as fostering passivity (14) and diminishing the necessity for case formulations by trainees (15).

In this paper we will address several of these unanswered questions. We queried psychiatric residents and their supervisors about the nature and goals of supervision and their feelings about videotaping for supervision and attempted to gauge the effect of the videotaping procedure on all the participants. We will submit some preliminary data regarding the effect of videotaping a session of continuing, traditionally supervised psychotherapy for supervision. To our knowledge, this is the first attempt to systematically assess the value of videotapes in continuing supervision versus secondhand reporting. Although we will

At the time this work was done, Dr. Friedmann was a third-year resident and Dr. Yamamoto was Professor and Director, Adult Outpatient Clinic, Department of Psychiatry, University of Southern California, Los Angeles, Calif., where Dr. Wolkon is Associate Professor of Psychiatry (Psychology), and Ms. Davis is Research Assistant. Dr. Friedmann is now Assistant Professor-in-Residence, and Dr. Yamamoto is Professor of Psychiatry, Department of Psychiatry, University of California, Los Angeles. Dr. Friedmann is also Chief, Adult Psychiatry Crisis and Outpatient Departments, Harbor General Hospital, Torrance, Calif. Address reprint requests to him at Harbor General Hospital, D-5, Psychiatry, 1000 W. Carson St., Torrance, Calif. 90509.

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not provide the definitive answer of whether or not a picture is worth a thousand words, we hope to provide a sound basis for future scientific study.

METHOD

All of the clinical supervisors and second-year psychiatric residents in the Adult Psychiatry Outpatient Clinic of the University of Southern California-Los Angeles County Hospital were asked to complete a questionnaire about their clinical supervision. Most of the supervisors were private practitioners in the community, but a few were full-time faculty. The majority had a dynamic-analytic orientation. Second-year residents were working full-time in the outpatient clinic for the entire year, spending over 100 hours a month seeing patients. Our questionnaire contained a rating scale to assess experience with various modes of supervision, the supervisors' and residents' perception of the goals of supervision, and their opinions regarding the potential impact of videotaping one session of psychotherapy currently under traditional supervision.

Next, all of the residents were asked to videotape such a session and to use the tape during the next supervisory session. Permission from the patient and supervisor were required. Half of the residents, 11, volunteered. The taping residents and their supervisors were required to complete a modified Walter Reed Mental Status Evaluation (16) for their patient before taping and after viewing the tape. After viewing, they completed a questionnaire about how they felt the taping affected the patients, themselves, and the supervision.

The videotaping was performed by volunteers who used portable equipment set up in the therapist's office. The viewing was done in another, specially equipped room. The supervisors and residents answered the questionnaires in private and without consulting each other. The patients completed a short questionnaire after the taping that indicated their feelings about the session.

RESULTS

Previous Experience with Videotapes

We obtained responses from 21 of the 22 residents (95%) and 32 of the 37 supervisors (86%). On the average, each resident had worked with 50 outpatients and each supervisor with 50 residents. Only 16% of the residents and 40% of the supervisors had previously used videotapes in the supervision of psychotherapy. None used them regularly, and each used traditional supervision at least 85% of the time. There was no significant difference in previous experience between the 11 residents and 9 supervisors who subsequently videotaped a therapy session for this study and the 10 residents and 23 supervisors who did not. There were 2 supervisors who supervised 2 participating residents each, hence the discrepancy between the number of supervisors and 11 residents.

Opinions About the Goals of Supervision

The items considered by our respondents as "very much" goals of supervising psychodynamic psychotherapy were teaching psychodynamics, improving therapeutic techniques, improving patient care, and helping residents grow. The remaining 4 items were rated as "somewhat" important goals: giving emotional support, teaching psychiatric theory, teaching diagnostic techniques, and getting residents in touch with their own feelings. No categories were rated as "little" or "no" importance. There was a high degree of agreement across all 4 groups—residents who participated in a videotaped session and those who did not, and supervisors who participated and those who did not.

Attitudes Toward Videotaping

Before videotaping. The supervisors who were involved in videotaping and the residents with whom they showed significantly stronger agreement than the supervisors who were not and the residents with whom they agreed with the statement that videotaping would interfere with the patient's production of material ($t=2.$ $p<.01$, and $t=1.88$, $p<.05$, respectively). Residents in the taped session, although all 4 groups of residents agreed with the statement that taping would interfere with the resident's ability to conduct psychotherapy, nontaping supervisors had significantly higher expectations than the taping supervisors that videotaping would be more helpful than secondhand reporting in giving the resident self-understanding ($t=2.$ and in assessing the resident's clinical abilities ($t=1.$ $p<.05$). The taping residents saw the videotape as being significantly more helpful than the taping supervisors in understanding techniques ($t=2.50$, $p<.0$) and in making them a better therapist ($t=2.12$, $p<.05$). Thus the taping residents had more positive expectations about the benefit of videotaping than did the supervisors they were working with.

After videotaping. When we analyzed the responses to the videotape data for the 11 residents and their supervisors who participated in the taping, we noted significant shifts: despite pretape opinions that videotaping would interfere minimally with therapy, the supervisors involved in the taping showed a significant trend ($t=1.89$, $p<.05$) toward viewing the taping as interfering with the resident's ability to treat patients. Prior to taping, the residents felt their supervisors would be more technique oriented during the supervision using the tapes; subsequently they found the supervisors less technique oriented ($t=2.42$, $p<.0$ and found the videotapes less helpful in understanding techniques than they had anticipated ($t=2.$ $p<.05$).

Overall, the supervisors' pre- and post-taping views of videotaping for supervision did not differ significantly. However, residents' views became more po-

tive: of 199 possible responses, only 28 changed in a favorable direction, whereas 69 moved in a negative direction and 102 remained unchanged.

Patient Responses

Five patients reported that they were moderately anxious during the study, 1 reported being very anxious, and 5 said they were slightly or not at all anxious. None "greatly disliked" it, and 3 "greatly enjoyed it." All but 2 said the taping made the session "different" from all previous sessions, but the consensus was that the session was no worse nor better than any other and that they would not have said anything different had the session not been taped. Two-thirds of the patients said they would like to be taped again; the rest said they would not. These opinions contrast with those of the residents and supervisors, who felt the videotape had "somewhat" interfered with the patients' production of material, and agree with their estimations that the patients were "somewhat" more anxious during the session.

Rating of Patient Pathology

The taping residents and supervisors rated the patients in 102 specified areas of the modified Walter Reed Mental Status Evaluation (16). They estimated the degree of pathology and their confidence in these ratings before and after taping. Responses for each rating category were summed across all items and an overall confidence rating was obtained for both questionnaires. The supervisors were significantly more confident of their estimates of pathology after viewing the tape ($\chi^2=88.71$, df=3, $p<.01$). Residents' confidence ratings did not change significantly.

Comparisons of the pre- and posttape questionnaires revealed no statistically significant changes by either the residents or supervisors involved in the taping in terms of the nature or degree of pathology, diagnosis, or prognosis. However, for 3 patients the working diagnosis was altered by the supervisor as a result of the videotaped session. One was changed from depressive neurosis to passive-aggressive personality, 1 from anxiety neurosis to depressive neurosis, and in one case the diagnosis of passive-dependent personality was added to the previously perceived diagnosis.

DISCUSSION

Effects of Videotaping on Patients and Residents

Our data indicate that the videotaping of psychotherapy is not disruptive to the patient. Aside from experiencing a moderate increase in anxiety, the patients tolerated the procedure well and reported that it had no significant effect on their production of material or the therapy itself. Those who fear that videotaping is artifactual, intrusive, or counterproductive should be reassured.

Both taping residents and taping supervisors found that the videotaping was interfering and anxiety producing for the resident. Several explanations for this are possible: 1) that the residents were actually no worse than usual in these sessions but had unconsciously or consciously made themselves look good in prior supervision, as discussed by Stein and associates (10), 2) that the residents were afraid that the supervisor might use the videotape to evaluate the resident, or 3) that the supervisors were inexperienced in deriving maximum educational benefit from videotaping. Our data support Gruenberg (7), who feels it is the therapist, not the patient, who is inhibited by taping. Foreknowledge of the normalcy of such anxiety might diminish resistance to such procedures.

The Videotape and Assessment of Patient Pathology

Seeing the patient on tape had several effects on the supervisors. Prior to the taping they were rather unsure of their assessments of patient pathology, a fact that supports Grotjahn's feeling (8) that traditional supervision creates a great deal of uncertainty for the supervisor. Subsequent to viewing they became much more confident of their answers. Moreover, three supervisors made changes in the diagnoses given the patients. At least in some cases videotape can be of major diagnostic value.

The failure to produce more significant changes in patient assessment, such as those described by Stein and associates (10), Schuster and associates (9), and Buehler (11), may indicate that the initial interview situation is more subject to distortion than continuing cases wherein patients are discussed week after week. More pessimistically, it may be much more difficult to change one's opinion about a patient after several weeks of doing what one assumes is worthwhile and accurate supervision.

Videotapes and the Goals of Supervision

The unanimity of the respondents regarding the purpose of supervision deserves close scrutiny. The dichotomy found in the literature between the student-centered and patient-centered schools of supervision is not evident among our sample. Clearly, all aspects of psychiatry, including teaching clinical skills, improving patient care, and helping residents with their own problems, are acceptable goals. This sample, representing a significant percentage of our clinical staff, would accept a middle position between Tarachow (4) (patient-centered) and Fleming and Benedek (3) (supervision=therapy), disagree with Sloane (5) (teach only), and agree with Goin and Kline (6) that the resident's own feelings are important to discuss.

Although our respondents showed unanimity in regard to the goals of supervision, before taping there was some disagreement between the taping residents and supervisors about the uses of the videotape; the taping residents were more optimistic. After the supervisory session, however, the residents in general were disappointed. This disappointment could reflect the aforementioned differing expectations. Perhaps resident-supervisor teams could have profited by joint in-

struction on the educational uses of videotapes. The newness of the single videotape, without significant prior experience, possibly created problems, and multiple continuous taped sessions may be needed before its usefulness can be adequately ascertained.

CONCLUSIONS

Our data indicate that the videotaping of continuing psychotherapy for supervision is not harmful to patients, increases supervisors' certainty about diagnosis, and makes residents anxious. Better education regarding use of the technique, plus more extensive and controlled comparisons of taped and secondhand supervision will be needed before any clear-cut decisions about the educational value and cost effectiveness of such procedures can be made. The videotaping procedure is, at least, not a hindrance to supervision and is, to some extent, a valuable tool.

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Psychiatry Residents in a Milieu Participatory Democracy: A Resident's View

BY DENNIS GERSTEN, M.D.

Psychiatry residents respond with a variety of coping mechanisms to the lack of traditional structure in a milieu participatory democracy. To incorporate themselves into the system they must accept such democratic ideals as equality among staff and patients, group decision making, and free self-expression and give up some of their traditional ideas about staff and patient roles, treatment modalities, and the therapeutic environment. The author was a first-year resident in psychiatry on a university hospital inpatient therapeutic community; he discusses the conflicts between residents, who often adopt a "we-they" attitude, and the permanent staff, whose protectiveness of the ward community reflects their personal commitment to its ideals.

IN THE LAST FEW DECADES there has been an increase in the use of the therapeutic community for treating psychiatric inpatients. Although a lot of attention has been paid to patients' reactions to this type of system, little work has focused on the experience of residents in training in this setting. In this paper I will discuss psychiatric residents' experiences in such a system, the difficulties they encounter, and the coping styles they can adopt.

THE PHILOSOPHY OF THERAPEUTIC DEMOCRACY

In a therapeutic democracy there is no autocratic leader (whether a benign dictator or otherwise) to hand down decisions, but rather the community as a whole votes on almost all major issues and thus decides ward policy. Ward decisions are reached by vote and are presumably based on logic, common sense, and experience—not on formal credentials. The democratic policy extends to decisions on hiring and firing, role definition, promotion, discipline, and general ward policies. The implicit assumption in this therapeutic philosophy is that group decision is usually more valid than individual decision.

The concept of democracy extends to the treatment of patients. Decisions regarding patients' passes, class changes, discipline, responsibilities, and the management of difficult patients are voted on by staff and patients in community meetings. Each has an equal vote. Staff veto power exists but is rarely used. Staff-patient equality is fostered by the use of first names for everyone. Casual dress and the absence of identifying name tags or patient wrist bracelets emphasize the concept of equality.

The philosophy of democratic treatment includes the belief that this process will facilitate open and honest interaction between all community members and that the resolution of staff and staff-patient conflicts leads ultimately to good patient care. At the core of such a philosophy is the belief that it is possible to create an environment that is essentially humane and egalitarian and that patients improve by living in such an environment and integrating its ideals. The community process itself is thought to be the healing force (1)[1]. In practice, patients become members of the social system and are encouraged by their peers to act responsibly. They are rewarded for behavioral change by peers and staff.

A number of policies follow from these assumptions. Nonmedical staff ("primary contacts") are primarily responsible for coordinating patient care and use the resident as a supervisor. This modus operandi is prevalent in both traditional psychiatric hospitals and therapeutic communities in newer institutions. It accommodates changing treatment philosophies and aids nursing personnel who are in close contact with patients and who must make rapid decisions regarding patient care, often without concern for the power base (2).

THE RESIDENT'S EXPERIENCE

Entering the Milieu Environment

A psychiatric resident entering this system either directly from training on a medical ward or from a tradi-

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[1]This philosophy was originally espoused by the American moral therapists of the late nineteenth century.

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tional psychiatric ward is often more than surprised at the disparity between his self-perceived role and community expectations. He may be called doctor; he may not. His opinions are not accepted automatically because he has a medical degree: on the contrary his ideas about patient management, treatment, or ward staffing problems may be actively contested. Finally, he will discover that the patients he has expected to treat are usually not his to treat alone—each step is a group decision. The therapeutic democracy conveys to the resident a sense of constant chaos and continual crises on the ward and a profound sense of a lack of structure, or at least structure with which he is familiar. As a result of his experiences in training, the resident must at first assume that the ward is in crisis and that it will soon pass. He searches for causality in the crisis. Is it one or two difficult patients, is it the residue from a suicide last week, is it because a manic patient is tearing the place apart? Is it because the staff disagrees on the treatment plan and argues over the smorgasbord of drugs, insight, psychodrama, and Gestalt therapy? Or is it because of me? Finding no satisfying explanation, a resident may feel at times as if he is being swept along in a crowd, not knowing its direction, purpose, or destination.

As time passes, the resident may realize that this apparent disorder is the status quo. He draws on the coping mechanism that has always worked to counter chaos—the insistence that structure and order be imposed. His obsessive style has conquered medicine, and surely it will not let him down now. Usually he makes a few random attacks on certain symbols of the system—"People here should wear name tags," "I should be called doctor," "You should be called nurse," "You should be called patient." "The nursing station should be off-limits to patients (or at least the nursing station should have a sign on it saying 'Nursing Station')." Only much later might he learn that these issues are symbolic of a much deeper philosophical stance.

In the early stages of my residency in a university hospital milieu-oriented psychiatric ward, I felt acutely stressed. I felt that there had been a shift in the traditional power structure. It was true that I was the doctor, who did histories, physicals, workups and ordered medications; and it was true that the patients tended to regard me with traditional expectations of giving medications, providing some definitive knowledge, writing orders, and arranging for discharges. However, there was a difference; my opinions were challenged, and I felt I did not have the respect I had earlier in my training. Such a sense of decreased self-esteem was facilitated by covert attitudes and overt comments by patients and staff. In either case, as a fledgling psychiatrist I interpreted a lack of respect for my budding professional identity as an attack.

The Resident's Response

Sooner or later a resident must meet the new challenges of the milieu unit with new solutions and find ways of dealing with the polarities that ofte[illegible]. The less flexible resident often becomes in[illegible] authoritarian, makes unilateral decisions, [illegible] technical language, and insists on traditional [illegible] nurse-patient roles. He regains a sense of co[illegible] at the same time increases polarities, incre[illegible] sive-aggressive staff interactions, and force[illegible] isolation. Because the idea of equality is a c[illegible] of democratic philosophy, the authoritar[illegible] tends to catalyze struggles around that iss[illegible] scapegoat, and in so doing increase the co[illegible] rest of the staff at his expense.

Obviously the resident's range of respo[illegible] ed only by the number of his defensive s[illegible] dent may be primarily passive-aggressive [illegible] gress to a dependent position and turn o[illegible] responsibility to nursing personnel, he [illegible] highly obsessive and lose himself in the [illegible] per work, or he may conform in his beh[illegible] significantly changing any of his values.

The riskiest stance for the resident is [illegible] behavior and attitudes—risky because [illegible] must be willing to surrender and sett[illegible] tudes and be "adrift at sea" for some [illegible] integrates all or part of a different set [illegible]

Whichever defensive stance a reside[illegible] a milieu-oriented ward he must conte[illegible] symbolic ideals. Spontaneity is hig[illegible] many of the staff encourage patients [illegible] tion freely. Implicit in this attitude is [illegible] self-expression leads to free will and [illegible] termine one's own fate. The extreme [illegible] attitude is the avoidance of seclusio[illegible] avoidance of medication because [illegible] emotions," and encouragement of [illegible] ences in everyone. Ultimately th[illegible] struggle between those who consid[illegible] manists and those who uphold th[illegible] Once while this struggle was raging [illegible] eral staff members suggested that [illegible] into seclusion in order to understa[illegible] dents would at times insist on using [illegible] seclusion in treating patients.

During this kind of struggle, [illegible] forceful ones, were seen as inhum[illegible] interested only in superficial chang[illegible] residents in turn saw the advoca[illegible] without medication" as poorly t[illegible] trating, and undermining. In ac[illegible] that the staff members who acc[illegible] and insisted on treating psychos[illegible] were themselves being cruel t[illegible] valuable therapeutic modalitie[illegible] tients' suffering. As physicians [illegible] ty to use and understand medica[illegible] becoming comfortable with [illegible] helped define their identities [illegible] disagreements over medicatio[illegible] dents profoundly. However, [illegible] this kind residents could begin [illegible]

psychotherapeutic techniques and were in fact able to offer good treatment using less medication, think out their use of medications more carefully, and use them more appropriately.

Many of the issues debated on the unit seem unrelated, but most derive from the basic ward philosophy and the inherent built-in power struggles. Much of the distortion of this ideal of democracy results from the natural failure of its total actualization. On the one hand, nursing personnel want the benefits of the increased status and responsibility of being primary contacts but on the other hand are often dependent and demanding of the resident; when work does not get done, the resident inevitably picks up the slack. At the same time that equality is fostered, there are clearly several areas where staff are not equal. Residents tend to be far better at diagnosis and the use of medication; certain nursing personnel are excellent psychotherapists. However, the tendency to blur individual differences in the name of democracy results in the smoke screen of a melting pot rather than the true plurality that does in fact exist.

WARD LEADERSHIP

Another major difficulty a resident faces is the lack of clear leadership by the ward chief. The ward chief with whom I worked was, like those described elsewhere (2), a charismatic figure whose primary mode of leadership was often his charisma alone rather than the exercise of the power traditionally inherent in the position. He generally chose to "lead from behind" and allow others to emerge, fostering a more egalitarian system. Like other charismatic leaders (3, 4) he functioned more as a catalyst for the process of community than as an authority per se. However, inherent in his position was latent authority, which he used in times of crisis.

With this type of leadership, many staff members became more creative and autonomous and felt a sense of achievement that they had not felt in other psychiatric hospitals. Residents, on the other hand, often did not highly value personal growth by the nursing personnel and indeed were threatened at times by their increased independence.

Residents often did not share the ward chief's understanding of his role and saw his "leading from behind" as an absence of clear leadership. The possibility that he too was swept along in and by the democracy was far too threatening for residents to consider.

In attempting to understand ward dynamics, residents often perceive a we-they position. Indeed, it was only after I began carefully exploring ward philosophy on an individual basis that I realized the fallacy of this attitude. There was, as in any culture, a large middle ground not radically doctrinaire but allowing some freedom in various directions at different times. However, when the ward became polarized around a given issue, residents often felt "either you're with us or you're against us" and the rest of the unit responded similarly.

Where they are actually practiced, the ideals of democracy seem to clearly enhance equality in dealing with one another. The major problem is that people often act as if they want a firm leader whose decisions they can both respect and rebel against. In the absence of such a leader the staff either become disorganized or find a scapegoat within the system to attack in order to maintain their sense of cohesion. At such times it is often the residents who are singled out to serve as scapegoats. Without a traditional hierarchy, a new power structure forms, with the resident often feeling that he is at the bottom. In fact, it seemed to me at times that this abuse was a kind of initiation rite covertly sanctioned by the ward chief. This rite involved tolerating ambiguity yet nurturing staff who rejected the need for support and direction. The ambiguity of the situation is illustrated by the following example: On one occasion a resident asked a nurse if she minded letting him take a patient out of her psychotherapy group for an hour in order for him to do a history and physical. She became angry and, when later confronted with her anger, said, "Just say you're taking a patient out of group. Don't ask." The resident must learn how to walk the fine line between the role of authority and that of trainee.

It generally takes several months for a resident to begin to grasp what is meant by participatory democracy, because in the first months he is adjusting to the surface issues that arise which reflect the more basic attitudes. Many residents never get beyond dealing with these issues and leave the ward feeling disappointed, angry, and inadequate; others withdraw from the learning experience. One such resident in my ward commented that he had felt as though he had been thrown into a lion's den and that he was daily nibbled away by the nurses and psychiatric technicians. It is interesting that a resident at the Yale Psychiatric Institute milieu unit made the identical comment about that system (5).

THE WARD CULTURE

The essence of this system, the concept of democracy, is an important ideal to the permanent staff, and from this system many staff members derive a substantial amount of self-esteem. In fact, many refer to the ward community as their "culture" and are extremely protective of it. Thus, when residents question what seem at times to be trivial issues, they often threaten certain members profoundly. To tolerate the experience and grow from it, the resident must be able to empathize with and make an honest attempt to understand the staff position.

The mechanism by which this or any other subculture arises and is maintained is a complex issue. People who are compatible with the system are purposely selected to work in it. They generally like this

system because 1) it validates their own philosophical beliefs, and 2) it is syntonic with their personalities. It is risky to make generalizations about staff personalities, but the following seem to be true: many of the staff can attain high status on this ward regardless of their level of training; in their personal lives they set a high value on spontaneity and informality, which are compatible with the ward's philosophy toward expressive therapies; and they often feel alienated from the mainstream of psychiatry and the competitive ideal of American culture and find common support of their "ideal" in this system. The ward chief under whom I worked was well-suited to this situation for several reasons. He was very much committed to the ideals of participatory democracy and was determined to run a humane system. At the same time, it was compatible with his personality to give up his personal authority in order to facilitate group responsibility. His personality and philosophical outlook were harmonious with the ward and, combined with his charisma, facilitated continuation of the "culture."

It may seem that participatory democracy and a milieu-oriented ward are synonymous, but a resident must learn that they are distinct. In adapting to the ward, a resident must grapple with problems he would face in any milieu. Participatory democracy is a logical philosophy to wed to a milieu-oriented ward but it must be understood as just that—a philosophy that can in fact be divorced from the system with which it is associated. The ward structure (milieu) is the vehicle of the philosophy (participatory democracy). Only residents but also nonmedical personnel experience feelings of alienation and isolation before being accepted as an integral part of the ward. But the resident, who is transient, often remains outside. Despite his attempts to adjust, he will move on while the permanent staff remain. He feels no less lonely if he realizes that only part of the problem is his own. In order to cope with this experience most residents rely on their supervisors, former residents from the ward, or their chief resident for support. Ironically, it is usually only by this method that a resident can gradually come to understand the ward, integrate the new values (if he chooses to), dissolve the barriers between staff groups, and finally incorporate himself into the system.

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The Relationship of Community Mental Health Centers to the Professional Standards Review Organizations Program

BY O.B. TOWERY, M.D.

Given the realities of the professional standards review organization (PSRO) program and the necessity for accountability on the part of all health service providers, community mental health centers (CMHCs) should begin now to take an active role in planning their involvement in the PSRO program. The author points out the need for both psychiatrists and nonphysician professionals to become involved. There are many types of mental health services delivered in a given area, and the active involvement of CMHCs can be a major determinant of the shape and direction of this impact.

COMMUNITY MENTAL health centers (CMHCs) should begin now to actively plan their involvement in the professional standards review organization (PSRO) program. Although this program was authorized by amendments to the Social Security Act more than five years ago, until recently funding has been quite gradual. Physician groups in many PSRO areas had applied for planning grants, but there was little federal money available to support these efforts. However, recent funding appropriations for the support of local PSROs indicate that the program will rapidly become and remain the backbone of quality assurance activities nationwide. At this time PSROs are primarily focusing their efforts on the review of stays in acute, short-term general hospitals. Unlike many federal programs that are thoroughly conceived at the federal level and then implemented, the PSRO program is designed to evolve over the next several years, to experiment, and to establish policy based on whatever mechanisms seem to work well to assure quality care.

There are many ways in which PSRO review will have an impact on the types and quality of mental health services delivered in a given area, and it is hoped that the active involvement of CMHCs will be a major determinant of the shape and direction of this impact. The decision as to whether a particular admission will be paid for or will continue to be paid for beyond a certain point, as well as evaluations of the efficiency and effectiveness of the care given, will be made by either the PSRO itself or a delegated hospital committee. Clearly, the validity and usefulness of these determinations will depend on the degree of involvement by community mental health professionals.

The immediate emphasis of the PSRO program is on short-stay general hospitals and long-term care facilities, and direct involvement of mental health professionals in the review process is now limited to these levels of care. It will not be long, however, until PSROs begin to address ambulatory care. At that time PSRO policies and activities will have even greater consequences for the mental health care for which a CMHC is responsible. Even the functioning and fiscal viability of a center may be affected, especially if a national health insurance program with a broad range of mental health benefits is enacted.

Before considering the special situation of peer review of mental health services, it is necessary to briefly review the PSRO program and its functions.

PSROS

A PSRO is a voluntary, nonprofit professional organization representing a substantial proportion of the practicing physicians in a designated area. The PSRO must assure that health care provided to patients under the Medicare, Medicaid, and Maternal and Child Health programs is medically necessary and consistent with professionally recognized standards of care. Once a PSRO is operational (PSROs are accorded a "planning," "conditional," or "operational" status), the responsibilities currently held by Medicare contractors and Medicaid state agencies are relinquished to the PSRO. The PSRO's quality assurance program must be based on peer review and continuing education and must result in the improvement of quality, appropriate use of health care services, and continuing feedback about the effectiveness of the system.

A PSRO either performs or delegates to a hospital the responsibility of performing both concurrent and retrospective review. The purpose of concurrent review at present is to assure the necessity of admissions, the appropriateness of hospital stays, and the effectiveness of discharge planning. A PSRO or delegated hospital committee must also conduct medical

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care evaluation studies, which are usually retrospective. The purpose of these studies is to identify deficiencies in the quality of health care and in the organization and administration of its delivery and to correct these deficiencies through education and administrative change. These studies are usually of short duration and focus on particular problem areas. Explicit criteria must be developed for objectifying the concurrent and retrospective review procedures and for screening purposes. In the future a PSRO will regularly analyze profiles on practitioners and patients in its area in order to identify deviations in the provision and utilization of services.

INVOLVEMENT OF NONPHYSICIANS

There are many important issues yet to be resolved that will affect the relationship of PSROs and CMHCs. One of the most important of these concerns the role of nonphysicians in the PSRO review process. A basic principle of the CMHC program from the beginning has been that the delivery of mental health services need not and should not be directed exclusively by psychiatrists. Many CMHCs do not even employ full-time psychiatrists or other physicians. In some ways it is unfortunate for CMHCs that the PSRO review methodologies are having their beginnings in the short-stay general hospital. Regulations developed for reviewing services at this level of care are concerned with physician-initiated care and thus appropriately delegate review authority almost exclusively to physicians.

However, in a CMHC many of the services that will come under PSRO scrutiny are delivered independently by nonphysician mental health professionals. Further, the blurring of roles that has occurred in many centers has resulted in a situation where a given service may be competently performed by several different professionals. For instance, an intake evaluation might be performed by a nurse, social worker, or psychologist. One implication of this is that within a CMHC representatives of several of the different mental health professions can and should contribute as a team to the reviews surrounding such activities. On the other hand, each of these professionals still has unique contributions to make in the treatment process. Thus, for example, criteria for appropriate placement after hospital discharge might best be developed by social workers. Each profession has its own limitations as well, and for that reason only professional peers can fairly decide whether a given clinical decision is appropriate. For example, the criteria for the appropriateness of obtaining a medical consultation might be different for a nurse than for a social worker, depending on the educational and experiential background of each.

The required statutory mechanism for the involvement of nonphysician health care practitioners in the PSRO program is the PSRO Advisory Group (1). The Advisory Group relates to either the statewide Professional Standards Review Council or [illegible] the PSRO, depending on whether or not [illegible] fewer than three PSROs and therefore does [illegible] statewide council. If there is a statewide [illegible] should seek assistance in assuring the [illegible] effective involvement of nonphysician health [illegible] titioners in each PSRO in such activities [illegible] ment of criteria and of review mechanisms [illegible] physician services and the review of [illegible] nonphysician peers (2). The council should [illegible] vice from the Advisory Group relating to [illegible] informational and liaison activities with [illegible] that represent nonphysician practitioners [illegible] facilities. The Advisory Group may also [illegible] statewide council in evaluating the per[illegible] state PSROs in areas relating to nonphysic[illegible]

If there is no statewide Professional S[illegible] view Council, the Advisory Group will [illegible] to the PSRO in many of the same ways that [illegible] the statewide council, assuring that nonphy[illegible] ticipate appropriately in the review proce[illegible] sory Group should also examine and com[illegible] review plans of hospitals regarding their [illegible] of nonphysicians.

As in other matters relating to PSROs, t[illegible] Groups' influence will to a large extent de[illegible] eral factors, not the least of which is the [illegible] nonphysicians and their organizations. [illegible] group will also be concerned with the PS[illegible] ship to the various health care facilities [illegible] behooves CMHCs to take an early activ[illegible] sure that their special concerns are not o[illegible] group made up of dentists, podiatrists, an[illegible] ministrators, for example, might very w[illegible] sent the interests of the nonphysician [illegible] professionals in a CMHC.

In terms of the review process itself, [illegible] practitioners should be involved in the de[illegible] most medical care evaluation studies, [illegible] CMHC where a team approach is used. [illegible] mittal, "Policies Applicable to PSROs [illegible] Health Care Practitioners Other Than [illegible] Peer Review in Short-Stay Hospitals" [illegible] this involvement when care provided [illegible] physicians is being reviewed.) Even wh[illegible] related primarily to care rendered by ph[illegible] as a study to evaluate the use of psychot[illegible] nurse or pharmacist might well contribut[illegible] spective which augments that of the psy[illegible]

Finally, nonphysician reviewers sho[illegible] tive role in the corrective actions taken [illegible] the review. If this involves continuing e[illegible] practitioners should take part in identif[illegible] oping educational mechanisms related [illegible] ty. If deficiencies in service are related [illegible] tive policy or resource allocation, appr[illegible] mendations should be made in these ar[illegible]

As indicated previously, PSROs are a[illegible] reviewing care in short-stay general hos[illegible] long-term care facilities. In most cases th[illegible]

affect the care given to the patients of a CMHC only when the CMHC's inpatient unit is in a general hospital or psychiatric hospital whose services fall under PSRO review. The PSRO program is now just beginning to direct itself toward the review of ambulatory care, and, with this, both the necessity and the opportunity for CMHC involvement will increase.

REVIEW OF AMBULATORY CARE

Thus far the description has been of PSROs as they are to conduct review of inpatient hospital services. Noninstitutional services are now being reviewed only in relation to demonstration projects. However, Public Law 95-142 (the recently enacted 1977 Medicare-Medicaid antifraud and abuse amendments) requires that PSROs will now have to address ambulatory care within two years of the time they become operational. Soon, the review of ambulatory services will take its place alongside inpatient review. Ideally, such review will be integrated to the point that the whole spectrum of care provided to any single individual can be evaluated as a total picture. This will permit a much more meaningful review, especially for CMHCs, since questions of appropriateness and necessity of one level of care are so often related to the availability and acceptability of other levels of care within the service system.

At this time it is impossible to predict what form PSRO review of ambulatory services will take, since sophisticated methodologies for such review have simply not yet been developed. Most of the review activities that have been developed relate to individual claims payment by third parties and are focused almost completely on efficiency and cost containment. Issues usually addressed are those of patient eligibility for coverage by a given plan, whether the services rendered are covered by the plan, and the appropriateness of the provider's charges. Patient and provider profiles, as well as office practice audits, have also been used to monitor quality in the ambulatory setting, but only to a limited extent (4).

Within a CMHC, as in other settings, there are many additional issues related to quality of care that must be addressed. Some of the principles and procedures developed for the inpatient setting will probably be relevant, but others will not. For example, the use of explicit criteria in review activities will probably remain an important requirement. Medical care evaluation studies, or some modification of these, are likely to be applicable to the ambulatory setting. The principle of correction by education and other principles related to appropriate utilization and follow-up of the review findings will also continue to be relevant. Procedures for peer review of services delivered by nonphysician mental health practitioners must continue to be an important part of the review system.

Two models for the review of services in CMHCs have already been developed and successfully implemented. At the Peninsula Hospital and Medical Center CMHC in Burlingame, Calif., three-member peer review committees meet weekly to review cases presented to them by therapists in person. In these sessions each case is discussed, and a course of therapy is agreed upon by the committee and the therapist (5). The Connecticut Community Mental Health Center in New Haven has developed a three-level approach to the review of services (6). Criteria relating to admission, assignment to group therapy, precautions against suicide, prescription of medication, and other aspects of the care given are first used by professional nonclinical reviewers for screening. Then peer reviewers examine selected cases and provide feedback on their findings. Other models, such as the routine peer review of prescribing patterns at the West Philadelphia Community Mental Health Consortium (7), have also been developed.

OTHER ISSUES

Other issues that must be resolved have to do with the nature of mental health care in general. Can we develop generally agreed-upon criteria that permit evaluation of all of the many types of mental health services delivered in a CHMC? Can we develop objective measures of progress and success for the many treatment goals that relate to complex intrapsychic changes and alterations in subjective experience? Can we develop review methodologies that meet the special needs of mental health, such as maximal preservation of therapist-client confidentiality and allowance for the legitimate variations in treatment length and intensity that derive from the different philosophies of treatment in mental health? Can we develop tolerance for the degree to which therapeutic success is dependent on factors beyond the therapy itself, such as client motivation? The answers to all of these questions must be affirmative if CMHCs are to relate successfully to the PSRO program.

CONCLUSIONS

Given the realities of the PSRO program and the necessity for accountability on the part of all health service providers, CMHCs should begin now to take an active role in determining their relationship to PSROs. Although there are still many areas in which PSROs have not been established or are only in the planning stage, many more PSROs will soon become operational. The development of the local PSRO should be attended to carefully, and both psychiatrists and nonphysician professionals should make every effort to become involved in the organization's review activities. An effort should be made to understand the policies and procedures of the PSRO program, which at times may seem difficult since many of the concepts of peer review and much of the language used are as yet

unfamiliar to many care providers. Developments at the national level should be closely followed.

In addition, centers should establish internal quality assurance mechanisms and develop the capacity to monitor themselves effectively. This is a requirement in the CMHC amendments of 1975 for all federally funded CMHCs, and a process should be undertaken by other centers as well. The review methodologies developed for general medicine and other specialties will not always be appropriate for mental health services, and usually those developed by mental health staff in a mental health setting will provide a more meaningful review process.

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Borderline Diagnoses

BY CHARLES L. RICH, M.D.

The author describes the development of the borderline concept and the apparent inaccuracy of the original hypothetical framework. The signs and symptoms used to describe a "borderline syndrome" are often shared with other psychiatric illnesses. The author recommends that clinicians consider patients with only those features as "undiagnosed" until further research determines whether a distinct syndrome exists.

IT IS A COMMON and expected phenomenon for physicians to adopt and make use of catchall terms to cover their diagnostic limitations. Words such as "essential," "functional," and "idiopathic" are frequently found in the medical literature. They often describe symptoms or symptom complexes for [illegible] known of etiology, pathology, and/or [illegible] large the utilitarian purpose of such [illegible] ognized by the practicing physician. [illegible] aware, in essence, that he or she is saying [illegible] tient has an undiagnosed condition.

There are attendant risks when such [illegible] accepted as distinct diagnostic categories. A major risk is premature closure of diagnostic [illegible] leads to the application of such general [illegible] variety of other specific conditions that may [illegible] symptoms. This may deny patients potentially effective treatments. The problem of shared symptoms in the face of a shortage of confirmatory diagnostic laboratory tests) is one that plagues psychiatry as well. The word "borderline" has achieved substantial acceptance in psychiatry. This report discusses the development of the use of the word "borderline" from theoretical as well as practical standpoints. An attempt is made to define issues that have led to the confusion associated with the use of the word. Finally, an attempt is made to bring together information from several sources [illegible]

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ing the so-called borderline syndrome to other studies of personality disorders.

THE HEALTHY-NEUROTIC-PSYCHOTIC CONTINUUM

One school of classifying psychiatric illness maintains (simply stated) that the various symptom complexes or syndromes are manifestations of a single psychopathological process that leads from health, through the neuroses, to psychosis. The syndromes are considered points on this continuum. Borderline patients are felt to comprise an unstable group that vacillates back and forth across the neurotic-psychotic boundary. Dickes (1) has argued (by analogy to other typical medical illnesses) that this single continuum concept is not accurate. He recommended abandoning the borderline concept as well as the terminology.

There is, indeed, systematic evidence pertinent to psychiatric illness that supports Dickes' stand. Studies comparing manic and schizophrenic patients (2) and depressive and schizophrenic patients (3) have shown qualitatively indistinguishable patterns of thought disorder in these otherwise clinically distinguishable syndromes. It has also been reported (4) that specific symptoms of psychosis (hallucinations) occur in several, more clearly defined syndromes (affective disorder, schizophrenia, Briquet's syndrome, organic brain syndromes, alcoholism), some of which are not usually considered psychotic. Finally, patients with schizophrenia, for whom the sine qua non is psychosis, are being seen more frequently sliding back and forth over that border as they are treated and then default from neuroleptic therapy and relapse (5).

It would seem, then, that the words "psychotic" and "nonpsychotic" (as opposed to "neurotic") may be useful clinical descriptors, but they should be used to modify rather than supplant more valid diagnostic categories. The evidence supports consideration of each syndrome as a separate entity, rather than as a point on a single continuum. It would also seem that research methodology should not attempt to compare "borderline" subjects to "neurotics" and "psychotics," since all three of these would appear to be unstable diagnostic labels.

MULTIPLE USES OF THE WORD "BORDERLINE"

A recent study (6) reviewed 20 patient charts that had been randomly selected for containing the word "borderline" in diagnostic terminology. It was found that 9 different borderline diagnoses were used. The diagnoses included borderline, borderline state, borderline personality, borderline syndrome, borderline psychosis, borderline schizophrenia, borderline schizoid personality, borderline hysterical character, and borderline character. Further, 7 of the 20 patients received 2 or more different borderline diagnoses; 5 received 2 such diagnoses and 2 received 3 borderline diagnoses.

This confusing and inconsistent clinical use of the word "borderline" has blossomed despite the fact that the only officially sanctioned use of the word is *DSM-II*'s "borderline mental retardation" (7). A second diagnostic framework devised for research purposes included "borderline features" as an adjectival modifier to another diagnosis, but not as a distinct syndrome (8). This has been recently changed to "schizotypal features," implying a closer relationship to schizophrenia than to the personality disorders (9). A recent preliminary draft of *DSM-III* does not include the word "borderline" anywhere, even in regard to mental retardation (10). It seems clear that the intent is to promote the use of more specific rather than general diagnostic terminology. This intent should be fostered, since psychiatry is under justified fire for its often casual approach to diagnosis. The addition of the word "borderline" to other diagnoses to denote atypical (as in "borderline features") and mild or, perhaps, incomplete (as in "borderline schizophrenia" [11]) forms of illness should be supplanted by the more specific adjectives.

EXCLUSION CRITERIA AND FOLLOW-UP STUDIES

Despite the loose clinical use of the word "borderline," its popularity suggests the possibility that there may be a common core of symptoms that clinicians and researchers are trying to describe and isolate. These signs and symptoms were initially studied by Grinker and associates (12). They were further refined by Gunderson (13), who, by using a semistructured interview, found five "summarizing statements" that significantly separated "borderlines" from both "neurotic depressives" and "schizophrenics." His study suffered from the shortcoming of starting with patients who had a certain clinical diagnosis of borderline despite the obvious lack of criteria for such certainty. In addition, Gunderson only excluded patients with evidence of organicity or a primary diagnosis of alcoholism or drug habituation, neurotic depression, and schizophrenia (the last two comprising the comparison groups).

Gunderson's summarizing statements, however, are quite similar to Small and associates' descriptions of patients with passive-aggressive personality (14) and Liss and associates' report of patients with undiagnosed personality disorders (15). The common clinical descriptions from all three studies included suicide attempts, drug abuse, manipulation, alcohol abuse, and anger. Liss and. associates were meticulous about systematically rediagnosing their patients both at initial inclusion and at follow-up (16). This excluded a number of patients who met the criteria for another, more well-established syndrome (17), again pointing out the necessity of providing rigorous exclusion cri-

teria in order to avoid as best as possible the shared symptom dilemma. Similarly, in a recent report (6) I found that 12 of the 20 patients who received some sort of borderline diagnosis met the criteria for some other illness as defined by Feighner and associates (17).

Despite their variation in quantity and quality, follow-up studies of patients with diagnoses of "borderline" (18), "passive-aggressive personality" (14), and "undiagnosed personality disorder" (16) suggest that there is a sizable group of such patients whose course is chronic and who remain characterized only by a persistence of the core signs and symptoms described by Gunderson (13). However, further studies aimed at better defining this possibly valid syndrome must pay scrupulous attention to excluding all other psychiatric syndromes because of the high frequency of shared symptoms. The approach necessary to do this has been outlined by Guze (19). It is also extremely important that clinicians pay scrupulous attention to ruling out other disorders that may be more treatable before assigning a patient to this group for which no effective treatment has been defined in controlled studies.

CONCLUSIONS

Research data suggest that the original basis for the borderline concept (i.e., the unitary health–neurosis–psychosis continuum hypothesis) is inaccurate. Loose and confusing clinical usage of the word "borderline" has moved it in the direction of clinical uselessness, which weighs in favor of dropping it from psychiatric nosology. This had wisely been done by the drafters of the proposed *DSM-III*. Gunderson (13) has devised inclusion criteria for a possibly valid syndrome. The high frequency with which these signs and symptoms are shared with a variety of other psychiatric illnesses makes systematic exclusion of those other illnesses imperative in further research. If there is validation of a distinct syndrome characterized by the signs and symptoms delineated by Gunderson, it should be assigned a neutral name (excluding "borderline") to avoid confusion with the multitude of other popular uses of the word. Until validation occurs, these patients should be considered "undiagnosed," as recommended by Hudgens (20). Finally, clinicians should remain alert that these signs and symptoms are frequently shared by other, possibly more treatable illnesses.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Drug History and Tardive Dyskinesia

BY ROBERT C. SMITH, M.D., PH.D., MICHAEL STRIZICH, AND DAVID KLASS, M.D.

Although there is a good deal of epidemiologic and experimental research clearly implicating neuroleptic drugs in the pathogenesis of tardive dyskinesia, it remains unclear whether any specific neuroleptics or dosage schedules contribute more strongly to the risk of tardive dyskinesia. This report presents preliminary findings from a study of drug history and tardive dyskinesia in chronically hospitalized mental patients.

Method

The subjects were 103 patients 50 years old and older selected for the drug-history study from a larger survey of tardive dyskinesia at Manteno State Hospital using stratified random sampling procedures based on tardive dyskinesia scores. Most patients were rated for tardive dyskinesia on at least two or three separate occasions with the Smith Tardive Dyskinesia Scale (described in part in previous publications [1, 2]); mean scores on a facial dyskinesia subscale are used in this report. The amount of neuroleptic drugs a patient had received over the entire period of hospitalization in the state system was quantified from medication records and doctors' orders. Because of changes in the procedures for medication records in the late 1960s, drug history records from 1968 on proved to be much more accurate; therefore, in this preliminary report, only post-1967 drug history data will be used. The amount of neuroleptics was measured both in grams and chlorpromazine equivalents (CPZ-E), using the conversion ratios of Davis (3). Correlations and partial correlations of several drug variables with mean facial dyskinesia scores are presented.

Results

Our analysis revealed that only one drug, fluphenazine, consistently had small to moderate positive correlations with tardive dyskinesia scores (see table 1). There was a much higher correlation when we separately examined the relationship between dyskinesia and the proportion of intramuscular fluphenazine a patient had received. In a small number of patients (N=12) who received any appreciable amount of intramuscular fluphenazine enanthate, the correlation between facial dyskinesia and proportion of neuroleptic medication given as fluphenazine was fairly high ($r=.70$, $p\approx.01$). The 5 patients in this group who had definite facial dyskinesia had received about twice as high a proportion of fluphenazine in their neuroleptic treatment (mean±SEM mg CPZ-E fluphenazine/g CPZ-E total neuroleptics=258±47) than patients with no signs or borderline signs of facial dyskinesia (mean fluphenazine=109±27), a statistically significant difference ($t=2.52$, $df=9$, $p<.05$). The total amount of thioridazine a patient had received tended to have a consistently small negative association with the dyskinesia score, although only some of these correlations were statistically significant. No other single drug had a consistent positive or negative association with the tardive dyskinesia score.

Contrary to our initial expectations, tardive dyskinesia was significantly negatively related to the total

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TABLE 1
Correlation Between Tardive Dyskinesia (TD) and Drug History

Drug History Variable	N	Correlation with TD Score: Zero Order	Correlation with TD Score: Partial r Controlling for Total Neuroleptics
Total amount of neuroleptics during hospitalization (CPZ-E)	103	−.26*	
Maximum amount of neuroleptics in one year (CPZ-E)	101	−.21*	+.17**
Amount thioridazine (CPZ-E)	101	−.22*	−.14***
Amount thioridazine (g)	101	−.22†	−.13***
Proportion of total neuroleptics patients received that was fluphenazine	99	+.29*	+.28*
Proportion of total neuroleptics given as intramuscular fluphenazine	12	+.70†	—††

*p<.005.
**p<.05.
***p<.10.
†p<.01.
††CPZ equivalence of total neuroleptics is controlled for in the original variable.

amount of neuroleptics that a patient had received. This finding held true when partial correlations were done to control for the amount of neuroleptic the patient received on the day of rating (partial correlation r=−.17, p<.05).

Discussion

Although our results suggest that one drug, fluphenazine, may be more toxic in regard to tardive dyskinesia, this conclusion should be regarded with caution because of several factors. First, all the patients had been treated with neuroleptic medications for many years and no patient had received only fluphenazine. Although we tried to control for some confounding factors by using partial correlations, it is still possible that a factor which was not controlled might account for the significant positive correlations with fluphenazine. Second, some patients who had not received substantial amounts of fluphenazine also had definite tardive dyskinesia. Third, in a single sample in which many correlations are computed, it is statistically possible to find a few significant correlations on a chance basis. Finally, our results on intramuscular fluphenazine, which showed the strongest relationship with tardive dyskinesia, are based on a small subsample.

It is important to note, however, that recently three other groups of investigators (4–6) studying the relationship of drug history to tardive dyskinesia found significant associations of tardive dyskinesia with intramuscular fluphenazine. We believe that additional work is now needed to determine whether these preliminary findings, based on research using retrospective methods, can be confirmed in more extensive prospective studies.

The fact that neuroleptic drugs are associated with tardive dyskinesia can lead to the idea that the higher the total amount of neuroleptics a patient has received the higher the risk or severity of tardive dyskinesia. However, when we reviewed the literature we found only one or two prior studies (7, 8) suggesting an association between the total amount of neuroleptic a patient had received and tardive dyskinesia. Patients who had received a relatively low total amount of neuroleptics (e.g., 1.5–112.5 g CPZ-E). [illegible] patients who had received far greater amounts of neuroleptics (up to 2500 g CPZ-E) found no relationship between total amount of neuroleptics and dyskinesia. The results of our study [illegible] the total amount of neuroleptic medication [illegible] has received over many years is not the [illegible] related to current tardive dyskinesia. We find a small positive correlation between the amount of neuroleptic that a patient received per year and tardive dyskinesia score (partial r=+.17). This correlation is very similar in direction and magnitude, to findings reported by Crane (10) that the maximum dose of neuroleptic was positively associated with tardive dyskinesia (correlation r=+.19). These results suggest that planning studies to investigate the relationship between neuroleptics and tardive dyskinesia it may be important to examine the schedule and consistency of neuroleptic administration rather than the total amount of medication a patient has received.

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Filicide During Psychotropic-Induced Somnambulism: A Case Report

BY DANIEL J. LUCHINS, M.D., PAUL M. SHERWOOD, M.D., J. CHRISTIAN GILLIN, M.D., WALLACE B. MENDELSON, M.D., AND RICHARD JED WYATT, M.D.

In this paper, we report the case of a patient with drug-induced somnambulism who was studied in a sleep laboratory setting. To our knowledge, this is the first such report in the literature.

Case Report

The patient, Ms. A, is a 44-year-old woman with a history of one somnambulistic episode during adolescence but none after that until age 40. She also had enuresis until she was 16. There is no family history of somnambulism, enuresis, epilepsy, or psychiatric disorders. Ms. A was seen briefly by a psychiatrist when she was an adolescent for the enuresis and again in her 20s for anxiety subsequent to an unhappy love affair. She had no other psychiatric problems until she was 39, when she developed a florid psychosis with agitation and persecutory delusions. She was diagnosed as schizophrenic, hospitalized for 2 weeks, and treated with thioridazine, 25–100 mg q.i.d., with apparent recovery. A year later, she began to re-experience anxiety symptoms and resumed treatment on an outpatient basis. Because of insomnia, she received bedtime thioridazine and the hypnotic trichloroethanol (the active metabolite of chloral hydrate). Three months later, after taking both her bedtime thioridazine and a double dose of trichloroethanol, she arose from her bed at 3 a.m. and, apparently in a dissociated state, stabbed her daughter to death. When she was examined the next morning, she was found to be perplexed, hallucinating, and delusional, and was again diagnosed as schizophrenic. At her trial, she was acquitted by reason of insanity and hospitalized under court order.

With phenothiazines and psychotherapy, her psychosis slowly cleared. However, she was noted to sleepwalk 2–4 times a week. Diphenylhydantoin, 100 mg t.i.d., ethosuximide, 1250 mg q.d., and amitriptyline, 150 mg q.h.s., had no effect on her sleepwalking. Repeated EEGs, including one with nasopharyngeal leads, a pneumoencephalogram, and a CT scan revealed no abnormality.

Investigation

After four years in the hospital, Ms. A was transferred to a National Institute of Mental Health research ward at Saint Elizabeths Hospital for investigation of her somnambulism. Because she showed no evidence of active psychosis, either schizophrenic or affective, her medication (thioridazine, 300 mg q.h.s.) was discontinued. During a 1-month period, 11 all-night EEGs were carried out using conventional recording and scoring techniques along with continuous video monitoring (1). There were no sleepwalking episodes. She was then placed on thioridazine, 300 mg q.h.s., and chloral hydrate, 1000 mg q.h.s.—the same combination she took the night she killed her daughter. Episodes of somnambulism were recorded 3 out of 4 recording nights. Since she had been sleepwalking previously while she was taking only thioridazine, 300 mg q.h.s., she was again put on this regime and sleepwalked 1 of the 5 nights recorded. She was then briefly tried on chloral hydrate 2000 mg q.h.s., but did not sleepwalk on the 3 recording nights. A double-blind placebo trial was carried out. On placebo, there was no sleepwalking during 5 recording nights. On the first night of thioridazine, 300 mg q.h.s., and chloral hydrate, 1000 mg q.h.s., she sleepwalked. Table 1 summarizes the results of these investigations.

Sleep EEG Findings

The patient's sleep EEG had several unusual features. Both on and off medications she showed awakenings from stage 4 sleep in over half the nights, and many more brief episodes (5–15 seconds) of movements during stage 4. Also, regardless of medications, she had a short REM latency of about 30 minutes (49 minutes is about average).

All recorded episodes of sleepwalking occurred out of stage 4 sleep, which is consistent with other reports (2, 3). These episodes occurred during the first half of night, when stage 4 sleep is most prevalent. A feature which, to our knowledge, has not been noted previously is that at least one REM period elapsed before each episode.

In order to understand thioridazine's ability to induce sleepwalking in this patient, we compared sleep indices during 9 nights on thioridazine and 9 nights without thioridazine. Thioridazine significantly increased REM time (mean±SEM=103±19 minutes versus 82±21, $t=2.2$, $p<.05$, two-tailed test), and there was a suggestion of an increase in percentage REM sleep (28±3 versus 23±5, $t=1.89$, $p<.1$). There was also a suggestion of increases in total sleep time (385±30 minutes versus 348±49, $t=1.83$, $p<.1$) and stage 4 time (62±28 minutes versus 40±17, $t=1.88$, $p<.1$) on thioridazine.

Discussion

These findings indicate that Ms. A only sleepwalks while on certain psychotropic medications. We are aware of only three previous reports of drug-induced somnambulism. Christianson and Perry (4) reported two dermatologic patients who developed somnambulism on bedtime chloral hydrate. Huapaya (5) reported sleepwalking after bedtime methaqualone in two cases, chlorprothoxene in another, and a combination of

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thioridazine and methaqualone in a fourth case. Flemenbaum (6), in the context of reporting several cases of pavor nocturnus (a stage 4 sleep abnormality) induced by large bedtime doses of neuroleptics and tricyclics, mentioned one case of somnambulism.

In all cases, the sleepwalking could be controlled by eliminating the offending substance totally, reducing its dose, or avoiding bedtime administration. Unfortunately, we did not have an opportunity to determine whether Ms. A would sleepwalk on daytime thioridazine.

In the one report in which data on childhood sleepwalking were available, sleepwalking was noted in two cases, sleeptalking in the third, and neither in the fourth (L. Huapaya, personal communication, 1978). Ms. A sleepwalked once in adolescence and also had a history of enuresis. Unfortunately, no data are available on a history of enuresis in the other cases.

Interpretation of the previously mentioned features of Ms. A's sleep EEG is difficult. We are not aware of other reports of frequent movement and awakening from stage 4 in patients with somnambulism or of the onset of somnambulism after at least one REM episode and the much shortened REM latency. The two latter findings might be related to each other but unrelated to somnambulism, since a decrease in REM latency is reported in schizophrenics in remission (7).

There was an indication that in the case of Ms. A, bedtime thioridazine may have increased REM and stage 4 sleep. Jus and associates (8) have reported increased REM and stage 4 sleep after large bedtime doses of neuroleptics but observed no such effect when the same dosages were administered in multiple daytime doses. Since somnambulism is a stage 4 phenomenon, it would be tempting to suggest that thioridazine's ability to induce sleepwalking is related to its action of increasing stage 4 sleep when given in large bedtime doses. However, this would not explain how methaqualone can induce sleepwalking, since this drug does not appear to increase stage 4 sleep (9).

The mechanism by which psychotropic drugs induce sleepwalking, therefore, remains obscure. It is possible that they interfere with an inhibitory system which normally prevents somnambulism. Future studies with "purer" pharmacologic agents may help elucidate such a system. For the present, administration of large bedtime doses of various psychot[illegible] with a history of sleepwalking (and, [illegible] stage 4 abnormalities) should be done [illegible].

TABLE 1
Relationship Between Bedtime Thioridazine and Som[illegible]

Medication	Nights with Sleepwalking
Thioridazine, 300 mg q.h.s.*	
with chloral hydrate, 1000 q.h.s.	4
Thioridazine alone	1
Total for thioridazine	5
No medication	0
Chloral hydrate, 2000 mg q.h.s.	0
Total for no thioridazine**	0

*The difference between thioridazine with and without [illegible] significant.
**The difference between the total for thioridazine and [illegible] significant (Fisher's exact test, p=.002).

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Prediction of Self-Mutilation in Hospitalized Schizophrenics

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We have recently seen several schizophrenic patients who injured themselves seriously during hospitalization on a locked inpatient ward. In each case no warning was identified before the self-mutilation. These events led to a search for predictive factors in order to develop a system approach capable of predicting and preventing self-injury in such patients.

Most of the literature dealing with prevention of self-injury has focused on psychotic children and the mentally retarded (1). There have also been a number of reports of self-injury in schizophrenics, described from a psychodynamic point of view (2–5). However, this literature only describes attempts to understand self-injurious behavior after the event or to control repeated episodes by behavior modification. To our knowledge, no one has developed a systematic method to identify which schizophrenic patients are at high risk for imminent injury.

Two examples of cases in which self-injurious behavior was not predicted and no specific preventive measures were taken are described below.

Case Reports

Case 1. A 46-year-old man was admitted to the hospital after inflicting superficial lacerations over much of his body with razor blades. The patient had an extensive psychiatric history and had been hospitalized in several VA hospitals, where he was diagnosed as having paranoid schizophrenia. At the referring hospital emergency room he stated that he was "the slasher" and that he still felt like hurting himself. On examination he was alert and oriented, with flat affect and loose associations. He denied homicidal or suicidal ideation, stating he did not know how the cuts had been made. He said someone else probably had cut him while he was asleep. During the first few days of hospitalization, the patient seemed preoccupied with auditory hallucinations. He told staff that he felt a great sense of personal guilt about world problems such as war and starvation. Later he attempted unsuccessfully to obtain a knife in order "to end it all," reiterating that he was "responsible for all sin." That evening the patient was found covered with blood. Both eyes had been enucleated. He later said that "a force" had overpowered him and taken control of his actions. He added that he had not feared the motives of this force.

Case 2. A 39-year-old man with a history of paranoid schizophrenia came to the hospital complaining of hearing confusing voices. He stated that he had been "under satanic attack" for several weeks and that people were out to get him. He had cut his forearm and neck seriously 24 hours before admission. At the time of examination he denied suicidal ideation and was calm and cooperative at all times. During his first day on the ward the patient appeared to be in good spirits and no longer reported the experience of a spiritual or moral struggle. Subsequently, however, he was found with a pencil through the inner canthus of his right eye. He stated that he had done this because "The Lord says, 'If thy right eye offend thee, then pluck it out.' " He added that he communicated to others through his eyes and that he was worried that others might thereby learn his secret thoughts. The patient was placed in restraints and tranquilized with neuroleptics over the next several days. Six days later he stated that he felt he had done "a stupid thing," that he "must have misconstrued the spiritual meanings." The patient exhibited no further self-injurious behavior during his stay.

Discussion

The two patients we have described had histories or obvious evidence of prior self-injury. In addition, both were outwardly calm preceding the event. One patient gave evidence of self-referential religious preoccupation, and the other believed he was under the control of a trusted outside force. All of the cases of self-injurious schizophrenics that we have seen have shared these features. We have also discovered other cases in the literature that fit this pattern (2–6).

Simultaneous with our study three investigators, (J.E. Crowder, C.A. Gross, and J.E. Heisen) accumulated a series of six cases of self-mutilation of the eye (unpublished data). Their findings are consistent with ours in that five of their patients had histories of previous self-injury, and all six had tyrannical consciences (usually with religious preoccupation). All six revealed guilt feelings displaced to the eye and all experienced a sense of relief from anxiety in the attempted self-enucleation.[1]

System Approach

We recommend the following approach to the hospitalized schizophrenic patient in order to assess the risk of imminent self-injury. The patient should be questioned and examined for history or evidence of pre-

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[1]Of the six patients in the Crowder and associates study, five were clearly or probably schizophrenic, and the sixth was diagnosed as a psychotic depressive with religious preoccupation and auditory command hallucinations.

vious self-injury. He should also be carefully examined for concrete religious preoccupation, for presence of auditory command hallucinations, and for delusions of reference or of control by outside forces. Careful questioning is then necessary to determine whether the patient trusts his concrete preoccupations, command hallucinations, or delusions. Patients who do not have this trust will often appear anxious and ask for relief from their torment. However, if the delusions and/or hallucinations are trusted, patients may appear outwardly calm. Just as the depressed suicidal patient who shows an elevation of mood and lack of distress may have finally resolved to kill himself, the schizophrenic patient, torn between the forces of God and the devil, may display outward calm, having decided upon the sacrifice of some part of his body for his greater spiritual good. The Bible tells us, "Better for part of you to be destroyed than for all of you to be cast into Hell."

We believe these outwardly calm patients present the greatest risk of self-mutilation, and we recommend rapid parenteral tranquilization as the treatment of choice. Such patients should be kept under constant observation until the rapid tranquilization brings the psychotic symptoms under control or until sedation supervenes. We consider the risks of rapid tranquilization as described by Ayd and others (7–9) to be minimal when compared with the risk of self-mutilation. When these patients are no longer psychotic, they usually regret their self-injurious behavior, saying, for example, "I misinterpreted the Bible and took it literally instead of metaphysically" (3).

Conclusions

No one can predict self-injurious behav[illegible] absolute certainty. Based on clinical exper[illegible] ever, we have proposed a method to dete[illegible] schizophrenic patients present an acute [illegible] episodes. When such a risk is establishe[illegible] quilization is recommended because it is [illegible] and can prevent self-injury. Our experie[illegible] method is encouraging. Since the gener[illegible] this system approach at our hospital, we [illegible] further episodes of serious self-injury a[illegible] ized schizophrenics.

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Anticholinergic Delirium in a Case of Munchausen Syndrome

BY MARC H. HOLLENDER, M.D., ROBERT C. JAMIESON, M.D., EMBRY A. MCKEE, M.D., AND HOWARD B. ROBACK, PH.D.

Atropine delirium resulting from the use of eye drops has been reported, usually in letters to the editor, for nearly a century (1). In almost all instances the medication had been used to prepare for a refraction or to treat an eye disease. In only one article was accidental exposure mentioned (2). The case we will report is unique in that an atropine-like substance was self-administered with the intention of producing dilated pupils to simulate a neurologic disorder[illegible] which was not expected by the patient, w[illegible] ric disorder (delirium).

Mention in psychiatric texts of atropine [illegible] sulting from eye drops is seldom more th[illegible] comment. Levin (3) stated in the *Americ[illegible] of Psychiatry*, "Poisoning can occur eve[illegible] doses [of atropine], such as those used [illegible] pupils." Chapman (4) noted, "Children [illegible] develop brief organic brain reactions after [illegible] of atropine in the eye." In the *Compre[illegible] book of Psychiatry*, Linn (5, p. 807) states [illegible] and its derivatives may cause characteristi[illegible]

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hallucinations in drug-sensitive adults receiving relatively small quantities in the form of eyedrops . . . ." In the same textbook, Peterson (5, p. 1114) noted, "Solutions of these agents [piperidine derivatives] are also widely used in ophthalmology. . . . These solutions may be swallowed in the tears by way of the nasolacrimal duct and produce anticholinergic intoxication."

In our search of periodicals, we found cases of atropine delirium resulting from eye drops cited in general medical, ophthalmologic, and pediatric journals, but not in psychiatric publications. Although psychiatrists are sometimes called on to treat this disorder, they seem less well acquainted with it than some other specialists. Kounis (6) commented, "Atropine . . . contributes more frequently than is appreciated, and in smaller doses than is believed, to . . . the atropine eyedrop delirium." Hopkins and Robyns-Jones (7) stated, "We are told by ophthalmologists that . . . delirious states associated with atropine administration are not uncommon." It has been suggested that mild mental disturbances following eye surgery "attributed to emotional stress of the post-operative regimen may be due in part to atropine intoxication" (8).

In recent years various atropine-like postsynaptic blockers have been substituted for atropine sulfate in the practice of ophthalmology. In addition, as in the case we will report, other anticholinergic medications may be used systemically, producing an additive effect. It seems appropriate, therefore, to speak of an anticholinergic delirium rather than an atropine delirium.

Case Report

Ms. A, a 28-year-old licensed practical nurse, came to the emergency room of Vanderbilt University Hospital late one evening stating that she began to experience frightening visual and auditory hallucinations a half an hour after taking two tablets of a proprietary medication (Sominex). The visual hallucinations were of bugs, dogs, long black snakes coming out of faucets, and laughing faces in cups of coffee, and the auditory hallucinations were of voices telling her how horrible her life situation was. She seemed to be disoriented to time, place, and person. (Subsequently, it was learned that, in addition to the Sominex, she had taken chlorpromazine, 200 mg, and amitriptyline, 150 mg.)

When seen in the emergency room, Ms. A was wearing a neck brace and a cast on her left forearm. She claimed to have broken her left radius and sprained her neck in a car accident. (During subsequent interviews, she reported first that her ulna was fractured, then that the radius and ulna were fractured, and still later that the fracture included the radius, ulna, and metacarpals.) X rays of the left forearm revealed no evidence of fractures, and the police had no record of a car accident in which she had been involved.

On physical examination Ms. A's pulse was rapid (116 beats per minute), and her pupils were markedly dilated and fixed. The pupils did not constrict in response to the application of 1% pilocarpine eye drops. Based on the clinical findings, the internist and neurosurgeon who examined her concluded that Ms. A was delirious. She was then referred to a psychiatrist who admitted her to the psychiatry unit.

Ms. A, previously diagnosed as having Munchausen syndrome, had had numerous hospital admissions in Nashville and elsewhere. She had at various times consulted many specialists—ophthalmologists, internists, neurologists, neurosurgeons, endocrinologists, orthopedic surgeons, and psychiatrists—for her "illnesses."

Soon after this admission to the psychiatric unit Ms. A had a seizure, presumably psychogenic. She was informed that in view of extensive neurologic studies in the past, no additional studies were planned. No other seizures occurred during this hospital stay.

Two bottles of tropicamide (Mydriacyl), a short-acting mydriatic, were found in Ms. A's hospital room. She was told that dilated pupils which did not respond to pilocarpine could only be due to eye drops and that she must be responsible for the pathological finding. Initially, she denied using eye drops, but she soon broke into tears and acknowledged that she had. Following discontinuation of the medication her pupils gradually returned to normal, but the hallucinations persisted for 2 weeks.

Comment

It is well known that atropine eye drops can produce delirium but less well known that delirium can appear with few or no significant peripheral signs of intoxication (7). In Ms. A's case the only physical signs were rapid pulse and dilated pupils. The old description of atropine intoxication—hot as a hare, red as a beet, dry as a bone, blind as a bat, and mad as a wet hen—applied only in part to her.

Cases of delirium have been ascribed to "inherent susceptibility to the drug" or to "idiosyncratic susceptibility." In other instances the large amount of the medication absorbed (one drop of the 1% solution contains 0.75 mg of atropine) probably reached a toxic level. Atropine eye drops are carried to the nasal mucosa and intestinal tract mixed with tears via the nasolacrimal ducts. How much eye medication Ms. A used is not known, but it is known that she did not exert pressure on the inner canthus of the eyes to prevent fluid from passing through the nasolacrimal ducts.

Although mention is made of special susceptibility to atropine delirium in the very young and the aged, many reported cases are in middle-aged patients. The age range is from early childhood to 82 years, and no age is spared.

The clinical picture may resemble delirium tremens because of visual hallucinations of insects and animals, as in Ms. A's case. Restlessness, fearfulness, agitation, confusion, and fever are commonly reported. Much less often there may be paranoid delusions and some resemblance to schizophrenia.

In most instances, discontinuation of the eye drops has resulted in the relief or disappearance of all symptoms of atropine intoxication in 6 to 48 hours. In only a few instances have the symptoms subsided in less than 6 hours or persisted for more than 48 hours. The diagnosis of atropine delirium has been confirmed by injection of small doses of atropine to reproduce the disorder (8, 9).

To differentiate pupillary change produced by eye

drops from neurologic disease, pilocarpine, a parasympathomimetic agent, is safe and reliable. It causes constriction of the iris sphincter unless atropine or another postsynaptic blocker, such as tropicamide, the substance involved in this case, has been used.

Physostigmine salicylate (Antilirium) was not used, but it is effective in treating anticholinergic intoxication when administered intramuscularly (1 mg repeated in 15–20 minutes if necessary).

In Ms. A's case, the anticholinergic effects of chlorpromazine, amitriptyline, scopolamine (Sominex) (10), and tropicamide were probably additive.

Although her disorientation cleared rapidly, Ms. A maintained that her hallucinations persisted during her 2-week hospitalization. However, it is difficult to know whether this was actually the case. Given the previous diagnosis of Munchausen syndrome and her unusual willingness to assume the psychiatric patient role as well as the medical-surgical patient role, it is not unlikely that she was attempting to prolong her hospitalization.

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Neuroleptic-Induced Supersensitivity Psychosis

BY GUY CHOUINARD, M.D., M.SC. (PHARMACOL), BARRY D. JONES, M.D., AND LAWRENCE ANNABLE, DIP. STAT.

Dopamine receptor binding sites have been reported to increase in the neostriatum after chronic treatment with neuroleptics, which could account for the dopamine hypersensitivity that induces tardive dyskinesia (1). We propose that similar changes may occur in the mesolimbic region in response to the chronic dopamine blockade of these drugs. Three kinds of clinical evidence are compatible with this hypothesis: 1) central nervous system (CNS) drug tolerance; 2) psychosis following neuroleptic withdrawal, which is correlated with signs of dopamine supersensitivity and which we would therefore term "supersensitivity psychosis"; and 3) psychosis associated with a sudden decrease in prolactin levels following neuroleptic withdrawal.

Study Reports

CNS drug tolerance. In a double-blind controlled study we compared fluphenazine enanthate given every 2 weeks with fluphenazine decanoate given every [illegible] maintenance treatment of 48 schizophrer[illegible] Before entering the trial, patients had rece[illegible] enanthate routinely for periods of 1 to 12 [illegible] 14). All patients underwent a further 1-mo[illegible] lization with fluphenazine enanthate. The [illegible] of the fluphenazine enanthate-treated pa[illegible] the trial ranged from 2.5 to 125 mg (medi[illegible] 39.3 mg) and after 7 months of treatment [illegible] 325 mg (median=50 mg, mean=69.1 mg). [illegible] increases in dosage were required to ma[illegible] therapeutic effect at the same level. In an[illegible] longed exposure to neuroleptics leads to [illegible] requirements to block the behavioral effec[illegible] (3, 4).

Psychosis associated with signs of [illegible] sensitivity. In a 6-week double-blind tria[illegible] serazide we studied the relationship betw[illegible] nesia and psychotic relapse in 32 patients [illegible] ophrenia (5). Half of the subjects rec[illegible] benserazide instead of their regular neu[illegible] and half received chlorpromazine. In the [illegible] the severity of tardive dyskinesia (assessed [illegible] ical impression scale of the Extrapyramid[illegible] Scale [2]) tended to be greater in the 8 pa[illegible] rated than in the 6 patients who did [illegible] 5.4±1.4 and 3.8±1.7, respectively, t=1.[illegible] ever, there was no difference in severity [illegible] nesia between the deteriorated (N=2) a[illegible] (N=14) chlorpromazine patients (means [illegible]

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3.9±1.9, respectively) or between the nondeteriorated patients in the two drug groups.

These results are consistent with the hypothesis that the relationship between severity of tardive dyskinesia and psychotic decompensation may be due to a common underlying mechanism of increased dopaminergic function covertly induced by long-term use of neuroleptics and made overt by drug discontinuation. There was no evidence that increased agitation caused a worsening of tardive dyskinesia, since the 2 deteriorated chlorpromazine patients did not show signs of having more severe tardive dyskinesia than the nondeteriorated tryptophan-benserazide patients.

Psychosis associated with sudden decline in prolactin levels. In a pilot study designed to test whether penicillin has an antipsychotic effect, 10 hospitalized schizophrenic patients chronically treated with neuroleptics had their medication withdrawn and replaced with oral penicillin for 6 weeks (6). Greater decreases in prolactin (geometric mean=22.6 ng/ml, range=11.7–51.7 ng/ml on day 0; geometric mean=6.0 ng/ml, range=3.7–10.3 ng/ml on day 42) tended to be associated ($r=.62$, $df=6$, $p<.10$) with symptomatic deterioration (mean BPRS total scores ±SD=37.5±8.8 on day 0 and 46.1±13.2 on day 42), and increased severity of tardive dyskinesia was significantly correlated with decreases in prolactin ($r=0.85$, $df=6$, $p<.01$).

There is evidence to suggest that supersensitivity does not occur in response to chronic dopamine blockade in the dopamine hypothalamoinfundibulum tract (7), so the extent of prolactin elevation can be seen as a measure of absolute dopamine blocking. That some patients require more dopamine blocking to control their symptoms, as suggested by their elevated prolactin levels, and relapse suddenly when the dopamine blocking is removed is consistent with the hypothesis that these patients have developed a supersensitivity in their mesolimbic dopamine receptor sites as they have in the neostriatum.

Discussion

The association between dyskinesia and psychotic relapse has been observed by others (8, 9). The hypothesis that tardive dyskinesia and supersensitivity psychosis may be caused by a similar mechanism occurring in different areas of the brain is suggested by the common factors that can alter the clinical picture of both syndromes: increasing the neuroleptic dosage decreases the severity of dyskinesia and psychosis, decreasing the dosage makes both worse, stress exacerbates both dyskinetic and psychotic symptoms, and L-dopa and amphetamine can increase the severity of both.

We suggest that neuroleptics can produce a dopamine supersensitivity that leads to both dyskinetic and psychotic symptoms. An implication is that the tendency toward psychotic relapse in a patient who has developed such a supersensitivity is determined by more than just the normal course of the illness. This may explain why Hogarty and associates (10) were unable to identify "good prognosis" patients who do not relapse when maintenance neuroleptics are discontinued.

Another implication is the possibility that this supersensitivity is irreversible. This is accepted to be true of tardive dyskinesia unless it is diagnosed early and medication is discontinued. If the same irreversibility is occurring in the mesolimbic region, the result would be patients who must remain on neuroleptics for the rest of their life regardless of the natural course of their illness. In the studies done by Hogarty's group, two-thirds of patients thought to be suitable for drug withdrawal after 2 years of drug therapy relapsed following drug discontinuation, causing the authors to state that "the need for maintenance chemotherapy may be indefinite" (10). In some of these cases, the need for continued neuroleptic treatment may itself be drug-induced.

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Tricyclic Overdose in a Patient Given Combined Tricyclic-MAOI Treatment

BY KERRIN WHITE, M.D

Recent reviews have documented the safety of combining monoamine oxidase inhibitors (MAOIs) and tricyclic antidepressants, particularly if one avoids parenteral medications, use of imipramine, or the delayed addition of tricyclics to established MAOI treatment (1–3). Reported overdoses have formed part of the "evidence" against use of tricyclic-MAOI combinations (4–8). Reviewing fatal versus nonfatal overdoses involving both MAOIs and tricyclics, Ananth and Luchins (3) remarked, "In none of the non-fatal overdose cases was the patient on a course of MAOIs before taking the tricyclics." In this paper I will describe such a case, in part to offset the alarm evoked by earlier reports, since fear of fatal reactions has prevented controlled research in this area for too long.

Case Report

A 21-year-old man was admitted with the chief complaint of hearing the voice of a dead friend telling him to kill himself. For the previous 6 months, after the death of this friend, he had been intermittently depressed and suicidal and had received chlorpromazine and amitriptyline in day care and in one previous psychiatric hospitalization. A few weeks before this admission, he stopped treatment, suffered fitful sleeping and a 9.1-kg weight loss, and began to hallucinate. When he was admitted he appeared depressed, hostile, and suicidal. Physician examination revealed no pathology other than obesity and chronic bronchitis. Complete blood count, serum electrolytes, and serum liver function tests were all essentially normal.

In part because of his history of poor response to amitriptyline, he was started on tranylcypromine, 10 mg q.a.m., and doxepin, 50 mg q.h.s.; gradually increased to tranylcypromine, 20 mg at 6:00 p.m., and doxepin, 150 mg q.h.s. He complained mainly of drowsiness and dry mouth but also had a stiff neck (relieved by diazepam), nausea, and vomiting. Blood pressure, recorded daily, never rose above 140/100. After 17 days on this treatment, he began to improve, and 17 days later he was discharged with a 2-week supply of both medications.

Two days after his discharge, the police found him unconscious. He had made a suicide attempt by overdosing on an unknown quantity of doxepin. He later persistently denied memory of the events immediately preceding his overdose. When he was first examined by paramedics, his pulse rate was 110 per minute, respiratory rate 40 per minute, and blood pressure 114/80. His pupils were equal and constricted, with slow response to light and dysconjugate gaze. On admission to our medical unit, within 3 hours after the police found him, he was lavaged and pink capsules were recovered. Pulse rate was 110 and regular, respirations were 36 and regular, blood pressure was 130/72 and his temperature was 98°. He was described as being in [illegible] i.e., purposefully responsive to painful stimuli [illegible] peared flushed, especially over the lower ex[illegible] flexes were normal. An ECG showed right [illegible] block. A complete blood count was normal [illegible] vated white count (14,800), serum electrolytes [illegible] except for elevated uric acid (8.3), and serum [illegible] tests were normal except for elevated creatin[illegible] nase (810). The patient awoke the next day, [illegible] otherwise without adverse after-effects and [illegible] abnormality.

Discussion

This patient, after more than a mont[illegible] tricyclic-MAOI treatment overdosed o[illegible] and presented a clinical picture consisten[illegible] tricyclic overdose. No hypertension, hyp[illegible] seizures occurred. This observation cont[illegible] few catastrophic case reports of similar c[illegible] ations, which have contributed to the pic[illegible] combination therapy. It seems significa[illegible] cyclic overdose occurred in a patient o[illegible] MAOI reputed to be most dangerous[illegible] mine. On the other hand, this overdose i[illegible] pin, a drug not (to my knowledge) pre[illegible] cated in presumed adverse reactions to[illegible] therapy. Although part of the explanation [illegible] more recent introduction of doxepin, thi[illegible] tainly been combined with MAOIs be[illegible] denced by Ayd's survey (9) of 55 such ca[illegible] ently without untoward effect. Perhaps [illegible] supersede amitriptyline as a drug of cho[illegible] nation with MAOIs.

Although this one case cannot prove th[illegible] bination therapy poses no special risks to [illegible] overdose, it is encouraging to those inte[illegible] bination therapy to know that one such [illegible] curred without unusual consequences [illegible] pay attention to the rare disaster while [illegible] the less dramatic, more commonplace co[illegible]

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Social Adjustment in Primary Affective Disorder

BY DAVID L. DUNNER, M.D., GERARD J. IGEL, AND RONALD R. FIEVE, M.D.

The purpose of this paper is to assess social adjustment in patients with primary affective disorder. Recently, Weissman and Paykel (1) studied social adjustment in depressed patients using a structured questionnaire—the Social Adjustment Scale—derived from an interview used by Gurland and associates (2). Their studies showed that the women depressives had worse adjustment in several social areas when they were depressed than when euthymic, and that euthymic depressed patients showed worse social adjustment than controls. The population studied by Weissman and Paykel was described as having primarily unipolar depression.

We have administered a self-report version of the Social Adjustment Scale (SAS-SR) to patients attending an outpatient lithium clinic (3). Our major interests were to describe the social adjustment states of these patients, most of whom had bipolar depression, and to compare data on the depressed women in our sample with the data reported by Weissman and Paykel.

Method

The study was conducted in the lithium clinic of the New York State Psychiatric Institute. All 169 subjects met the diagnostic criteria of Feighner and associates (4) for primary affective disorder and were further classified into bipolar and unipolar subtypes (5). Patients termed bipolar I had been hospitalized for mania at least once. Bipolar II patients had been hospitalized for depression only and had histories of hypomania. Patients termed "bipolar other" had never been hospitalized for affective disorder but had received outpatient treatment for depression and hypomania. Patients were classified as unipolar if they had been hospitalized or treated for depression and had no history of mania or hypomania.

All patients participated in the study voluntarily. Each patient was asked to complete the SAS-SR for the 2-week period preceding the clinic visit. This questionnaire consists of several questions in 6 areas of social adjustment (work, spare time, extended family, marital, parental, and family unit). The questions are each scored on a 5-point scale, with 1 representing excellent adjustment and 5 poorest adjustment. We obtained mean scores for each area and for the questionnaire overall. Patients were instructed on filling out the questionnaire by one of us (G.I.), and the questionnaire was reviewed for completeness of response.

At each clinic visit, the patient's mood was assessed by a trained nurse or psychiatrist (6). Mood ratings were assessed independently of social adjustment data. Most of the patients in this study were treated on an open or double-blind basis with lithium carbonate.

Results

The breakdown of this sample by diagnosis and sex is presented in table 1. The overall mean adjustment scores for these patients did not differ significantly by diagnostic group or by sex. These overall means were about 2, indicating good but not optimum adjustment.

The data were further analyzed by separating patients into euthymic and noneuthymic groups. Most of the noneuthymic patients were mildly depressed, and noneuthymic patients had higher mean scores (worse adjustment) than euthymic patients in most areas of social adjustment. When patients were separated by median age, younger patients had worse adjustment in work, spare time, and extended family. No significant differences emerged when the data from our younger women were compared with the SAS-SR data from Weissman and Bothwell (7). Weissman had evaluated the SAS-SR in a sample of up to 327 residents of the New Haven community (personal communication) and found that the mean values of the six social areas ranged from 1.3 to 1.9. Our 115 euthymic patients showed slightly higher mean scores, but these were within 1 standard deviation of the community controls.

Revised version of a paper presented at the VI World Congress of Psychiatry, Honolulu, Hawaii, Aug. 28–Sept. 3, 1977.

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TABLE 1
SAS-SR Scores by Sex and Diagnosis

Diagnosis	N	Age (years) Mean	Age (years) SD	SAS-SR Overall Score Mean	SAS-SR Overall Score SD
Bipolar I					
Men	31	46.4	13.3	1.82	.50
Women	43	43.3	14.5	1.90	.44
Bipolar II					
Men	20	50.9	14.2	1.87	.49
Women	26	53.5	12.6	1.96	.45
Bipolar other					
Men	9	42.0	13.7	2.20	.54
Women	14	38.4	12.8	2.15	.64
Unipolar					
Men	11	48.3	14.5	2.12	.67
Women	15	46.4	15.2	2.14	.53

Discussion

Our study, like those of Weissman and coworkers (1, 7), shows that age, sex, and mood state affect ratings of social adjustment. Age of subjects appeared to have less effect than mental state. Our younger bipolar I women patients showed lower (i.e., better) SAS-SR ratings in each social area compared with the Weissman and Bothwell sample of depressed women, and it is possible that the stabilization of mood disorder in this bipolar group resulted in improved social adjustment.

The community survey data, which were supplied to us by Weissman, indicate control subjects generally have mean scores of approximately 1.5 in each social area, with standard deviations of approximately 0.4. Thus, most of the control sample scored each question 1 (perfect adjustment) or 2 (slight problems). For our total sample (including noneuthymic patients), the overall means for the various diagnostic groups were ≈2.0, and standard deviations were ≈0.5. Since differences between controls and patients are slight, it is possible that further improvement in social [illegible] could not be assessed in these patients by [illegible] SAS-SR.

Weissman and associates (1, 7) have [illegible] their data as suggesting that depressives [illegible] measurable social impairment that improves with [illegible] They suggest that patients may have residual social impairment although symptomatically improved, and that psychotherapy (in combination with medication) may be of additional value [illegible] persistent social dysfunction. It is not clear [illegible] that patients whose moods have been stabilized with medication could show significant improvement in social adjustment, at least as measured by the SAS-SR. Our data do not suggest that there are consistent meaningful impairments in social functioning in euthymic patients sufficient to justify initial and maintenance treatment with psychotherapy. We suggest that patients with primary affective disorder can usually be well stabilized with medication alone and that psychotherapy (in addition to medication) should be employed only when the patient is clearly impaired in specific social areas.

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Laryngeal-Pharyngeal Dystonia as a Possible Cause of Asphyxia with Haloperidol Treatment

BY JOSEPH A. FLAHERTY, M.D., AND HENRY W. LAHMEYER, M.D.

Numerous case reports have associated sudden death with the use of phenothiazines. There has been speculation that these deaths were caused by seizures and asphyxiation, but recent literature has emphasized the cardiovascular causes (1).

We have recently seen several patients with respiratory distress associated with the use of the nonphenothiazine antipsychotic haloperidol whose symptoms were relieved by diphenhydramine. We will present two illustrative cases and discuss the possible association with the phenothiazine-related sudden deaths.

Case Reports

Case 1. Mr. A., a 28 year-old man, was admitted complaining of auditory hallucinations and external control. Examination revealed loose associations, suspiciousness, and paranoid delusions. Results of a physical examination were normal, as were urinalysis, blood chemistries, and a drug screen. He was started on 10 mg of haloperidol on day 4, and the dosage was increased to 20 mg/day 3 days later. He was improving symptomatically until day 10, when at 7:00 a.m. the nurse found him in bed making agonized gestures and noises and pointing to his throat. Although the respiratory rate was normal, the pulse was 96. The nurse thought he was having trouble breathing or swallowing. One of us (J.F.) arrived about 7:15 a.m. and saw the patient immediately. His skin and nail color was mildly cyanotic, auscultation revealed some upper airway sounds, and breathing was normal except for some intraclavicular retractions. Throat examination seemed normal, except for some collection of secretions and an absent gag reflex. The patient seemed extremely anxious. He was given 50 mg of diphenhydramine I.V. slowly and resumed normal breathing and relaxed immediately thereafter. No other dystonic or extrapyramidal symptoms were noted, and an otolaryngological exam later that day was unremarkable. Because of the possibility that this was a dystonic phenomenon, he was given 2 mg/day of benztropine during the next 2 weeks. No further dystonic phenomena were noted for the duration of the patient's stay (29 days). He had normal course of hospitalization, with a remission of the acute schizophrenic symptoms.

Case 2. Mr. B, a 39-year-old man with a history of four psychiatric hospitalizations for schizophrenia, was admitted with acute schizophrenic symptoms. He had not been taking antipsychotics. A drug screen was negative, and results of a physical examination were normal. He was started on haloperidol, 10 mg/day, which was increased to 20 mg/day by day 7. On day 8 he complained of "something wrong with his mouth," later saying that it was his throat. His pulse was 92 and his respiratory rate 16. One hour later he was found gasping in his bed, complaining that he could not breathe. He was extremely anxious and had intraclavicular retraction and stridor. Throat exam was normal; the epiglottis was visualized, but no abnormalities were noted. There was some tightness in the posterior neck muscles, which the patient later reported as painful. Mr. B was given 50 mg of diphenhydramine I.V., which produced a rapid improvement in breathing, muscle tension, and anxiety.

He was placed on oral benztropine, 2 mg b.i.d., without subsequent extrapyramidal symptoms or breathing difficulties. Two weeks later the benztropine was discontinued without any subsequent reactions.

Discussion

Most cases of sudden death associated with phenothiazines have been with the more sedating drugs (2). Many cardiac abnormalities have been reported with phenothiazine death (2, 3). Grollman (4) has noted that chlorpromazine acts directly on the autonomic centers of the midbrain and produces a blocking effect on sympathetic ganglia, resulting in hypotension. Postural hypotension is another possible cause of death, particularly in elderly patients given chlorpromazine. Unfortunately, as Leestma and Koenig (2) have noted, autopsy findings are not conclusive. As Peele and Von Loetze (5) suggested, it should also be remembered that there were sudden deaths in patients with lethal catatonia before the advent of phenothiazines.

There have been reports of syndromes similar to those we have described. Malitz and associates (6) and Solomon (7) have observed a similarity in symptoms between phenothiazine-associated sudden death and a bulbar palsylike syndrome. Plachta (8) has speculated that asphyxia might be caused by a disorder of the cricopharyngeal sphincters. Haeffner and associates (9) reported pharyngeal muscle spasm, neck cramps, swelling of the tongue, and cyanosis associated with phenothiazine treatment.

Our cases are similar to these in that the patients did not die. One patient was clearly cyanotic, and if he had not received diphenhydramine his course could have been different. Haeffner and associates (9) noted that pharyngeal spasms are usually relieved by antiparkinsonian drugs. Victor and Adams (10, p. 831) noted the efficacy of diphenhydramine in treating true parkinsonism, especially when used in combination with anticholinergics. The theoretical basis for this use is not certain. We chose diphenhydramine because it is easier to titrate and we have found its subjective antianxiety effect to be greater. We have encountered several other patients who experienced dyspnea and diffi-

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cult breathing within 1–2 weeks of starting haloperidol. The drug relationship was less clear because of previous drug use in one patient, the combination of haloperidol with lithium in another, and milder symptoms in a third. However, all reported subjective improvement following diphenhydramine treatment.

Is it possible that these patients were suffering from a laryngeal dystonia? Laryngeal spasms after intubation are known to compromise breathing. Also, dystonias are common in young men taking haloperidol during the first few days of treatment. These two patients experienced the symptoms early in the morning when blood levels are falling. This temporal relationship has been noted with dystonias. Both patients responded to diphenhydramine and had no recurrence on maintenance benztropine. The nonspecific sedative effects of intravenous or intramuscular anticholinergics might also account for the diminution of the subjective distress. As is the case with dyspnea in general, our patients seemed to be in marked subjective distress. The objective signs in these cases seem to rule out the possibility of simple panic attacks with hyperventilation, although a component of this seems likely. Allergic reactions seem unlikely because there were no other signs of allergic response, such as rash or wheezing.

The association between this syndrome [illegible] es of phenothiazine deaths is uncertain. [illegible] is a potentially lethal syndrome that respo[illegible] ment. Confirmation of our findings require[illegible] servations.

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Increase in Serum Prolactin by Exogenous and Endogenous Opiates: Evidence for Antidopamine and Antipsychotic Effects

BY MARK S. GOLD, M.D., D. EUGENE REDMOND, JR., M.D., RICHARD K. DONABEDIAN, M.D., FREDERICK K. GOODWIN, M.D., AND IRL EXTEIN, M.D.

There is extensive evidence from in vitro and in vivo studies of man and other primates (1) that prolactin secretion is predominantly controlled by a tonic inhibitory dopaminergic mechanism. The involvement of dopamine in the tonic inhibition of prolactin secretion and the action of antipsychotic medications at dopamine receptors have led to the suggestion that antipsychotic drugs can be identified by their stimulation of prolactin secretion (2, 3). Potent antipsychotic medications block dopamine receptors (3, 4) and stimulate prolactin secretion (2, 3); structurally similar drugs without such effects do not. These and more recent data suggesting that the prolactin-stimulation model predicts antipsychotic efficacy and cli[illegible] of me have led to the hypothesis that tube[illegible] dopamine blockade can be assessed in [illegible] tions in serum prolactin and that this [illegible] might be the best model system for scree[illegible] psychotic compounds (2, 3).

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Method

We studied the effect of morphine and [illegible]-8[illegible] (5), a synthetic *d*-alanine methionine-enk[illegible] ative, on serum prolactin in primates [illegible] month of acclimatization to the sample c[illegible] cedure (6), four *Macaca arctoides* wer[illegible] venous infusions of morphine sulfate and [illegible] two dosage levels, 0.4 mg/kg and 0.04 mg[illegible] dol, 0.1 mg/kg; and saline solution. As [illegible] viously (6) all monkeys received one of [illegible] dosage levels every week. Each experim[illegible] lasted 5 hours. During the last 4 hours [illegible] were drawn and drugs were administe[illegible] venous cannula, without handling or [illegible]

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Samples were taken at 60, 30, and 15 minutes before drug infusion, at the time of infusion, and at 15, 30, 45, 60, 90, 120, 150, and 180 minutes postinfusion. All samples were placed on ice and centrifuged within 1 hour. Serum was frozen at −20°C until assay. Prolactin was measured in duplicate by radioimmunoassay (6).

Results

Morphine in the 0.4 mg/kg dose produced a significant ($p<.01$) elevation in basal serum prolactin (expressed as nanograms per milliliter) from a mean (±SEM) of 16±2.75 to 64.25±13.3. Serum prolactin was significantly elevated at all time points from 15 to 90 minutes postinfusion. No significant increase in prolactin was found with the 0.04 mg/kg dose. FK 33-824 at both dosage levels produced a significant and sustained elevation in serum prolactin. Prolactin was significantly elevated ($p<.01$) from baseline at all time points after administration of 0.4 mg/kg of intravenous FK 33-824. Peak levels at 45 minutes postinfusion were 177.0±22.9.

The 0.04 mg/kg dose of FK 33-824 produced significant increases ($p<.01$) in serum prolactin at time points from 15 to 60 minutes after infusion. The peak level at 45 minutes postinfusion was 95±9.6. Haloperidol (0.1 mg/kg) produced a significant increase in serum prolactin to peak levels of 115.3±9.6. Saline did not produce any significant effects on serum prolactin.

Discussion

These data support speculations, behavioral studies, anecdotal reports from the era before antipsychotic medication, clinical experience in methadone maintenance programs, and recent pilot studies with psychotic humans that opiate agonists may be antipsychotic in man (7, 8).

Support for this hypothesis comes from data which suggest that, like the traditional antipsychotic medications, morphine, methadone, and other opioid agonists interfere with the postsynaptic action of dopamine (7, 9). Opiate agonists increase dopamine metabolites homovanillic acid and dihydroxyphenylacetic acid in rat brain and inhibit the enzyme dopamine-stimulated adenylate cyclase (9), as does the meperidine derivative haloperidol. These actions of opiate agonists are consistent with the prediction of antipsychotic efficacy on the basis of current theories of psychosis and the antipsychotic locus of action of the neuroleptics (4). Additional support for the prediction of antipsychotic efficacy comes from recent studies demonstrating that morphine and methadone increase serum prolactin in man and nonhuman primates (7) and endogenous opioid peptides increase serum prolactin in rodents by stimulation of opiate receptor sites that inhibit dopamine release (10). These prolactin data are consistent with the dopamine metabolite data which suggest that opiate agonists stimulate opiate receptors in brain to modify dopamine impulse flow and release, thereby interfering with the postsynaptic action of dopamine. Endogenous opioid peptides, the endorphins, may act as an inhibitory neuromodulator of dopamine activity, so that an endorphin deficiency could result in increased dopamine release and turnover and possibly psychosis. Clinical trials of endorphins, including the synthetic pentapeptide enkephalin FK 33-824, could provide extremely valuable tests of the current hypotheses of psychosis and conceivably could give clinicians an antipsychotic medication that, as a derivative of an endogenous compound, might reverse a primary deficiency state.

If opiate agonists are not antipsychotic in man, then a number of widely used model systems, (including serum prolactin, catalepsy reversed by apomorphine, dose-related elevation in dopamine metabolites, and inhibition of dopamine-stimulated adenylate cyclase) may not be particularly relevant to the antipsychotic locus of action of the neuroleptics.

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Another Look at Gluten in Schizophrenia

BY JAMES R. RICE, M.D., CHARLES H. HAM, M.D., AND WILLIAM E. GORE, PH.D.

Singh and Kay recently reported that

> schizophrenics maintained on cereal free diet and milk free diet and receiving optimal treatment with neuroleptic showed an interruption or reversal of their therapeutic progress during a period of blind, wheat gluten challenge. The exacerbation of the disease process was not due to variations in neuroleptics. After termination of the gluten challenge, the course of their improvement was reinstated. The observed effects seemed to be due to a primary schizophrenia-promoting effect of wheat gluten. (1, p. 401)

According to Dohan and Grasberger,

> Routinely treated schizophrenics, who on admission were randomly assigned to a diet free of cereal grains and milk while on a locked ward, were discharged from the hospital about twice as rapidly as control patients assigned to a high-cereal diet. Wheat gluten secretly added to the cereal-free diet abolished this effect. These and previous findings suggest that cereal grains may be pathogenic for those hereditarily predisposed to schizophrenia just as they are for celiac disease, a disorder that may be genetically related. (2, p. 685)

In previous writings Dohan (3) suggested that there is a genetic relationship between celiac disease and schizophrenia; he also suggested that there may be some correlation between the decreased incidence of schizophrenia and the decreased consumption of gluten products during World War II in Germany.

We decided to test the hypotheses that 1) chronic schizophrenics would experience an exacerbation of their illness after an increase in gluten intake, and 2) chronic schizophrenics who were receiving optimum treatment with neuroleptics would improve further with a diet lower in gluten.

Method

We began the study with 21 patients diagnosed as schizophrenic (5 schizo-affective, 11 paranoid, and 5 chronic, undifferentiated type). Five patients dropped out: 2 who left the hospital without permission, 1 who became too agitated to participate, 1 who was diagnosed as having lupus erythematosus, and 1 whose functioning was inadequate for continued participation. The 16 patients who completed the study had a mean of 9 years of hospitalization and a mean age of 38. Patients were selected according to Carpenter and associates' 12-point diagnostic system (4), a record review, and a history of chronic schizo[illegible] spent the first 6 weeks assessing base[illegible] functioning and adjusting neuroleptics t[illegible] mum level of functioning. During this [illegible] tient was rated twice on the Brief Psy[illegible] Scale by a blind rater. During the 6-wee[illegible] od patients were on a regular institu[illegible] ceived 30 g of soy flour blended in wit[illegible] sugar. Then an excess of gluten was [illegible] their regular diet by substitution of glute[illegible] in weekly intervals, starting with 30 g [illegible] by 5 g/week to a maximum of 45 g. At [illegible] week of increased gluten, patients we[illegible] blind rater using the Brief Psychiatric [illegible] and clinical observations were made by [illegible] nel using the Missouri Mental State[illegible] Scale. After the gluten phase we retur[illegible] Kool Aid drink phase for 4 weeks to [illegible] blind rating of the patient's level of fun[illegible] patient then began a gluten-free and mi[illegible] weeks, during the first 4 weeks of whic[illegible] soy drink. (All patients gave informed [illegible]

Results

After the gluten phase, 1 patient of [illegible] severe regression below baseline on t[illegible] atric Rating Scale and clinical obser[illegible] had paranoid schizophrenia since she [illegible] and has been in the South Carolina S[illegible] 14 years. She is now 29 years old. W[illegible] gesting excess gluten, she became se[illegible] uncooperative, and paranoid. We had [illegible] the gluten drink and give her intramus[illegible] for three days because she became u[illegible] refused to take her oral medications. [illegible] free diet phase, 2 patients improved [illegible] functioning above baseline on the B[illegible] Rating Scale and clinical observation[illegible] patients was the woman already [illegible] showed improvement in level of fu[illegible] creased level of paranoid ideation. [illegible] reduce her medications substantially [illegible] on a gluten-free diet after this study. [illegible] improvement she was never able to o[illegible] to her illness. She discontinued her c[illegible] was not closely observed and therefore [illegible] in the hospital. The other patient sh[illegible] able regression during the gluten ph[illegible] of functioning above baseline impro[illegible] on the Brief Psychiatric Rating Scale [illegible] servations during the gluten-free diet. S[illegible] a state hospital for over 13 years, wi[illegible] chronic schizophrenia, undifferentiate[illegible]

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of her improvement we were able to discharge her from the hospital and send her home to her family.

Discussion

In human research many variables can affect human behavior and the results of that research. We might conclude that the largest percentage of schizophrenic patients are not affected by gluten in their diet, but a small percentage of patients may be affected. According to Pfeiffer, "Dohan finds that 4 percent of hospitalized schizophrenics are sensitive to wheat gluten" (5, p. 417). Our work did not duplicate the success that was reported previously (1, 2). Since we studied only a few patients, we feel that additional studies involving more patients are necessary to confirm our conclusions.

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Reaction to Relaxation and Desensitization Outcome: Five Angry Treatment Failures

BY STEPHEN I. ABRAMOWITZ, PH.D., AND NOA WIESELBERG

The role of relaxation, once regarded as critical to successful desensitization, has come under increasing scrutiny in recent years. In Wolpe's original formulation (1), systematic desensitization was conceived as a counterconditioning process that required the pairing of the anxiety-evoking stimulus with an antagonistic response—usually muscle relaxation. However, London (2) suggested that the extinction procedure alone accounts for the major effects of desensitization. More recently, Rachman (3) and others have turned attention to such cognitive mediators as mental calmness, relabeling, and expectations. Empirical efforts directed toward extricating the comparative contributions of various aspects of relaxation to effective desensitization have yielded inconclusive findings (4). Some data support the counterconditioning position, whereas others dictate modification of the premise that certain components of relaxation training are essential ingredients.

Almost all of the relevant studies have been predicated on the assumption that technique variables alone explain desensitization outcome. However, it seems reasonable to suggest that the patient's ability or willingness to relax mediates any facilitative effect of relaxation training, physiological or otherwise. The inclusion of subjects who are relatively unreceptive, as well as those more receptive to relaxation, in desensitization-with-relaxation groups may attenuate the apparent impact of the training procedure.

This report is based on a project designed to illuminate the role of individual differences in imaging vividness in mediating the relative effectiveness of symbolic and in vivo desensitization of snake phobia.[1] Our data are entirely serendipitous: they were discovered on reviewing responses to a treatment evaluation item that asked if subjects had become angry when told to relax. However, they offer a fresh look at the old question of the place of relaxation in formulations of desensitization.

Method

Sixty-three persons who responded to newspaper announcements of the availability of treatment for snake phobia were administered the Fear Survey Schedule (5), an adaptation of the Attitudes Toward Snakes Scale (6), and the Behavioral Avoidance Test (7). Those 48 individuals who proved to be behaviorally phobic on the latter were randomly assigned to either imaginal or in vivo desensitization. From subgroups of 36 subjects who subsequently met a behavioral criterion of treatment success and 12 who did not, we obtained follow-up samples of 17 and 5, respectively. Nine original subjects treated by one of us (N.W.) were eliminated from the follow-up to reduce the potential for experimenter bias. Of the 17 remaining subjects who did not participate, roughly equivalent num-

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The authors wish to acknowledge the contributions of John M. Dyckman.

[1]The follow-up data are based on N.W.'s senior thesis, which was an outgrowth of a dissertation done by John M. Dyckman under the supervision of Philip A. Cowan at the University of California, Berkeley, submitted for publication as "Imaging Vividness and the Outcome of in vivo and Imagined Scene Presentation."

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bers had left the area, could not be reached by phone, or repeatedly failed to keep follow-up appointments.

Six advanced undergraduates trained in desensitization theory and technique conducted the five-session standardized treatments. Each session lasted about half an hour, and the five sessions were spaced over a 2½–3-week period. Both forms of desensitization were conducted in the same room. Fuller descriptions of the assessment battery and treatment procedures can be obtained from N.W.

The same two instruments and behavioral test taken previously were completed at posttesting. For the purpose of this research, whether or not a subject was able to handle the snake at that time was the behavioral criterion for assignment to the successfully or the unsuccessfully desensitized subgroup.

Subjects were invited to the clinic for follow-up approximately 9 months after treatment. They once again completed the psychometric measures and behavioral test and filled out brief treatment generalizability and satisfaction scales. The latter device included the statement: "I was angered when I was told to relax." Subjects were classified by their retrospective agreement or disagreement with this item.

Results and Discussion

All 5 individuals who did not meet the posttreatment behavioral criterion but only 1 of the 17 who did recalled having reacted angrily to being told to relax, a statistically significant difference ($p<.001$, Fisher's exact test, two-tailed).

To rule out the likelihood that initial differences in anxiety might have influenced reactions to the instruction to relax and thus the relationship with desensitization outcome, successful subjects' pretreatment means were compared with unsuccessful subjects' means on the psychometric and behavioral measures. No significant differences were found. However, the unsuccessful subjects' recollections of annoyance at receiving relaxation instructions could have resulted from a process of defensive rationalization. In that case, the negative reactions would have been a by-product of, rather than a factor in, the treatment failure.

O'Donnell and Worell (8) found that [illegible] rated depth of relaxation was related to [illegible] desensitization of anger. The present data [illegible] extend to a circumscribed phobia the notion that individual differences in perceived responsiveness to [illegible] cular relaxation are correlated with [illegible] outcome. Consistent with Borkovec and [illegible] servations (4), replication attempts [illegible] more seriously phobic individuals and [illegible] nonspecific demands for improvement.

The results support Rachman's emphasis [illegible] calmness (3), as well as the venerable [illegible] putatious (9) view of relaxation as an [illegible] gredient in successful desensitization. Clients may be less willing than others to relax when placed in an anxiety-arousing situation or, conceivably, such individuals are prone to relaxation resistance when asked to relax. Such information could lead to more efficient desensitization and the development of pretesting procedures to identify patients who are likely to encounter difficulty.

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Treatment of Imipramine-Resistant Depression and Lithium-Refractory Mania Through Drug Interactions

BY BARNETT MEYERS, M.D.

Discoveries in the area of drug interactions are of potentially great import to the practice of clinical psychiatry. Recent reports in the literature have demonstrated the usefulness of supplementing supposedly therapeutic dosages of imipramine with methylphenidate to raise blood levels of the antidepressant in previously unresponsive patients (1) and of adding chlorothiazide diuretics to lithium to reverse a nephrogenic diabetes insipidus (NDI) syndrome and to achieve satisfactory serum lithium levels (2).

The following case report involves the application of both techniques in a patient who had been resistant to treatment.

Case Report

The patient, a 33-year-old divorced man, was transferred to Northern Westchester Hospital after 2 weeks of treatment at a county facility for an acute manic episode. The patient had been hospitalized three times in the past 5 years because of affective illness and had a history of recurrent alcohol abuse, which increased during each manic episode including the present one. He had been treated on an outpatient basis for more than 1 year with 1800 mg of lithium carbonate and 100 mg of chlorpromazine a day. On admission to the county hospital, his serum lithium level was 0.9 mEq/liter. The daily dosages were then increased to 2100 mg of lithium and 200 mg of chlorpromazine, but the serum lithium level fell during the next 2 weeks to 0.67 mEq/liter.

When the patient was transferred to Northern Westchester Hospital, he was agitated, with a labile, expansive, and predominantly angry affect. He demonstrated flight of ideas and reported thought racing and insomnia. He had persistent paranoid ideation but no delusions or hallucinations. The sensorium was clear.

At Northern Westchester Hospital, the patient was continued on 2100 mg of lithium carbonate and 200 mg of chlorpromazine a day. The urine specific gravity (SG) on admission was 1.005. Other laboratory studies, including serum electrolytes, were normal. He complained of severe polyuria and polydipsia. A repeat morning urine SG of 1.005 was obtained, and the 24-hour urine volume was found to be 6800 cc. Renal concentrating ability in response to dehydration or vasopressin was not tested. Because of the gross evidence of lithium-induced NDI, the patient's lithium dosage was reduced to 1200 mg/day, and 50 mg/day of hydrochlorothiazide was added. One week later, the urine volume had fallen to 4900 cc, with the serum lithium level remaining constant at 0.65–0.70 mEq/liter. Further adjustments of both the diuretic and lithium were made, and at daily dosages of 1200 mg of lithium and 100 mg of hydrochlorothiazide, the patient's daily urine output fell to 2200 cc, with a morning SG of 1.014. The serum lithium rose to 0.93 mEq/liter and the manic affect disappeared. This was followed by the onset of depression. The chlorpromazine was discontinued, lithium was continued, and imipramine was started. The patient manifested depressed affect, hypersomnia, and lassitude and expressed feelings of hopelessness and helplessness. The depression persisted unchanged after 4 weeks of treatment with 250 mg/day of imipramine. Methylphenidate, 10 mg/day, was started, and within 1 week the patient's mood improved. He was discharged in complete remission. Two weeks later the methylphenidate was discontinued without any return of depression. The imipramine dosage was tapered off and eventually discontinued, and the patient has been maintained for 1 year on daily doses of 1200 mg of lithium carbonate and 50 mg of hydrochlorothiazide, with potassium supplements. His affect has remained stable and normal, he has returned to work, and he has not resumed drinking.

Discussion

The treatment decisions in this case relied heavily on clinical investigations of imipramine and lithium interactions with other medications. Glassman and Perel (3) have reviewed the literature demonstrating wide individual variations in the metabolism of tricyclic antidepressants and of blood levels after a given oral dose. Glassman and coworkers (4) recently added to the body of knowledge indicating that blood levels are closely correlated with clinical response by suggesting that there seems to be a therapeutic threshold serum level for imipramine. Levels achieved are largely determined by the hepatic microsomal enzyme system, which is known to be influenced by a variety of pharmacologic agents. Barbiturates, alcohol, and nicotine, putative enzyme inducers (5), would thus be expected to reduce the blood levels and clinical efficacy of imipramine. This patient's episodes of binge drinking before admission might have contributed to his unresponsiveness to the 250-mg dosage of imipramine (3.8 mg/kg).

Wharton and coworkers (1) used methylphenidate's property as a microsomal enzyme inhibitor to cause a 250% increase in plasma imipramine and successfully treated a series of patients who had unipolar depression that was previously unresponsive to treatment. The patient discussed here differed from their sample in that he had bipolar illness. It is of note that the addi-

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tion of methylphenidate did not "switch" this patient into a manic or hypomanic illness, possibly because of a prophylactic effect of his maintenance lithium level.

The major clinical problem encountered in this case was the treatment of a manic episode in a bipolar patient who developed signs of lithium-induced NDI. This syndrome is thought to result in part from the inhibition by lithium of the action of vasopressin on renal adenyl cyclase, resulting in diminished distal reabsorption of water (6, 7). Although a definitive work-up for this syndrome was not done, presumptive evidence for its presence was found in the excretion of nearly 7 liters of urine per day, with polydipsia and low specific gravity morning urines. The reversal of all of these abnormalities on a reduced dosage of lithium in combination with hydrochlorothiazide supports this diagnostic impression. Himmelhoch and associates (2) demonstrated that "thiazide-lithium synergy" can reverse the NDI syndrome and produce clinical improvement in lithium-refractory patients. This effect is presumed to result from a relatively greater reabsorption of lithium along with sodium in the proximal tubule secondary to relative sodium depletion (2). Consequently, serum lithium would increase, while less lithium would reach the distal tubule and collecting system to interfere with vasopressin activity. It could be speculated further that the consequent reduction in lithium dosage further diminishes the absolute amount of lithium reaching and affecting the distal kidney. This mechanism is more complex than that posited to explain the action of hydrochlorothiazide in reversing non-lithium-induced NDI (8).

Conclusions

It has been estimated that approximately 70% of significant depressive episodes respond to supposedly adequate oral dosage of imipramine (9) and that 80% of manic episodes respond to lithium (10).

Idiosyncratic rapid metabolism of the tricyclic has been offered as an explanation for some imipramine treatment failures (3) while the developme[illegible] syndrome may explain many lithium failu[illegible] I have described is an additional demon[illegible] applying principles of drug interaction ca[illegible] numbers of patients who are fundamental[illegible] sive to these two therapeutic agents. The [illegible] ticularly noteworthy in that the admini[illegible] methylphenidate to a patient with bipol[illegible] maintenance serum lithium levels did n[illegible] recurrence of a manic episode.

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Phenelzine-Induced Psychosis

BY L. MICHAEL SHEEHY, M.D., AND JERROLD S. MAXMEN, M.D.

There is a growing body of evidence suggesting that monoamine oxidase inhibitors (MAOIs) exert therapeutic effects on patients who have a heterogeneous group of "neurotic" depressive disorders, some of which have been labeled "atypical depression" (1), "hysteroid dysphoria" (2), and "phobic anxiety-depersonalization syndrome" (3). Although symptom patterns vary, patients with these disorders often have persistent dysphoria, hysterical traits (e.g., rejection sensitivity, overreactive affect), phobias, panic attacks, chronic anxiety, hypersomnolence, lethargy, hyperphagia, inverse diurnal mood variation, and initial insomnia. Classical vegetative signs of depression and psychotic symptoms are absent.[1] Because these individuals constitute a large proportion of outpatients and because the wider use of MAOIs for these patients is rapidly gaining acceptance in North America (4), it is becoming increasingly important to be aware of their desirable and undesirable effects.

Some authorities maintain that the overall incidence of MAOI side effects, with the possible exception of hypertensive crisis, has been underestimated (2), especially behavioral toxicities (4). In this paper we will describe a patient who seemed to have had a phenelzine-induced psychosis that did not resemble the usual, albeit infrequently reported, behavioral side effects of MAOIs.

Case Report

The patient was a 30-year-old married part-time student who complained of depression, academic difficulty, shyness, fears that she was not heterosexual (despite absence of homosexual feelings), little enjoyment from life, and failure to achieve orgasm during sexual intercourse. The patient had experienced these symptoms for 9 years, with the exception of shyness, which had existed since childhood. Anorexia, weight changes, delusions, hallucinations, hypomanic or psychotic symptoms, and suicidal ideas were absent. The patient's history did not reveal other psychiatric symptoms or substance abuse. Family history included chronic depression in her mother, father, and sister, without history of psychiatric hospitalization. The patient was given a diagnosis of chronic depressive disorder.

Because of the absence of endogenous features and the presence of initial insomnia and a tendency to oversleep, the patient was started on phenelzine, 45 mg/day p.o. Thirteen days after medication was begun, she experienced 2 days of slight drowsiness, which cleared spontaneously. Eight days later, she suddenly became terrified that she and her husband would kill each other. She experienced a friend's voice on the telephone as deep and sinister and noted that her husband's eyes also looked evil. She ran from the house in terror after becoming convinced that a cockroach she had seen was growing larger and would shortly explode. In addition, she expressed the conviction that she had been sexually abused by a male friend of her husband's, with the husband's compliance. Reassurance that this had not occurred did not prove helpful. Phenelzine was discontinued and the patient was given oral flurazepam, 30 mg, and haloperidol, 5 mg, by a physician friend. Despite this medication, she remained afraid of killing her husband, thought she might jump out a window, and was still actively delusional when she was admitted to the hospital 18 hours after the symptoms began.

Mental status on admission. The patient was a slender, shy, attractive woman with a gentle manner. Her affect was labile, shifting from anxiety to depression, and her mood was depressed. She was distractible and circumstantial and recalled many events from her childhood. Formal tests of attention, as well as memory functions and orientation, were completely normal. The primary admission diagnosis was atypical psychosis. Brief reactive psychosis and drug-induced delusional disorder were considered as alternate diagnoses.

Hospital course. The admission physical examination, review of nonpsychiatric systems, and vital signs were within normal limits. The patient was treated solely with psychosocial care, apart from one 10-mg dose of oral diazepam for restlessness. The first night in the hospital was characterized by periodic awakening in great fear. Within 24 hours all symptoms disappeared spontaneously and the patient appeared tired and more depressed than she had been before the symptoms appeared. She said she missed the intense moments of feeling she had experienced during her illness. Her symptoms remained absent after 48 hours of hospitalization and she was discharged with a diagnosis of drug-induced delusional disorder (*DSM-III*). She has been followed for 4 months since her discharge, and there has been no recurrence of her symptoms. A trial of imipramine, 200 mg/day for 1 month, produced no therapeutic benefits or side effects other than dry mouth.

Discussion

After 21 days of a regimen consisting of 45 mg/day of phenelzine this chronically depressed woman abruptly developed intense fears of losing control (e.g., becoming assaultive and suicidal), paranoid ideation, quasi-delusional perceptions, labile affect, distractible speech and thought, hypermnesia for childhood events, paninsomnia, thematic circumstantiality, and heightened emotional intensity. She had no signs of an organic brain syndrome or of a hypertensive crisis. There was no evidence of precipitating environmental stress. Her psychosis resolved within 48 hours after phenelzine was discontinued.

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[1]Unpublished data from F. Quitkin, A. Rifkin, and D. F. Klein.

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Although the cause of her brief psychosis cannot be determined with certainty, it appeared to be either a direct phenelzine-induced side effect or a drug-triggered psychosis in a highly vulnerable patient. Because the psychosis occurred when full clinical response to phenelzine would first be expected and resolved promptly after it was discontinued, it appears that phenelzine was the offending agent. Although readministering the drug would have helped to determine if the patient had a phenelzine-induced psychosis, this action was precluded by ethical considerations.

An alternative hypothesis is that the patient's psychosis was completely unrelated to phenelzine. This explanation is implausible because of the absence of a precipitating environmental stress, the timing of the onset, and the prompt resolution after phenelzine was discontinued.

An unusual aspect of this apparent phenelzine-induced psychosis is that it did not resemble the types of behavioral toxicities previously attributed to MAOIs. The overall incidence of these toxicities averages between 11% and 15%. The most frequently observed behavioral side effects have been hypomanic and manic episodes, restlessness, hyperactivity, agitation, irritability, and occasional confusion (4).

This report has several practical implic
clinicians should be alert to the possibility
treated with phenelzine (and possibly o
may develop a wide variety of psycho
Second, these psychotic reactions may
on discontinuation of phenelzine and
portive care. Third, it may be not
but also inadvisable to treat these brief
neuroleptics. Neuroleptic administratio
the natural course of the psychosis and
velopment of additional side effects.
growing role for MAOIs, especially
clinicians who formerly were reluct
them should now become acquainte
with their use.

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Treatment of Leukopenia with Lithium Carbonate: A Preliminary Report

BY RAMZY YASSA, M.D., VASAVAN NAIR, M.D., AND GEORGE SCHWARTZ, B.SC.

Although lithium treatment is not usually associated with blood dyscrasias, hematologic changes associated with the drug have been reported in both men and animals. These changes are in the direction of leukocytosis. Mayfield and Brown (1) observed white blood cell (WBC) counts as high as 16,000 cells/mm^3 in a group of 11 male manic patients treated with lithium carbonate. They reported a consistent WBC elevation in all patients. Murphy and associates (2) reported a similar WBC increase in 20 depressed and 11 manic patients during administration of lithium carbonte. O'Connell (3) reported an intermittent leukocytosis in 25 psychiatric patients maintained on lithium carbonate. Shopsin and associates (4) examined peripheral blood specimens from 22 hospitalized psychiatric patients: 21 patients showed leukocytosis during lithium treatment that was not related to diagnosis, severity, or duration of illness. These findings prompted us to administer lithium carbonate to thre
consistently showed leukopenia.

Dr. Yassa is Coordinator of Undergraduate Training, Douglas Hospital Centre, 6875 LaSalle Blvd., Montreal, Que., Canada H4H 1R3, where Dr. Nair is Director of Research Services and Mr. Schwartz is Research Associate. Dr. Yassa is also Lecturer in Psychiatry and Dr. Nair is Assistant Professor of Psychiatry, McGill University.

Method

The subjects were 3 patients with
phrenia hospitalized at the Douglas Hos
Montreal. Subject 1, a 52-year-old wo
had epilepsy, was receiving perphenaz
dephenylhydantoin, 300 mg/day; and fl
30 mg/day. Subject 2, a 30-year-old man
thioproperazine mesylate, 60 mg/day; c
500 mg/day; and procyclidine HCl, 15
3, a 53-year-old man with epilepsy, wa
inal, 60 mg/day; fluorzepam HCl, 3
diazepam, 10 mg/day.

Baseline peripheral blood counts were
patient was included in the study if the
count was less than 2200. Lithium, 300
given to the 3 selected patients for 3
which time the WBC (total and differen
lithium level were measured weekly.
ate was then discontinued and the WBC
ferential) was measured weekly for thre
There were no changes in the patients

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TABLE 1
White Blood Cell Count and Neutrophil Count for Three Patients*

Period	Patient 1		Patient 2		Patient 3		Mean	
	WBC	Neutrophils	WBC	Neutrophils	WBC	Neutrophils	WBC	Neutrophils
Baseline	2500	1500	3900	2145	2700	1400	3033	1682
Lithium administration								
Week 1	2800	1800	4400	2640	3400	2006	3533*	2148*
Week 2	2500	1750	4800	2880	3300	2013	3533	2214
Week 3	3400	2074	5000	3024	3800	2318	4067*	2472*
Lithium withdrawal								
Week 4	4000	1920	4100	2788	2900	1769	3666	2159*
Week 5	3600	2268	4100	2296	2400	1344	3366	1969
Week 6	3200	1600	4200	2255	2300	1288	3233	1714

*Significantly different from prelithium baseline ($p<.05$, t test).

during the 6-week trial or the preceding 3-month period.

Results

There was a consistent and significant increase in total WBC as well as in the neutrophil count in these patients during the 3 weeks of lithium administration, reaching a maximum by the end of the third week (see table 1). After discontinuation of lithium, there was a steady decline to baseline levels. The WBC increase was not correlated with serum lithium levels.

Discussion

Our results indicate that lithium carbonate counteracted leukopenias induced by phenothiazines in 2 patients and by barbiturates in 1. Greco and associates (5) and Stein and associates (6) have also found that lithium corrects neutropenia caused by cancer chemotherapy. Gupta and associates (7) have been successful in treating neutropenia accompanying Felty's syndrome with lithium.

The mechanism of lithium's hematologic action has not been established. Lithium ions stimulate granulopoiesis in vivo (2, 8) and in vitro (9). Lithium has been reported to act by increasing production rather than by redistributing granulocytes from the bone marrow to the circulating granulocyte pool (8, 10). This finding may be of great importance in the treatment of leukopenia due to various causes.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Associa[illegible] requests for information, etc. Letters should be submitted in duplicate to the Editor, who make[s] [illegible] regarding publication. Letters must be typed double spaced throughout and should not contain [illegible] words and 5 pertinent references. Criticisms of published articles will automatically be sent to th[illegible] response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we [illegible] writers of the disposition of letters or return those which are not printed.*

Megavitamin Therapy for Autistic Children

SIR: We wish to respond to the brief report entitled "The Effect of High Doses of Vitamin B_6 on Autistic Children: A Double-Blind Crossover Study" (April 1978 issue) by Enoch Callaway, M.D., Bernard Rimland, Ph.D., and Pierre Dreyfus, M.D. Unfortunately, a number of serious methodological flaws in the study make its conclusions misleading in several respects.

Two flaws pertain to the sample. There is considerable question regarding diagnostic accuracy for the children studied. In the report's title, the sample is termed "autistic," while in the body it is alternately referred to as "autistic" or "autistic-type." This discrepancy is quite confusing, especially in view of the fact that the senior investigator asserted elsewhere (1) the importance of precise diagnosis in relation to such severely disturbed children. He himself has advocated diagnostic distinction between "autistic" and "autistic-type" children, lamenting the blurring of the criteria that had confounded previous double-blind studies (2); using the senior author's own diagnostic criteria, less than half of the sample qualified as classically autistic. Brief mention of this limitation in the Comment section of the report does not negate the grave constraint imposed on external validity.

Even more serious limitations result from the sampling recruitment procedures. All children who participated in this study had formerly been identified as vitamin-sensitive. At best, then, the findings pertain only to that sector of the population of autistic-type children who are vitamin-sensitive. Any generalization from such findings to autistic children as a group is premature and unwarranted.

The study's third major area of difficulty involves behavior rating procedures. The reader is not given information regarding the actual criteria by which the behavioral data were rated, nor are interjudge reliability data provided. Without such information, the reader has no assurance that different judges would have made inferences similar to those reported and is unable to replicate the study.

The authors correctly predicted behavior patterns for 11 out of 15 children. Thus, almost one-third of the cases were misclassified. Moreover, the authors claim accurate prediction regardless of the magnitude of their success—some of the classifications depended on mere one-point judgment differences. In addition, prediction was accurate for only 4 of the 6 reliably autistic children, not a significant proportion.

Other design shortcomings include lack of direct control over experimental implementation by parents and failure to specify other medications being taken concurrently by subjects, thus ignoring possibly crucial chemical [illegible] fects.

In conclusion, because of serious flaws in [illegible] racy, sampling recruitment, and reliability of [illegible] the new study, inferences regarding the ef[illegible] doses of vitamin B_6 with autistic children sho[illegible] as very tentative and subjected to rigorous [illegible] tiny.

The seriousness of the disorder for the fa[illegible] and the desperation with which parents awai[illegible] tory explanations and improved treatment f[illegible] they face demand that more than the usual [illegible] cautions be applied to work in this area. To [illegible] conclusions is a grossly unfair enticement to [illegible] in distress.

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HAROLD BOV[illegible]
Succe[illegible]

Dr. Callaway and Associates Reply

SIR: Ms. Moss and Dr. Boverman co[illegible] some of the points we made in our article. [illegible] clinically heterogeneous, and the nature of [illegible] lation precluded the use of many otherw[illegible] trols and refinements. They might also hav[illegible] remark that none of the children had bee[illegible] megavitamin regimes. We are delighted tha[illegible] ther investigation is called for.

They do fall into the common error of [illegible] measure of reliability is needed before positi[illegible] accepted. Low reliability could indeed giv[illegible] when a real effect existed (false negative), b[illegible] chance effect, then the level of reliability [illegible] used is a moot point. (Had it been possible, w[illegible] included a measure of reliability.)

The invidious implications of their last paragraph seem uncalled for. The therapy, B_6, and even Rimland's more extensive regimen, is safe. At worst, they cause upset stomachs. On the other hand, some percentage of autistic-type children seem to be helped by B_6, and this impression is supported by double-blind withdrawal. These are observations, not conclusions.

We regret that our efforts upset Ms. Moss and Dr. Boverman to the point where our observations become "conclusions" and "grossly unfair enticement" to the parents of their patients. We can only hope they will join us in our active commitment to the "rigorous empirical scrutiny" which they recommend to others.

ENOCH CALLAWAY, M.D.
BERNARD RIMLAND, PH.D.
PIERRE DREYFUS, M.D.
San Francisco, Calif.

Sex Role Identification

SIR: The article by David Krueger, M.D., "Symptom Passing in a Transvestite Father and Three Sons" (June 1978 issue) raises an important theoretical point about the roles of observation, identification, and modeling in inappropriate sex-role behavior. We recently evaluated a 21-year-old patient in our clinic who reported concerns about cross-dressing behavior of ten years' duration. An interview with the patient's mother indicated that our patient's father was a "private transvestite" in that he wore a woman's nightgown in their bedroom. However, she reported that the patient had never directly observed this behavior. The patient did acknowledge hearing many fights between his parents, but never reported any awareness of his father's cross-dressing. Unfortunately, our patient has failed to return to the clinic for further evaluation, leaving us with an incomplete understanding of the paternal parent's contribution to our patient's transvestism. Nonetheless, we agree with Dr. Krueger that the role of the father should be examined in great detail in future studies of transvestism.

DUANGJAI KASANTIKUL, M.D.
HOWARD ROBACK, PH.D.
Nashville, Tenn.

Dr. Krueger Replies

SIR: Drs. Kasantikul and Roback point out one of several questions and areas of potential exploration regarding the roles of modeling, observation, and imitation in the identification process, especially as these processes relate to sexual role identity vis-à-vis the (inadequately studied) role of fathers. The absence of fathers has been demonstrated to be a psychopathological force to young children. Factors that have hindered consideration of the importance of fathers in development—besides their either providing a supportive environment or being absent—may include physical inaccessibility of fathers due to work, the provision of basic nourishment and affection by the mothers in the child's early years, resistance of the fathers to becoming therapeutically involved, and, perhaps, the immunity of patriarchy. These factors may have contributed to the fact that the literature deals mostly with the extreme situation of the absence of the father and does not deal sufficiently with less extreme situations.

The question implied by Drs. Kasantikul and Roback is the *necessity* for observation per se to occur in determining the specificity of a symptom choice in sexual role identity conflicts. Although not answered completely, there are suggestions in the literature which may serve to shape further research in this area.

The attitudes and examples communicated from parent to child are powerful and instrumental in creating masculine and feminine identities (or conflicts thereof) in both sexes. There is both conscious and unconscious reinforcement of the child's imitation of his parent's (or model's) behavior. Johnson (1) illustrates this dramatically in her collaborative treatments of parent and child, noting that in every case a child with a fixation at the genital level had a parent with a similar fixation, and further illustrates that a common element in many of the identifications is the choice of specific symptoms based on observation of a parent symptomatology: fire setting as a family symptom for three generations: soiling and messiness by the mother and subsequently the daughters; and bestiality being explicitly offered as a symptom choice. Additionally, it is well known that the choice of a fetishistic object is often presented to the child by the parents, who then exalt its value and use it as a deflecting focal point for some of their own problems.

The role of the father per se has been addressed. Biller (2) found that the segment of the father's behavior which the boy can directly observe seems to have the most significance in terms of his masculine development. Liten and associates (3) believe that the mother's problems are the greater determinants of sexual pathology in the pregenital period, and the father's influence is stronger during the oedipal and later periods.

The parents may subtly define a pattern of sexual behavior, and the parental gratification from the child's behavior would perpetuate it. Liten and associates (3) describe a case of a transvestite whose father remarked (as the father did in the case which I described), "His dressing up is just a phase. I went through it myself." Emch (4) observed that "the acted out identifications exhibit caricatured reproductions of long-past parental behavior."

How may we then scrutinize with greater precision the role of the fathers in this identification process? Naturalistic experiments involving both normal and pathological development may provide some of the data on which to modify and reconstruct our theory. This type of work is exemplified by Cramer (5), who reported on the longitudinal study of three boys with particularly outstanding developmental progression from a group of 45 boys studied from age 4 to onset of puberty. One of the central findings in these outstanding boys was their strong masculine identification with their fathers, which became the core for organization of defenses in character traits. These outstanding boys very clearly had an intense desire to identify with their fathers, and took on their attitudes and values, shared their activities and coping styles, wanting to be exactly like their fathers when they grew up. The beginning of this identification with fathers was in the oedipal period and remained strong until the end of their puberty.

The area of both normal and developmentally deviant impacts of specific interactive and intrapsychic constellations of the father is largely unexplored. The specific sexual identity behavior, attachment, discipline, and their effect on family organization would seem critical. An example would be the very different motivational force impinging on identification: from fear of castration and identification with the aggressor being a primary motivator at one end of the spec-

trum, to love and affection primarily motivating identification at the other end.

Implications for future research are suggested in several areas. First, the age of onset of transvestism (or other sexual deviation), including the detail and circumstances of the first episode, whether being self-initiated, being cross-dressed by the mother, or after observation of this behavior in some important figure. The specific relationships with both mother and father and their interrelationship are critical. Transvestism may have several meanings as well as being multidetermined in an individual. I now have in analytic therapy a patient who began cross-dressing when his mother suddenly died at his age 10, and the dressing has been only in her clothes and served the purpose of the perpetuation of her image in his mind and denial of her death. The "bottom-line dynamic" of relieving castration anxiety by acting out the fantasy of the phallic woman with whom the man can then identify should receive increasing skepticism as a ubiquitous and primary pathological force in sexual deviation.

Other areas of study might include family studies, including naturalistic observation of sexual behavior, normal and abnormal, as well as data that the psychoanalyst can collect, such as the unconscious processes of childhood motivational influences that determine present behavior and fantasy. Close collaborative observations between therapists when both child and parent have been treated, especially focusing on the dynamic emotional interplay between child and both parents, may be fruitful.

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DAVID W. KRUEGER, M.D.
Houston, Tex.

Subjective and Objective Analysis of Patients

SIR: That tardive dyskinesia can be more than a nuisance and that it can threaten life is important information. Daniel E. Casey, M.D., and Peter Rabins, M.D., deserve credit for discerning this and sharing with us their report "Tardive Dyskinesia as a Life-Threatening Illness" (April 1978 issue).

I am alarmed, however, that practically nothing is reported about what this particular patient felt and thought or what she may have worried about, nor did there seem to be any inquiry into the possible connection between these psychological factors and her physiological signs and symptoms. Perhaps it was merely the editorial restriction to 1,000 words that deterred the authors. But in light of the currently raging controversy about the "medical model" in psychiatry (of which the Letters to the Editor section in the same issue is an eloquent example) I worry that the purely physiological approach of this report is an omen of things to come, if these are not already present.

Even from a purely biochemical point of view, the treatment of depression and some anxiety with chl[illegible] puzzling, unless one assumes that in 1958 [illegible] were the only widely known psychotropic me[illegible] why no attention to the obvious reactive (gr[illegible] the presenting symptoms? Why no concern th[illegible] recurrent anxiety upon reduction of medicat[illegible] ticipation of no longer seeing the doctor. p[illegible] remaining supportive figure in her widowhoo[illegible] so strange if her "separation anxiety" (th[illegible] analytic myth) reached extreme degrees on[illegible] that her (presumably) new and enthusiastic [illegible] termined to take her off medications? Cou[illegible] contribute to, or wholly explain, the more un[illegible] symptoms observed, e.g., irregular breath[illegible] (anxious people do swallow air), as well as th[illegible] retching (in itself possibly secondary to ac[illegible] watch a baby regurgitate if it is not burped[illegible] gastroscopy, barium swallow, etc., were giv[illegible] chiatric interview was reported.

Finally, all signs are attributed to tardive d[illegible] by a process of elimination (not of psychogen[illegible] ever) than by positive evidence. True, an e[illegible] cussion on how an imbalance of neurotransm[illegible] tered sensitivity of receptor sites could expla[illegible] tions is offered. Apparently ignored is [illegible] understanding the physicochemical mechanis[illegible] and symptoms of a psychological state (be th[illegible] pression, elation, or what have you) does no[illegible] need to be fully aware of the patient's subject[illegible] as well and to deal with it on an interperso[illegible] mean more than just "bedside manners" or "[illegible] chology"). In fact, it is the competence to de[illegible] special expertise of the psychiatrist.

My quarrel is not with the authors; my qu[illegible] ominous trend in psychiatry which, in the na[illegible] ing to the mainstream of medicine" would ma[illegible] and pillpushers of us all, for the greater glor[illegible] and the detriment of our patients.

EDMUND [illegible]
Cup[illegible]

Drs. Casey and Rabins Reply

SIR: We agree that psychiatry is uniquely [illegible] its special expertise in dealing with psycholo[illegible] ological dimensions of patients. To take sides [illegible] dualism argument both neglects the relevan[illegible] view and misses the important unifying co[illegible] beings. Patients are best viewed subjectively [illegible] uals with unique empathically understandabl[illegible] responses, and objectively, i.e., as organis[illegible] interrelated functional systems.

Issues of personality, psychological needs, [illegible] ation are crucial in the care of all patients, [illegible] assure Dr. Kal that we have attended to these [illegible] ever, the compelling aspect of this patient's [illegible] we emphasized in our report was the unusual [illegible] constellation of symptoms and their increasin[illegible] though her symptoms increased with anxiet[illegible] evidence that the etiology of her symptoms [illegible] psychological conflicts. To be blind to the [illegible] physiological nature of the findings that form[illegible] this case report could have led to more seriou[illegible] and psychological sequelae.

We disagree with the notion that psychiatry's "returning to the mainstream of medicine would make biochemists and pillpushers of us all, for the greater glory of 'science' and the detriment of our patients." Rather, returning to the mainstream of medicine should lead to better diagnostic criteria and improved indications for our available treatment modalities. The biological contributions to our advanced understanding of psychiatric illnesses have immeasurably been to the benefit rather than to the detriment of our patients.

DANIEL E. CASEY, M.D.
Portland, Ore.
PETER RABINS, M.D.
Baltimore, Md.

Generic Versus Brand Name Psychotropics

SIR: The spreading legalization of the substitution of generic for brand-name drugs, including psychotropics, is responsible for an escalating number of patients receiving generic rather than the prescribed brand-name drug from the dispensing pharmacist. In the case of tricyclic antidepressants, this practice can be hazardous because, as the Food and Drug Administration has stated in the Federal Register of February 17, 1978, "available data suggest that the various marketed brands of the same oral tricyclic antidepressant may not have comparable therapeutic effects" (due to bioequivalence differences) and "the substitution of a poorly bioavailable form in the regimen of a patient controlled on a fully available form would result in reversion to the depressed state."

I am gathering instances of 1) depressed patients who did not respond to initial treatment with a generic tricyclic antidepressant but did respond to a subsequently administered brand-name tricyclic antidepressant, and 2) depressed patients who responded to a brand-name tricyclic but relapsed when a generic form was substituted. I would be grateful if my fellow psychiatrists who have had patients adversely affected by treatment with a generic tricyclic antidepressant would share their data with me.

FRANK J. AYD, JR., M.D.
Baltimore, Md.

Bromism from Over-the-Counter Medications

SIR: In his article "Bromism: Alive in Well" (July 1978 issue) Ira Brenner, M.D., pointed out that neuropsychiatric symptoms can occur below the normally accepted cut-off point of 50 mg/100 ml. We have reason to believe that bromism is not a rare disorder and have recently seen a case here. The patient, a 33-year-old man, was admitted for treatment of a depression that had been intermittent and manifested by sudden episodes of his feeling "very depressed," which lasted minutes to hours and occurred several times weekly. The patient had had these episodes for approximately 3 months and had been treated previously in a hospital. He is now an outpatient and has been given adequate doses of tricyclic antidepressants, with no relief. The patient reported a history of diminished libido and decreased concentration over the previous 2 years, although he denied sleep or appetite disturbance, suicidality, delusions, or hallucinations. He was unable to perform his work as an engineer and was socially withdrawn from his premorbid life style. He had poor impulse control that was worse when he drank alcohol, which he used to self-medicate for his subjective depression.

Psychological testing showed that the patient had a verbal IQ of 124 and a performance IQ of 106. The Graham Kendall Memory Test reflected normal perceptual motor abilities. The Bender-Gestalt was normal. Neurological and physical examinations were within normal limits. All laboratory tests, skull series, and CT scan were within normal limits except for a bromide level of 42 mg/100 ml. The patient was kept in the hospital (medication-free) for 1 month, with resolution of his episodes of depression. The bromide level gradually decreased to 30 over 2 weeks. The patient did not evince any of the impulsivity that had been reported prior to admission, and his ability to concentrate appeared to return by the time of discharge. Short-term follow-up at 1 month has disclosed the absence of his previous symptoms on no medications.

The patient reported that he had been taking an over-the-counter sedative but was unable to remember the name; the bromide probably came from this source. After his recovery, discussion with the patient revealed that he had been feeling anxious about his job performance and started taking these over-the-counter preparations, which did not initially relieve his anxiety; he increased the frequency of ingestion as a way of attempting to get control of his anxiety. This behavior had continued for 7 or 8 months before admission and had not been detected by previous psychiatrists or in previous hospitalizations over the six months before admission. Contact with the patient's previous physicians revealed that they had appropriately asked him about any medication he was taking at the time they evaluated him, but the patient reports that he did not consider over-the-counter medications as the type of medication the physicians were asking about. This suggests that in all evaluations patients should be directed to inform the physician about all pills they are ingesting (including over-the-counter medications, vitamins, oral contraceptives, etc.) since the patients may not relate these to the reason they went to see the physician.

ROBERT H. GERNER, M.D.
Los Angeles, Calif.

Another Reason for Underutilization of Partial Hospitalization?

SIR: I would like to comment on the article titled "The Paradoxical Underutilization of Partial Hospitalization" by Edward B. Fink, M.D., Richard Longabaugh, Ed.D., and Robert Stout, Ph.D. (June 1978 issue). The authors conclude that the underutilization of partial hospitalization "appears to be largely attributable to the subjective discomforts of physicians and patients' families with this alternative treatment setting." The authors only briefly mention that there have been refusals of third-party payers to finance partial hospitalization treatment and do not seem to feel that it is important enough to include in their conclusions.

In a brief telephone survey of six major health insurance providers and state and federal medical providers I found that Blue Cross, Midland Mutual, and Medicaid (State of Ohio Welfare Department) will pay no percentage of partial psychiatric hospitalization; depending on the individual or group policy, Blue Shield and Travelers will pay no percentage of hospital bills and some percentage of physicians' bills; Prudential will pay some percentage; and Aetna will pay 50%–80% but does not cover meals. Medicaid (U.S. government for patients over 65 or disabled for 2 years or longer)

will pay no percentage of hospital costs but will pay physicians' bills. The results of this survey show that only two companies, Prudential and Aetna, will write some policies that cover a percentage of day hospital treatment. Blue Cross, Travelers, Midland, and most Blue Shield plans will not pay for partial hospitalization. I submit that the authors' conclusions that the underutilization of partial hospitalization is attributable to subjective discomfort on the part of the physician and patients to this alternative treatment are incomplete.

Another conclusion might be that it is an ethical response on the part of psychiatrists and psychologists not to have patients admitted to psychiatric facilities when insurance and family resources cannot cover the accrued bills (and it is rare to see patients pay for hospital services without the help of third-party payments).

CARL L. TISHLER, PH.D.
Columbus, Ohio

Dr. Fink and Associates Reply

SIR: Dr. Tishler's letter addresses a very important problem in partial hospital utilization; namely, that many health insurance providers do not pay for psychiatric treatment in a partial hospital setting. He contends that this is a more important reason for underutilization than those we emphasized.

In the day hospital described in our article, however, the lack of third-party payment for day hospitalization was not a problem. Prior to the establishment of our partial hospital program in 1973, extensive negotiations were completed between the hospital and Blue Cross/Blue Shield of Rhode Island, the major third-party payer in the state. On a trial basis, the insurance carrier agreed to offer its subscribers partial hospitalization coverage as an alternative to inpatient coverage. The agreement with the hospital included the condition that the hospital complete a treatment outcome study with 18-month follow-up data. The study has documented, to the satisfaction of Blue Cross/Blue Shield, that partial hospitalization is beneficial to patients and family members, as well as to the third-party payer. Partial hospital coverage now is a permanent feature of Blue Cross/Blue Shield contracts in Rhode Island.

Although the negotiations and the completion of the outcome study required extensive efforts on the part of the hospital administration and clinicians, it was judged that such efforts were worthwhile. Other psychiatric institutions have followed our early example, and many hospitals in the surrounding area discussed their specific third-party payer problems with us and learned of our experience. Several of these hospitals have by now successfully negotiated analogous arrangements with insurance providers. In contrast to Dr. Tishler's survey, our local Blue Cross/Blue Shield office reports that its affiliates in as many as 17 other states are providing coverage for partial hospital programs as of this date. (A complete listing is available on request.)

Dr. Tishler's letter also suggests that for ethical reasons clinicians should consider not admitting patients to partial hospital programs if third-party payers do not cover the cost of medical care. As documented in the introduction to our article (see references 1–4, p. 716), many clinicians and researchers contend that such a practice would not be in the best interest of patient care. Perhaps an alternative more productive than Dr. Tishler's would be to increase our efforts to educate the third-party carriers, as well as patients and their families, regarding the advantages of treatment in the partial hospital setting. The development of alternative clinical services which meet the needs of patients thereby would be fostered.

There is no doubt that the development of partial hospitalization programs presents many potential obstacles, as well as many significant ones. Our experience with Blue Cross/Blue Shield in this state, however, has been successful. We hope that our experience will continue to encourage the efforts of others.

EDWARD B[illegible]
RICHARD LONGABAUGH, M.D.
ROBERT S[illegible]
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Maladaptation in Prehistory

SIR: In "The 'Relief Effect': A Sociobiological Model for Neurotic Distress and Large-Group Therapy" (this issue) Marc Galanter, M.D., put forward a sociobiological model to explain vulnerability to neurotic symptoms. He argued that because affiliation with a cohesive group engenders a significant decline in distress, the adaptive value of this vulnerability may lie in assuring effective integration with a large social unit, thereby leading to a more adaptive social unit.

I recently put forward another hypothesis to explain human vulnerability to neurotic distress symptoms. My hypothesis is based on the notion that both adaptation and maladaptation are active processes which can be understood the concept of adaptive and maladaptive cycles. A maladaptive cycle, which includes neurotic distress, is based on the premise that failure may lead to failure. These ideas led to a hypothesis about the origin of neurotic maladaptation in prehistory. As natural selection operates on genetic differences, the two methods of warfare have magnified the impact of these genetic differences, thereby accelerating the weeding action of natural selection. This dual process would have accelerated the maladaptation of various animal species, including man.

Dr. Galanter's hypothesis and mine differ in that he refers to adaptive and maladaptive cycles, he refers to them in terms of a single continuum, the adaptive end of which overlaps with his is that of Price, who sees neurosis as playing an adaptive biological role (3). Both Price and Galanter view neurosis as promoting social behavior.

Dr. Galanter did not attempt to provide a sociobiological formulation explaining why very cohesive groups are more successful in contributing to a decline in neurotic distress. One knows that less cohesive families are more likely to generate neurotic distress symptoms in their offspring; one can differentiate between peer or family groups that are functional and those that are dysfunctional in terms of the support they provide for the members of the group. What is the sociobiological function of this difference? It is well known that when groups compete, those who win tend to show euphoria and those who are defeated tend to exhibit depression and anxiety. This even when representatives of groups compete. I would suggest that in prehistoric times the morale or cohesiveness of a group would have been a rough index of the biological fitness of the members of the group. The well-integrated peer group would have been able to provide greater support to their members than would the less well-integrated group.

This would be an illustration of inclusive fitness. For example, if the members of the group were on the whole not well fitted for survival, they would have been less successful in intergroup competition than those who were relatively well fitted for survival. In view of their poor morale they would have been less able to provide support for each other, so that the members of the group would therefore have been at an increased disadvantage. This would have tended to lead to a more rapid elimination of the latter group as a whole. On the other hand, groups who were better adapted would have been able to provide their members with more support, so that their chances of survival would have been increased. By promoting the rapidity of natural selection, this mechanism would have accelerated the evolution of early man.

The sociobiological model just described enables one to postulate an ideal family or peer group system which in a pretechnological age would have reflected a high level of biological adaptation. The structure of the ideal system in the past would have been designed to promote the adaptation and ultimately the survival of the individual family members.

It is my contention that sociobiology can illuminate our understanding of why some groups are well geared to promote Dr. Galanter's "relief effect" and that a delineation of the structure of these groups could also provide clinical guidelines in group and family therapy. In conclusion, it is noteworthy that Galanter, Price and I have independently arrived at related conclusions about the relevance of sociobiological principles in furthering our understanding of human neurosis.

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Dr. Galanter Replies

SIR: A useful perspective on the biological basis of man's psychiatric problems may lie in his evolutionary past. In this regard, Dr. Sloman raises an important issue by pointing to the availability of alternative sociobiological hypotheses to explain the adaptive value of neuroticism. His proposal also reflects the value of "crucial" experiments, in which alternate possible outcomes may exclude one or another competing hypothesis. In sociobiology of the higher species, however, difficulty in experimental manipulation means that we can, generally, only examine experiments of nature when they occur. In the study I reported, such an examination was made of a charismatic sect in an attempt to disconfirm the proposed "relief effect" hypothesis; however, the hypothesis was supported by an analysis of the empirical data.

Dr. Sloman has suggested that the evolutionary value of neuroticism lay originally in promoting more rapid elimination of poorly adapted individuals. This would presuppose significant ecological pressure in this direction, most likely due to competition for limited resources and expendability to the group of some of its adult members. The fact that neither experimental support nor rejection is available for Dr. Sloman's hypothesis reflects the difficulty in obtaining relevant naturalistic data but does not detract from the fact that any hypothesis must ultimately stand the test of empirical examination.

In addition, any mechanism proposed to persist on an evolutionary basis must be scrutinized with regard to its cost/benefit ratio in relation to group survival. This allows us to apply a measure of indirect evidence, even with our limited knowledge of the social ecology of early man. Using this approach, we are better able to evaluate hypotheses on human sociobiology on their a priori merits.

In applying this principle to Dr. Sloman's proposed mechanism, its adaptive value would hinge on the loss of the adults who were debilitated to the point of performing so little useful work that they detracted from the inclusive fitness of the group. On the other hand, this mechanism also implies an increased risk of mortality even to adults temporarily or partially disabled by neurotic symptoms. An increase in the loss of such viable individuals after infancy, however, would significantly detract from the adaptive capacity of any primate species, since a large portion of available labor is dedicated to prolonged nurturance and training of progeny. This prior investment means that adult mortalities come at considerable cost to the group. Indeed, it has been pointed out (1) that, for this very reason, in species with substantial parental investment in the young, mortality is generally concentrated in the earliest ages. I would therefore think that the evolutionary role of neurosis is best explained in a way which does not imply enhanced fitness based on increased adult mortalities.

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Neurobiology and Dreams: Further Queries

SIR: I read with great interest the articles by J. Allan Hobson, M.D., and Robert W. McCarley, M.D., on "The Neurobiological Origins of Psychoanalytic Dream Theory" and "The Brain as a Dream State Generator: An Activation-Synthesis Hypothesis of the Dream Process" (November and December 1977 issues). I would like to comment on the psychological implications of their work from the viewpoint of recent contributions by Piaget.

The authors assume, as did Freud, that total isomorphism exists between the simultaneous physiological and psychological events of dreaming. Piaget (1) argues that the isomorphism between these systems is at best "partial," the physiological events being homeostatic whereas the psychological events are characterized by progressive equilibration, or "homeorhesis." Piaget (2) says that mental imagery is not "perceptual," in the sense of being an internally driven continuation of perception, but "quasi-perceptual," i.e., an internalized imitation of perception that is closely related to cognitive development as a whole.

Until REMs can acquire some degree of direction and organization as a result of their interaction with a sufficiently adapted intelligence capable of mental imagery (at the completion of the sensorimotor stage), there is no reason to assume that children (or cats, or opossums, in which REMs are even more prominent) experience the hallucinoid images synonymous with dreaming in the adult. Drs. Hobson and McCarley state that hallucinoid dreaming is the psychological concomitant of desynchronized (D) sleep; Piaget would say that activation, as undirected and unorganized motor excitation resulting from the autonomous process of nervous detoxification, exists before synthesis into dreams in early childhood and together with it only when the child develops mental imagery.

Dreaming assimilates reality to the ego, invariably distorting it (3). In psychoanalytic theory, this is called condensation, displacement, splitting, and symbol formation, suggesting that it is the work of the censor. Drs. Hobson and McCarley suggest that these phenomena are genotypically determined by the purely physiological, homeostatic activity of the brain stem. For instance, change of scene and time compression may be direct results of physiological changes in the generator system, but Piaget has shown that symbol formation is not explained on this basis (3). Like Hobson and McCarley, Piaget finds no need for repression, or for a censor, whose purpose is to disguise. Distortion in the symbols results from the excessive assimilation inherent in symbol formation. If there were no accommodation at all, however, there would be no images and certainly no conscious awareness of them.

In summary, dreaming seems to result from the necessary interaction of two relatively autonomous systems, the physiological activation system of the pontine reticular formation and the psychological cognitive system, which has a widespread neurological substrate. This explanation of dreaming, which is both modern and plausible, avoids reducing the psychological to the physiological while recognizing the partial isomorphism that exists between the two levels of organization.

Almost paradoxically, this composite dream theory permits one to salvage the psychological, wish-fulfillment part of Freud's dream theory if one extends the definition of "wishing" to include the free assimilation of reality to the ego.

Again, I applaud Drs. Hobson and McCarley for providing an important element in understanding dreams.

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SIR: I would like to add to the commentary on the fascinating hypothesis set forth by Drs. McCarley and Hobson. Relevant to their activation-synthesis hypothesis of dream process is a recent paper by Gazzaniga and associates (1), who demonstrated in a callosal-sectioned patient two major, independent language systems in the right and left hemispheres. The right hemisphere, although not [illegible] pressive speech, could comprehend nouns [illegible] possessed the motor engrams necessary to [illegible] and pictorial commands. Of particular interest [illegible] ner in which the left hemisphere reacted to [illegible] sponse to commands presented to the right h[illegible] trials requiring that the right hemisphere initi[illegible] the left half brain was forced into a positio[illegible] responses of unknown origin. Yet in each tri[illegible] sphere provided a reasonable explanation fo[illegible] responses. The verbal system of the left he[illegible] fore attributed causes to the behavioral re[illegible] right hemisphere. For example, when the co[illegible] was flashed to the right hemisphere, the subje[illegible] back of his head with his left hand. When aske[illegible] ize the response, the patient (i.e., the left he[illegible] bally responded "itch." Thus the left hemisph[illegible] the response and attempted to characterize it[illegible]

This neural process, the mechanism of whic[illegible] can be viewed, in principle, as akin to the [illegible] brain stem stimuli by the forebrain in the drea[illegible] brain, in both cases, attempts to synthesize [illegible] cepts from incomplete data provided by p[illegible] Furthermore, it is probable that both synthes[illegible] are profoundly influenced by specific psych[illegible] and events.

This process of attribution by the verbal sys[illegible] hemisphere in waking and the elaboration of bi[illegible] uli during dreaming represents a major advan[illegible] understanding of isomorphisms between bi[illegible] processes.

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DENNIS S. C[illegible]
New Ha[illegible]

SIR: Drs. McCarley and Hobson showed [illegible] worked the nineteenth-century discovery [illegible] servation of energy into his psychological mo[illegible] imagined flow rather than transduction of e[illegible] central nervous system. They observed that c[illegible] be cancelled in Freud's model and that Freud's [illegible] cal model is his physiological model in disguis[illegible] of "mind-body isomorphism."

I doubt that Freud simply incorporated neu[illegible] into psychological theory, as the authors clai[illegible] of clinical studies helped him to generate co[illegible] like symbolism, repression, and the unconscio[illegible] ed in "Preliminary Communications" (1) and [illegible] Hysteria" (2), which antedated the "Project" [illegible] tories were the substrate for Freud's view of [illegible] symbolic reminiscences of traumatic events. P[illegible] toms disappeared when repressed memories er[illegible] this start, Freud failed to see the importance [illegible] (cancellation type) as part of his neuron model. [illegible] Exner had mentioned it. Freud's clinical we[illegible] lured him away from the idea of inhibition beca[illegible] traumata failed to produce symptoms. Rather [illegible] model shaping his psychological model, Freud's [illegible] cal insights about symptom relationships may [illegible] him away from a more workable physiology.

The concept of psychic energy has been doubtful for years. No one has found this entity or measured it. Still, energy must be part of every working model—a mind model is no exception. Freud abandoned his search for connections between nerves and dreams but kept the psychological model. Drs. McCarley and Hobson have proposed a newer physiological model, asking for major changes in analytic theory. The pons governs dreams in their model. They suggest that dream content comes from cortical efforts to make "the best of a bad job" served up by pontine circuits.

Is mind-body isomorphism really any more applicable today than it was in 1895? Drs. Hobson and McCarley describe the way pontine neurons turn dreams on and off, but this describes the clock, not the time-teller. Dream content may be far removed from individual nerve functioning. For example, the authors suggest that the sensation of sluggish motion in a chase dream is adequately explained by perception of pontine motor repression; invoking a wish to be caught is superfluous. However, most dreams lack chases, and some chase dreams must lack retarded motion sense. How could automatic circuits let through a sense of motor repression only some of the time?

Freud's energy transmission model does not work as physiology, but the psychological model has much clinical affirmation. The principle of mind-body isomorphism must be applied with great care. Every modeler will want to use it sooner or later. However, pontine activity and dreaming may be no more related than a brick is related to the architecture of a brick building. New knowledge about one does not demand an automatic change in the other.

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Drs. McCarley and Hobson Reply

SIR: We are pleased to continue the discussion of our papers (see Letters to the Editor, May 1978 issue). We are especially grateful for the presentation of additional data and theories relevant to our hypotheses. We will group our comments into three areas.

1. *Unifying physiology and psychology with information from bisected brains.* The paper by Gazzaniga and associates (1) is indeed of immediate and important relevance to our activation-synthesis hypothesis. These authors presented tests to the separately functioning hemispheres of a patient with a complete "split brain." When the right hemisphere was required to initiate motor acts, the left half brain was forced into a position of observing responses whose source was unknown. The left hemisphere proved particularly adept at providing an explanation for the responses, as if the response had been initiated therein, i.e., the left hemisphere conscious system accepted the response as its own act.

The activation-synthesis hypothesis proposes that one of the important elements of dream construction is the activation of motor pattern generators during desynchronized (D) sleep, including those normally responsible for many "automatic" motor behaviors such as walking and running. Although the paralysis of the output neurons during D sleep prevents the execution of these acts, the rest of the brain still receives the same information as if the act had occurred. The new information is that synthetic efforts of the conscious system may, in fact, structure this kind of information as if it had been initiated in the usual way. We have recently given several examples from dreams of how information from such motor pattern generators may be smoothly incorporated into dreams (2), and the above data show how the conscious system (i.e., subjective awareness) may come to accept this activity as its own.

A recent report on the dreams of patients with commissurotomies (3) is also relevant because it discusses a hypothesis attributing the distinctive form and structure of dreams to their being primarily a right (nondominant) hemisphere phenomenon. In this view, dreaming takes on many of the characteristics of the visual and form-oriented right hemisphere rather than the verbal left hemisphere. If visual dreaming were mediated in the right hemisphere, patients with commissurotomies should be unable to make a verbal report of the visual components of their dreams, since the disconnected verbal left hemisphere would have no access to the visual experience of the right hemisphere. This theory was not supported; characteristic and usual reports of visual dreams were elicited during D sleep periods from a subject with complete callosal and commisure sections.

2. *Isomorphisms.* Although developed independently, our use of the term "isomorphisms" stems from the same roots as Piaget's: the desire for an appropriate formalism to describe the relationship between biology and psychology, brain and mind. Our work is, however, at a more modest and empirical but more easily verifiable level than Piaget's. For example, we seek isomorphisms between dream sensations and the activation of various sensory systems, whereas Piaget seeks correspondences at a different level, e.g., between order structures in cognitive processes (e.g., the ordering of action sequences and perception) and order in biology (e.g., the ordering of the DNA code) (4, pp. 164–165). At the most abstract level of isomorphism, Piaget is working on the hypothesis that the logico-mathematical thought processes are developed by reflection and abstraction on the inherent organization of action and peripheral processes which, in turn, are derived "from nervous coordinations, and so on back to the most widely generalizing functions of life" (4, p. 342). We think the question of whether isomorphisms are partial or complete is open, but we would note that Piaget's emphasis on partial isomorphism may reflect the high level of abstraction at which he is working. We concur with Piaget that reductionism is not the way to relate brain and mind and that it is both important and necessary for biologists and psychologists to collaborate in the future.

We emphasize again that the principle of isomorphism, by definition, does not mean a "simple incorporation" or transformation from biology to psychology (or vice versa). We point to our earlier comments (Letters to the Editor, May 1978) on the strategic choice of models of phenomena. These comments also apply to the query about the direction of the mutual influence between Freud's psychology and his physiology. The critical point is that had Freud possessed our current physiological knowledge, he could not have conceived his physiological model as he did, which would have had

very important consequences for the isomorphic psychological model. It is important to note that Freud did not contradict any accepted physiological principle known in the 1890s in his model: inhibitory elements in the nervous system at that time were a tentative, developing, and unproven concept. Freud had distinguished company, including the great Golgi, in not including inhibitory elements in a nervous system model. Our conclusion is that the isomorphism principle works quite well, but one must continually alter either physiological or psychological models when data critical to one or the other isomorphic domains are proven wrong. The problem is that in early psychoanalytic theory the isomorphic psychological model was not altered as knowledge of physiology progressed (see our comments below on psychic energy).

3. Dream form and dream content. First, some metaphorical carpentry: we are not talking about dream "bricks," but dream "form," and the proper metaphorical contrast would be architectural form versus architectural content. On this subject of form and content Robbe-Grillet (5) commented,

> what makes the cinema an art [is that] it creates a reality with forms. It is in its form that we must look for its true content. The same is true for any work of art, of any novel for instance: the choice of a narrative style, of a grammatical tense, of a rhythm of phrasing, of a vocabulary carries more weight than the actual story . . . the initial idea for a novel involves both the story and its style; often the latter may actually come first in the author's mind, as a painter may conceive of a canvas entirely in terms of vertical lines before deciding to depict a skyscraper group.

Our contention is that emphasis on dream form goes to the heart of what is dreamlike in dreams. We are in accord with Freud that the sensing of the physiological phenomenon of dream paralysis accounts for the ubiquity of dream reports of paralysis. Such sensing of physiological data can be observed in other systems, as in this sleep laboratory dream report example (2).

> I was spinning, my body was spinning around. The circus performers put the bit in their horses and they spin around. The trapeze was spinning like that. Hands at my sides and yet there was nothing touching me. I was as nature made me and I was revolving at 45 rpm record speed. Had a big hole in the center of my head. Spinning, spinning, and spinning and at the same time orbiting. Orbiting what I don't know. I'd stop for a second, stop this orbit and spinning.

Here, both form and content are dominated by the sensation of whirling in space, probably derived from the sensing of vestibular activation, a phenomenon especially prominent during D sleep. One might ask why all of our dreams are not of paralysis and spinning or any sensation whose physiological bases are present during all D periods. First, one may point to genuine physiological differences from one D sleep period to another in the degree of activation. Second, the phenomenon is akin to that of conscious attention in general; it is common to have a continually present physiological input that is only intermittently or episodically noted. One may have a high temperature but be aware only episodically of feeling hot. We expect that a more precise answer to this query will be found in further investigations of the physiology and psychology of attention, a developing but incomplete area. We emphasize again that the distinctive features of dreams as contrasted to waking experience are isomorphic to the physiology: The preeminence of visual imagery in dreams is isomorphic to the preeminence of visual system activation, and the presence of the striking sensation of paralysis or spinning (or floating) is characteristic of dreams, not of waking, and is isomorphic to characteristic of sleep physiological events that are rare in waking.

4. Psychic energy. We believe this concept, more than any other, illustrates the need for continual interchange between biology and psychology, between brain and mind theory. Advances in physiology since 1895 have made untenable Freud's concept of "nervous energy," which was isomorphic to psychic energy. Unfortunately, the corresponding concept of psychic energy has been left unmodified. We are extremely skeptical that outmoded physiological theory can persist as an isomorphic base for psychological theory. We know of no case where an outmoded physiological theory serves as a respectable and useful psychological theory. There is every indication that many psychoanalysts are aware of the problems with psychic energy, as we have noted in previous communications. Our own notion is that psychology can do well without the concept of psychic energy but cannot do without a theory of motivation. We think motivational theory is strongest when related to biology and see the source of the particular vigor of psychoanalytic theory in its biological roots and its correspondence to interest in motivation—an interest that, until recently, was lacking in traditional academic psychology. We see future growth coming through efforts to link physiological and psychological processes thought to be critical to motivation.

5. Dream process and dream symbol. Piaget's descriptive of the dream process in *Play, Dreams and Imitation in Childhood* (6) has some intriguing relationships to activation-synthesis hypothesis of dreaming. Piaget was writing 1951 and therefore could not have known that mnemonic sensorimotor centers are directly and intensely activated from pontine source during D sleep. Yet Piaget's discussion of the fate of external stimuli and how they are formed or cleared might be taken as paradigmatic for what might happen to internally generated stimuli. Piaget notes that external stimuli impinging on the body are often not represented in the dream as being part of the dreamer's body because the dreamer's mental state is analogous to a baby in the early or not stage of development, i.e., lacking consciousness of the "ego" in the sense of being unable to distinguish self from nonself, inside from outside. (Incidentally but important, Piaget's anecdotal observations about the fate of external stimuli and their frequent representation in characters or objects outside the dreamer have been confirmed in a number of controlled sleep laboratory studies.) In the dream, as in the sensorimotor stage, there is a lack of coordination between the tactile-kinesthetic and the visual system, and since the dreamer cannot identify the body sensation as his own, he "seeks it were a visual correspondence, but since he is motorically passive and is even unaware that he is involved. In the inevitable constructing images, he has recourse to an "assimilation which offers some point of resemblance" (p. 32). Thus for Piaget, much of the visual material of the dream may derive from innate symbolic representation.

Obviously, in view of what is known today about the intense, direct, but internal activation of the visual system, it would appear that Piaget's view that the dreamer "can do nothing" is in need of revision. We do, however, note that the tendency of many areas in the brain to be reciprocally activated during D sleep may further the process of symbolic representation so eloquently described by Piaget.

There are other interesting correspondences and links between Piaget's work and what may be happening in dreams. For example, the process of coordinating sensorimotor schemata (both in the sense of Piaget's theories and in the physiological sense) with the visual world is extremely important. This is an area of intense investigation in physiology and, interestingly enough, much of this sensorimotor and visual coordination is being traced to brain stem neuronal activity. For example, some of the motoric "action schemata" (simple motor acts such as walking) described by Piaget have been shown to have a clear physiological substrate, and the concept of a motor pattern generator for these acts is a most useful one. Some of these physiological motor pattern generators may bear an interesting isomorphic relationship to the cognitive representations that the infant in the sensorimotor stage is working to develop. Piaget speaks of play as useful in development and of dreams as often showing processes cognitively analogous to those in play. Viewed from the physiological side, it may be that the activation of motor pattern generators and the visual system in D sleep is functionally important in furthering development and coordination of these systems, just as Piaget sees dreams as important in furthering the development of possibly isomorphic cognitive systems. It is clear that this is a rich area for investigation of isomorphic links between physiology and psychology.

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Medical School Admission Screening by Psychiatrists

SIR: Psychiatrists have not been excluded from medical school admission screening because of the newness of the art, the isolation of the practitioners, or the advocacy of the practitioners, as Henriette Klein, M.D., and Emily Mumford, Ph.D., suggest in "The Bent Twig: Psychiatry and Medical Education" (March 1978 issue). If psychiatrists have been excluded, it is because of their medical colleagues' embarrassment over the extravagance of their claims of expertise and the dearth of data supporting such claims. Both of these faults also affect Drs. Klein and Mumford's paper.

The authors suggest that because medical students are becoming less sensitive, psychiatric and psychological testing should be used to screen applicants for medical school. Such screening is almost surely made illegal by the Federal Rehabilitation Act of 1974. Drs. Klein and Mumford essentially concede this point when they acknowledge that such testing is vulnerable to legal challenges, as grade points are not. I am currently representing one medical school student denied readmission after psychiatric testing. That vulnerability is not a simple prejudice against psychiatrists but a recognition by courts that no person should be denied medical school admission on the basis of decisions that are demonstrably unreliable and invalid and therefore subject to misuse.

Perhaps more important for readers of the *American Journal of Psychiatry*, Drs. Klein and Mumford totally ignore the substantial number of psychiatric studies collected by Professors Alan Stone and Alan Dershowitz which demonstrate quite clearly that psychiatrists and psychologists are "not especially qualified to prognosticate the success, failure or risk of a college, graduate school or job applicant" (1). It is worth noting that the views expressed by Professors Stone and Dershowitz were officially endorsed by the American Orthopsychiatric Association.

Drs. Klein and Mumford provide absolutely no data that contradict this conclusion. Although their paper is filled with references, only one is even arguably relevant to their proposal that psychiatrists screen medical school applicants, and it apparently concludes that psychiatrists can tell—after the fact—who has committed suicide.

Although the goal of more humane and sensitive physicians is one we all support, psychiatric screening of medical school applicants will not achieve this goal. I suspect it would instead deprive the medical profession of many sensitive and humane people who would have been exactly the type of doctor that Drs. Klein and Mumford want.

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An Explanation for Seasonality of Births in Schizophrenia

SIR: A large American study of season of birth and mental disorder was recently published in the *Archives of General Psychiatry* (1), and a similar study was published in the *British Journal of Psychiatry* in 1974 (2). Both studies showed a highly significant excess of births in the first quarter of the year for people who later became schizophrenic. The excess was approximately 8% for January, February, March, and April. The American study corrected for small differences in month of birth for the general population and provided a comprehensive literature review. Both articles listed a number of explanations for this finding, but they seem to have missed the simplest and most plausible explanation.

The schizophrenic patients in both studies were born between 1920 and 1955. During that period, a large number of schizophrenics of childbearing age were institutionalized. A schizophrenic psychosis at that time probably meant a mental hospital admission lasting from at least three months to two years. Although the sexual activity of hospitalized schizophrenics is probably less than that of the nonhospitalized population, it certainly is not nonexistent. Male and female patients were on different wards but were permitted ground

privileges in good weather. It is well known to the staff of most mental hospitals that in the private areas of the grounds a fair amount of sexual activity takes place in good weather. In such cases, given the distribution of diagnoses in hospitalized patients, at least one of the two procreating adults, often both, would have had a schizophrenic disorder.

The excess births of schizophrenic patients in the months of January, February, March, and April could easily be accounted for by the offspring of only a small percentage of the thousands of hospitalized patients. If conception did indeed take place during the warm months (June, July, August, and September) then a transmission rate of only 3%–5% would account for the excess number of schizophrenics born the next winter and early spring.

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Research on Attitudes Toward the Mentally Ill

SIR: In their article "Fear of the Mentally Ill: Empirical Support for the Common Man's Response" (October 1977 issue) John M. Lagos, Ph.D., and associates provided admirable, concise discussions of the literature on the dangerousness of the mentally ill and the implications of public attitudes for community treatment. One wishes, however, that the original empirical study presented in the article had been performed more rigorously.

First, descriptions of violent activity were based on the admissions records of 400 patients, apparently with no checks on their validity in cases where the alleged violent behavior did not occur in the presence of the admitting physician. Then, assuming without demonstrating that the notes were reasonably accurate descriptions of actual behavior, the authors concluded that perhaps the public has reason to fear mental patients as a class because a significant percentage of the sample was found to have been violent before admission to a hospital. However, there was no control group, an elementary necessity in a study that attempts implicitly to compare one class of persons (the mentally ill) with another (normal individuals). The degree to which the public fears any group must depend in part on base rates, and the claim that the public may have reason to fear the mentally ill is an unwarranted interpretation.

More than a third (36%) of the mental patients committed one form of violence or another, but an even higher percentage of nonpatients may also behave violently. If so, the appropriate conclusion would be that all of us have great reason to fear many people and even more reason to fear nonpatients than patients.

Studies on the dangerousness of the mentally ill have immediate relevance to the politicized debates about civil commitment. At a time when psychiatry is often attacked as being unscientific and insufficiently concerned with civil liberties, this study can only add power to the critiques. One wishes the authors had added a control group or simply limited the paper to a fuller review of the literature [illegible] of its implications.

STEPHEN J. MORSE, [illegible]
Los An[illegible]

Dr. Lagos Replies

SIR: Dr. Morse points out that the design [illegible] not include a check on the validity of the re[illegible] acts found in the hospital charts. This is true [illegible] tutes a definite limitation that further studies [illegible] to avoid.

Dr. Morse also notes that no control gro[illegible] show whether violent acts are equally preva[illegible] al population. Once again he is correct. How[illegible] suggest that patients were more violent than [illegible] ulation. We categorically stated, "These [illegible] substantiate the proposition that ex-mental p[illegible] violent than the general population."

It seems to me that Dr. Morse has no[illegible] What we tried to say was that the research [illegible] so narrowly focused that the twin issues of [illegible] and community fear have been denied full sc[illegible] We did not claim to have settled anything. R[illegible] were assembled to illustrate the inadequacy [illegible] paradigm for research on patient violence [illegible] new approach to the education of communi[illegible] lence among mental patients is an issue.

Dr. Morse implies that our article reflect[illegible] concern for the civil liberties of psychiatr[illegible] doubtedly, the interests of patients will be[illegible] understanding them better. Our work has [illegible] dimension to the study of violence amon[illegible] tients as well as to the education of comm[illegible] patient violence. It is my belief that this [illegible] that in the long run this work will contrib[illegible] liberation than to the confinement of psych[illegible]

JOHN M[illegible], [illegible]

Peptidergic Influences: Tip of the Iceberg?

SIR: I would like to make a few comm[illegible] the excellent article "The Opiate Recepto[illegible] Like Peptides in the Brain" by Solomon [illegible] (June 1978 issue). Dr. Snyder calls partic[illegible] somewhat surprising density of enkepha[illegible] rons in the globus pallidus, along with evid[illegible] way arising from cell bodies in the caud[illegible] terminals in the globus pallidus release [illegible] ronal populations in that site. The caud[illegible] globus pallidus are usually viewed some[illegible] regulators of motor behavior, which seems [illegible] the observation that the globus pallidus c[illegible] kephalin than any other part of the brain [illegible] zance of the almost inextricable relations hip[illegible] and emotion (1–3), it is apparent that such [illegible] pendent on and coordinated with a whole [illegible] states, such as attention, motivation, pl[illegible] pleasure.

There is good evidence that the extrap[illegible] shares rich connections with the neocortex [illegible]

key limbic structures involved in these states of the CNS. It is known that neuronal output of the corpus striatum to brain stem extrapyramidal neurons maintains a tonic inhibitory influence on them, suppressing their interaction on rostral thalamic and neocortical sites of action. This striatal tonic influence is funnelled to the brain stem, thalamus, and overlying cortex through the globus pallidus, the site of rich enkephalin transmitter release, as indicated by Dr. Snyder. The striatal outflow of suppressor type is enhanced by cholinergic synaptic influence and diminished by dopaminergic influence on an interneuronal system in the caudate putamen. It would make sense to have this striatal output modified in the pallidum by a peptidergic influence that lends, perhaps, expressive shape to motivated and emotionally determined motor behavior.

The corpus striatum, rich in enkephalin-containing neurons, may also play a role in neocortical arousal and attentional processes by means of enkephalin release in the outflow station, the globus pallidus. Administration of L-dopa to animals produces EEG activation and signs of behavioral stimulation in doses and time course correlating with increased levels of dopamine rather than norepinephrine. Activation of dopaminergic nerve terminals touching on the caudate putamen is believed to inhibit them, resulting in reduction of their suppressor influence on globus pallidus neurons, and to bring about indirectly, through reduction in a diffuse thalamic recruiting system, cerebral cortical arousal that facilitates attention and motivated behavior. That an enkephalin influence in the globus pallidus appears to be a rich one might suggest that an important peptidergic neurotransmitter system needs to be taken account of in addition to the better known influence herein of dopamine, acetyl choline, and γ-aminobutyric acid.

That the extrapyramidal system exerts an effector influence both on cerebral cortical and older cortical centers in the limbic brain and is in turn influenced by them appears to be a concept growing more clear through current neurobiological studies. The function of peptidergic systems, along with other neurotransmitters and neuromodulators, in information processing in such neural assemblies will be of great interest as data continue to be developed.

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WILLIAM F. KIELY, M.D.
Los Angeles, Calif.

Dr. Snyder Replies

SIR: I certainly agree with Dr. Kiely that the well-known association of the corpus striatum with regulation of motor behavior does not mean that enkephalin in these structures does not have to do with emotional behavior. Indeed, the embryologic development of the corpus striatum closely apposed to the amygdala in virtually a single structure argues strongly for some contribution of the corpus striatum to feeling states. At the same time, one should not focus so strongly upon the traditional role of opiates in pain perception and affective regulation as to neglect other possible roles of enkephalin systems. John Hughes and Hans Kosterlitz specifically selected the term "enkephalin," derived from the Greek meaning simply "in the head," in order to free us from prejudices about the functions of this new neurotransmitter peptide. High doses of opiates and opioid peptides do produce muscular rigidity in animals quite similar to that produced by neuroleptic drugs and perhaps resembling the rigidity of Parkinson's disease. I suspect that enkephalin neuronal systems now being mapped in diverse areas of the brain may be involved in a large number of brain functions and that we have only begun to appreciate some of the most obvious.

SOLOMON H. SNYDER, M.D.
Baltimore, Md.

Concurrent Validity of DACL Questioned

SIR: I was happy to have my attention drawn to the article "The Concurrent Validity of the Depression Adjective Check List in a Normal Population" (May 1978 issue) by Roger Christenfeld, Ph.D., Bernard Lubin, Ph.D., and Maurice Satin, Ph.D., because I was the Principal Investigator of record on grant MH–19966, which was partially credited in the footnote for support of this investigation.[1] (Grant MH–20681 also supported the study).

The authors restate the title's assertion toward the end of the article: "The overall findings of this study support the concurrent validity of the DACL(E) as judged by psychiatrists' ratings of depressed mood. . . ." A hasty reader might think that this statement meant that the DACL score will tell you approximately the same thing regarding a person's mood as you would learn if a trained research psychiatrist interviewed the individual and gave you a rating. Such an interpretation would be wrong.

The two research psychiatrists whom I selected and worked with rated 7 of 309 subjects at level 3 (mood depression as severe as that sometimes seen in patients being treated for depressions). Four of these had DACL scores of 10 or more, but so did 12 subjects who showed no evidence of depressed mood (and so did 21 people whom the psychiatrists rated at level 1 or 2, both less severe than level 3). By lowering the cut–off score to below 10, more of the level 3 classification rates get even higher. Using a score higher than 10 produces even more false negatives. These high misclassification rates lead me to conclude that the DACL is a poor indicator of what the psychiatrists were observing, a conclusion that does not agree with the assertion of validity stated by Christenfeld and associates. How can this be?

My figures on the high rates of misclassification that the DACL score produces could be anticipated from the figures reported by Christenfeld and associates. The mean test scores at the 4 mood levels differ by less than a standard deviation in table 2. The correlation coefficient of 0.35 reported in table 1 is statistically significant, but I do not think it signifies "validity." I would want a correlation coefficient to be about twice this size before I would judge an index

[1] I was also Director of the Psychiatric Epidemiology Research Unit in the New York State Department of Mental Hygiene, the resources of which were essential for the conduct of this study. Maurice Satin was a predoctoral trainee in psychiatric epidemiology at the time these data were gathered. Dr. Christenfeld was a member of the research unit team.

measure to be a good indicator of a criterion measure.

Indeed, I find it remarkable that the correlation coefficient is so low since this self-rating scale offers a series of adjectives to be checked if the subject feels they are suitable replies to "How do you feel today?"—adjectives such as blue, hopeless, dejected, forlorn, glum. The list reads like a thesaurus entry of synonyms for the word depressed. These same words entered into the verbal give and take between psychiatrist and subject during the interview. Many of the same verbal signals were thus used in both appraisals and I expected a closer relation than we found. After all, there are only a finite number of words in our language with which to communicate a depressed mood. I think Christenfeld and associates were right to use the psychiatric rating as a criterion because the trained observer had two advantages over any paper and pencil test. He could ask the subject to elaborate on the meaning of each word used, and he could observe nonverbal signals such as tone, manner, posture, and facial expression.

I think the authors put too much emphasis on the statistical significance of the calculated correlation coefficient between the DACL score and the psychiatric rating. I believe that only capricious reactions to the words used in both contexts could have produced zero order correlations of no statistical significance. For that kind of finding I would suggest an article titled "Mental Hospital Staff Used Affective Terminology Chaotically." That simply was not the way the helpful staff at Norwich Hospital went about the task of telling us about their current mood.

I am preparing a more accurate report of this investigation for publication in which I will point out what inferences I think can be drawn from the data obtained.

ERNEST M. GRUENBERG, M.D., D.P.H.
Baltimore, Md.

Dr. Christenfeld and Associates Reply

SIR: We were interested to read Dr. Gruenberg's opinion, although we find his letter puzzling on the face of it. Our article presented the correlations in a hospital staff sample of the DACL with other depression measures, most pertinently with psychiatrists' judgment of depressed mood. Gruenberg submits "I would want a correlation coefficient to be about twice this size. . . ." He has, of course, the prerogative of each reader to set his own level of acceptable correlation; whether one infers that the results are validating or invalidating is akin to calling a glass half-full or half-empty.

There is, as all know, a vast difference between a psychiatric interview and a pencil and paper test even when both focus on the same trait. The field of personality assessment is not known for high reliability coefficients, and we continue to regard the correlations we found as supportive. We do not, however, as Gruenberg suggests, necessarily view imperfect correlations as flaws in the questionnaire. A psychiatric interview gains flexibility and insight at the cost of introducing the vagaries of interpretation and interpersonal dynamics. For instance, in this study it may be plausible that the correlation of DACL and psychiatric rating is lower for females partly because the male psychiatrists interpreted the responses of women with some bias. Lacking ultimate criteria we rely on concurrent validation. All four of the instruments we used, as well as the psychiatric ratings, helped to clarify the concept of depressed mood.

Gruenberg's invocation of the "hasty reader" who might assume that the DACL is equivalent to psychi[illegible] for individuals involves a serious misperceptio[illegible] of this article. The study[1] was undertaken to [illegible] pressed mood scale that could be used epidemi[illegible] to describe patterns in populations, not in indivi[illegible] lation coefficients with a (heuristic) criterion [illegible] ly to what extent a scale can do this, and [illegible] individual misclassifications is not relevant [illegible] clear that the DACL is a research instrument [illegible] cal one.

The data from this study, collected in 1971, [illegible] source and allow a variety of analyses, a num[illegible] we have planned. We are pleased to learn [illegible] berg is also preparing a report, but the conc[illegible] will be "more accurate" is not justified by h[illegible] the present article.

ROGER CHRISTEN[illegible]
Poughke[illegible]
BERNARD [illegible]
Kans[illegible]
MAURICE S[illegible]
Ston[illegible]

[1]The principal investigator of the study on which t[illegible] which received major support from grant MH-20[illegible] tional Institute of Mental Health, awarded to Dr [illegible] and partial support from MH-19966, was the sec[illegible] article.

Psychiatric History: Potential for Abuse

SIR: Regarding the "Position Statement on [illegible] Against Persons with Previous Psychiatric Tr[illegible] 1978 issue, Official Actions) I am glad to see [illegible] being given to this extremely important area. [illegible] my colleagues I have long felt that the oblig[illegible] forms declaring whether one has received ps[illegible] ment is both an invasion of privacy and a pot[illegible] covert or overt discrimination. A number of [illegible] have been quite ethical in other respects her[illegible] able about lying when presented with this [illegible] reason that leaving the question unanswered [illegible] mission of having received psychiatric treat[illegible] and, since the question is unjustified and [illegible] er, feel ethically justified in answering it in the [illegible] of course opens them to the accusation of [illegible] information.

I wonder, however, if the statement goes [illegible] particularly concerned about the following req[illegible]

> With adequate safeguards and with the [illegible] of confidentiality under a code of ethics [illegible] medical consultant should be able to ask [illegible] needed medical information from applican[illegible] termine whether the person is capable of [illegible] specific functions normally expected in the [illegible] which he or she is being evaluated. The [illegible] about diagnosis and history should not be [illegible] ed to anyone outside of the medical unit.

It should be evident to anyone who has wo[illegible] or government systems that the physician in [illegible] has a primary obligation to his or her employ[illegible]

him or her not to communicate with the employer about the prospective employee is asking too much. Furthermore, the safeguards regarding medical documents are by no means as airtight as we would like to think. Any physician who is entrusted with the task of "screening" a prospective applicant is primarily serving the company, not the applicant. His access to private and therefore potentially prejudicial past information is no different from the access of the potential employer. In my view, judgments should be made on the information available at that time, even if it means overlooking potentially troublesome material. The alternative is a return to the system we have now, wherein a person's medical history can be used against him.

A simple and effective way around the difficulty, which has already been adopted by a number of agencies, is to include a question asking applicants whether they to the best of their knowledge have any medical condition that would interfere with safe and successful discharge of their duties. A person who conceals such information would be appropriately subject to penalty. An example might be the willful concealment of uncontrolled epilepsy by a person entrusted with the operation of hazardous machinery or motor vehicles. I think it is fair to expect that individuals applying for responsible and sensitive jobs would have the integrity to reveal those problems which could interfere with the discharge of their duties. The responsibility should be left to the individual. No other system is acceptable in an open and free society.

I hope that others will respond and that the American Psychiatric Association will add its voice to those attempting to protect the rights of individuals.

JOHN RACY, M.D.
Rochester, N.Y.

Neurological Basis for Syndromes and Symptoms of Disordered Recognition?

SIR: The case of intermetamorphosis syndrome reported by Demetrios E. Malliaras, M.D., and colleagues (Clinical and Research Reports, August 1978 issue) has several interesting features, particularly those suggesting a possible organic component of the illness. An aspect that was not discussed is the patient's delusional notion that her theology instructor (the subject of her misidentifications) was in love with her, a symptom often linked with the name de Clérambault (1). That this association of delusional phenomena may not be entirely fortuitous is suggested by a previous report (2) of their coexistence, although in that case the misidentification was of the Capgras type. In yet another report (3), where symptoms of both delusional hyperidentification (intermetamorphosis) and hypoidentification (Capgras) were present, the author thought that "repressed erotic desires" were an important psychopathological factor.

Aficionados of the syndrome of doubles will also recall a case described by Capgras and Carrette (4) in which the patient made flagrantly erotic advances toward her father, whom she perceived as a "double." In this case, like that reported by Dr. Malliaras and associates, a number of features (false feelings of strangeness and familiarity, olfactory hallucinations, episodic outbursts of aggression, well-preserved affect, and lack of incoherence of either spoken or written language) suggest a psychomotor component to the illness.

A review of Flor-Henry's recent writings (5) on temporal-limbic laterality and psychopathology suggests a link not only between the phenomenon of disordered recognition and the nondominant temporal lobe but also between hypersexuality and the right hemisphere. Nondominant temporal dysfunction may also be reflected by the marked verbal-performance discrepancy of the patient described by Dr. Malliaras and associates. It is tempting to speculate not only that symptoms of misidentification and erotic delusions might logically coexist but that the common pathology might be localized in the nondominant temporal lobe.

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JOHN L. BARTON, M.B., CH.D., M.R.C.PSYCH.
St. Louis, Mo.

More on a Neglected Problem

SIR: The article "Father-Son Incest: Underreported Psychiatric Problem?" by Katharine N. Dixon, M.D., and associates (July 1978 issue) was of great interest to me. We have seen a case in our child guidance clinic that involved two brothers, aged 15 and 9, who had been used sexually by their father for at least 4 years, with their mother's full knowledge. The case came to our attention after the older boy informed his minister of his experiences.

Needless to say, the case presented serious management problems. The parents had maintained a conspiracy of silence for years and were enraged that this matter had come to the attention of people outside the family. Although the case was reported to the Child Protective Services as one of child abuse, the court made only a mild recommendation that the children obtain psychotherapy, a suggestion the parents angrily rejected.

Like the cases reported by Dr. Dixon and associates, this one emphasizes the extreme delicacy and complexity of such situations. I hope others in the field will be alerted to the existence of a problem that has been neglected. Dr. Dixon and her coworkers have performed a valuable service through their paper.

AARON H. ESMAN, M.D.
New York, N.Y.

Medical Information Card for Patients Taking Lithium

SIR: At the Genesee Mental Health Center, we recently designed a wallet-sized information and warning card for our patients who are taking lithium carbonate. The card, which is analogous to those carried by patients taking disulfiram, was designed to alert these patients and their physicians

about the unique cautions that must be observed when patients are on maintenance lithium therapy. In addition, the card serves to inform others (e.g., family, ambulance drivers, police officers) about certain symptoms of lithium toxicity so that they can be recognized and medical evaluation and treatment can be instituted without unnecessary delay. One side of the card contains the following information: "I am on lithium carbonate. At any signs of diarrhea, nausea, vomiting, visual disturbance, tremor, unsteady walk, drowsiness, weakness, slurred speech, or mental confusion, please call my therapist." The phone number of the center and of an emergency room are then given. The reverse side gives the patient's name, address, and phone number, the name of his or her primary therapist, and a list of incompatible drugs (diuretics, sodium, potassium, and weight reduction pills).

We would be happy to hear any comments that readers might have about this idea.

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Procedures for Taste Aversion Therapy for Alcoholism

SIR: Taste aversion therapy, one of the earliest attempts to apply experimentally derived principles of learning to clinical treatment (1), involves the pairing of the sight, smell, and taste of alcohol with the effects of an emetic. Recent basic research into the acquisition of taste aversions (2) has led to suggestions that taste aversion therapy be used more frequently (3).

The most widely used emetic in alcohol aversion therapy is emetine HCl. A frequently used regimen[1] consists of 5 treatments over a 10-day period. The oral dose of emetine is 100 mg per session, and the I.M. dose increases from 52 to 78 mg. The total treatment dosage during the series is 345 mg I.M. and 500 mg p.o. Rollo (4) has stated that emetine dosage should not exceed 60 mg/day I.M. or 600 mg I.M. in 10 days. Thus this treatment exceeds the maximum recommended daily emetine dosage and, depending on how much of the oral emetine is absorbed, approaches or exceeds the maximum recommended total emetine dosage over a 10-day course.

In attempting to develop a more conservative regimen, we found that lower dosages failed to produce reliable emesis, which is crucial to this treatment because of the quantity of alcohol consumed during conditioning. Syrup of ipecac, a much safer emetic, did not seem to produce good conditioning, perhaps owing to the short duration of its effects.

Currently we employ both 20 ml of ipecac p.o. and 42 mg of emetine I.M. The ipecac produces reliable emesis and the emetine prolongs the malaise. Strong aversions develop, but none of the symptoms of emetine toxicity (hypotension, precordial pain, tachycardia, dyspnea, and ECG [illegible] has been observed.

As in the more widely used procedure, [illegible] ephedrine are also given in the injection with [illegible] The pilocarpine dosage increases across sess[illegible] 18 mg, and the ephedrine dosage increases fr[illegible] Pilocarpine prevents the absorption of alcoh[illegible] small intestine by closing the pyloric sphinc[illegible] edrine protects against shock. In some cases [illegible] essary to reduce the pilocarpine dosage bec[illegible] pleasant side effects (e.g., abdominal cramp[illegible] utes after the oral ipecac and the emetine inj[illegible] are given about 2400 ml of a variety of alcoh[illegible] The inpatient course of 5 treatments is follo[illegible] patient treatment sessions over the next 6 mo[illegible]

Preliminary results of a clinical research p[illegible] in progress suggest that taste aversion the[illegible] adjunctive therapy in a comprehensive alcoho[illegible] tion program. Additional information on the [illegible] cedures may be obtained from the authors.

[1]Unpublished protocol, Raleigh Hills Hospital, Portland, Ore.

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DALE S. C[illegible]
GARY M. STEPH[illegible]
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Veterans Administrat[illegible]
Salt Lake City [illegible]

Corrections

There was an error in the author affiliation[illegible] cyte Concentrations of the Lithium Ion: Cli[illegible] and Mechanisms of Action" by Frazer and [illegible] September issue. Dr. London is Chief, Com[illegible] Department of Radiology, Hospital of the [illegible] Pennsylvania, Philadelphia, Pa.

There was also an error in the book review [illegible] Karasu of *Therapeutic Partnership: Ethical [illegible] Psychotherapy* by Carl Goldberg in the Septe[illegible] the next to last paragraph the quotation shou[illegible] therapist must teach the client how to ratio[illegible] power or how to regain his lost power."

The staff regrets these errors.

BOOK REVIEWS

The Psychological Society: A Critical Analysis of Psychiatry, Psychotherapy, Psychoanalysis and the Psychological Revolution, *by Martin L. Gross. New York, N.Y., Random House, 1978, 354 pp., $10.95.*

Psychotherapy is thought by some critics to be the Achilles' heel of contemporary psychiatry. Ever since Eysenck, in a 1952 monograph, claimed to have proven that the results of psychotherapy were not any better than no therapy at all, similar iconoclastic attacks have appeared periodically and have followed certain predictable patterns. For example, it has been charged that dynamic psychotherapy is useless; that if it works at all, it does so only through suggestion; that its favorable outcomes are the results of spontaneous remissions; that it does as much harm as good; that its presumptive scientific basis is pure hogwash; and that psychiatrists are simply twentieth century versions of primitive tribal witch doctors and their therapies depend on a similar kind of myth and magic.

The Psychological Society is the latest and one of the more sophisticated of this critical genre. Gross, a well-known editor and writer (*The Brain Watchers* and *The Doctors*), has done a good deal of research into psychiatric literature and has interviewed many psychiatrists and psychologists in the course of preparing this book. He has come up with a wide-ranging and hard-hitting attack on all dynamic psychiatry and particularly on Freudian psychoanalysis, which he considers to be the ideological wellspring of such psychiatric thought.

Gross's thesis, essentially, is that our society has been overpsychologized to its great detriment. Psychology (and psychiatry) has become "an art, science, therapy, religion, moral code, life-style, philosophy, and cult." The quest for an idealized "mental health," he asserts, has created self-doubt in millions of people who have been labeled as "neurotic" because they reflect the ordinary anxieties and insecurities of everyday living, and who are then subjected to an essentially useless variety of psychotherapies.

The bête noir behind this unhealthy state of affairs, according to the author, is psychoanalytic theory, which is attacked as a totally fanciful, unvalidated, and unscientific creation of Freud, whom Gross describes as a naïvely superstitious, mystical, and neurotic man.

Gross asserts that psychiatrists and psychologists, building on this dubious foundation, have put themselves forth as experts ("new seers") on a wide variety of human events, in effect replacing the oracles and prophets of old. He holds up to ridicule the often conflicting and mutually contradictory "authoritative" statements made by psychiatrists and psychologists about crime, criminals, politics, and international affairs, no less than statements about psychosomatic diseases, cancer, and homosexuality. He attacks theories about the psychogenic origins of the psychoses as being totally without merit and asserts that the influence of psychoanalytic theory in childrearing and in education has been harmful and misleading. Indeed, he quotes with obvious approval a characterization of this influence as "the psychological crime of the century."

The book is not to be lightly dismissed, however. Its views are buttressed by quotations from numerous psychiatrists and psychologists, many of outstanding eminence. Many of its criticisms are justified and cogently point up some of the soft spots in both psychiatry and clinical psychology—unwarranted promises, exaggerated claims, unscientific theorizing, fanciful analyses of dead historical figures, and unethical psychologizing about living ones. Where the book falls down is in its heavily biased and grossly (no pun intended) one-sided point of view. The author's view of human personality and psychopathology is that they are almost totally determined by genetic factors. Although he pays occasional lip service to the interplay of life events with these factors, his bias is so strongly biological as to be practically a twentieth-century caricature of nineteenth-century psychiatric thinking. Gross denies the existence of unconscious psychological elements and replaces them with "gene-determined neurological-biochemical circuitry." Reactions to love, adventure, threat, sex, and friendship may be "partially learned," he concedes, but are more importantly attributed to "thousands of protein-coded genes each of us has been endowed with since the moment of conception." Homosexuals are all "born that way." Neurosis is just a "Freudian fantasy," and the symptoms ascribed to it are more often due to "physical sickness." The psychoses are all due solely to biological factors and will eventually be prevented and cured by biochemical means alone. Dreams are merely accidental offshoots of neural activity and "have no particular meaning." Infantile and childhood sexuality are myths. "The human child is generally a non-sexual being." The child's personality has little or nothing to do with patterns of upbringing; it is almost entirely due to "temperament, nature, constitution, [and] inborn traits."

The author, moreover, like most intemperate critics, makes the error of throwing the baby out with the bath water. To dismiss the totality of Freud's contribution as a massive and harmful fraud perpetrated on an entire society is to lose all historical perspective with regard to that contribution. The ad hominem attack on Freud's personality is unworthy of the author and has no bearing on the value of Freud's work. Even if some of Freud's metapsychology has indeed proven to be in error, it still constituted a revolutionary breakthrough in its time and paved the way for more sophisticated later theories. In addition, Freud's formulation of a technique for studying psychological processes by psychological methods was a vitally important methodological advance that significantly furthered our understanding of human behavior and motivation.

Most of the criticisms that Gross advances come from psychiatrists and psychoanalysts themselves, pointing out errors and weaknesses in previous theory. Such dissensions are an intrinsic aspect of the scientific method, and similar criticisms could be compiled in any other field of science. Even so, however, almost all of the experts whom the author

chooses to quote (occasionally out of context) reflect a particular point of view, and Gross makes no effort to give a fair hearing to differing views.

Most of all, the one-sided and simplistic biological bias of the book does a massive injustice to the sophistication of biopsychosocial approaches and contemporary systems thinking in modern psychiatric theory and practice. To minimize the importance in human development of interpersonal life experiences and the social matrix in which they take place is just as egregious an error as it would be to ignore or minimize the importance of the biological factors involved. By the same token, to equate contemporary efforts at scientific psychotherapy with the magical incantations of the primitive tribal shaman is equally fallacious. The fact that they both achieve roughly equivalent results in some instances is quite beside the point. Ancient herbal medicines may cure some ailments just as well as modern antibiotics, but only by pursuing the methods of science can we hope to achieve the rational breakthroughs that lead to significant scientific advances. Continuing our efforts to rationally understand the sources of human emotional disorders and to treat them on the basis of such reasoned understanding is in the finest tradition of science. Whatever the limitations of our present knowledge, there is no reason to doubt that these efforts will ultimately be successful.

JUDD MARMOR, M.D.
Los Angeles, Calif.

The Technique of Psychotherapy, 3rd ed., Parts 1 and 2, *by Lewis R. Wolberg, M.D., New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 1,301 pp., $69.00.*

It is a brave person indeed who would undertake to write a comprehensive textbook of psychotherapy, a field that, to quote the author, is "balanced precariously on the pinnacle of profound ambiguities. What seems like the truth of today may be the exploded myth of tomorrow" (p. 842). Dr. Wolberg accepted the challenge about a quarter of a century ago, and the appearance of this third edition is the measure of his success in meeting it. The size of the book itself indicates the magnitude of the task—it contains more than 1,300 double-column pages of text and more than 3,000 references.

The author succeeds in encompassing the chaotic field of psychotherapy by organizing it around a guiding concept. Although this concept is nowhere explicitly stated, it is clear that the author's basic conceptualization is a flexible psychoanalytic one. According to this model, other things being equal, the definitive treatment for neuroses and personality disorders is dyadic therapy based on efforts to bring into consciousness and thereby resolve unconscious conflicts and motivations, with special emphasis on handling the transference. This is termed "reconstructive therapy" in that, when successful, it is believed to achieve alteration of character structure and expansion of personality growth. More modest goals, which have their legitimate places, are supportive and reeducative therapies.

There is no convincing evidence that any forms of psychotherapy, including psychoanalysis, are capable of significantly restructuring personality, or that analytically oriented approaches produce more profound personality changes than other techniques. Still, it is hard to think of any better way of bringing order into the field of psychotherapies, and the author's framework would probably gain more general acceptance than any alternative. Wolberg manages to force most current psychotherapies into his co[illegible] even though this creates some unlikely bed[fellows] surgery and sociotherapy, for example, [illegible] forms of supportive therapy, while "Hare K[rishna] havior therapy are both classed as reeducat[ive] impose order also finds expression in a pl[ethora] tables, and charts that provide varying degr[ees] tion.

Given the author's orientation, the actu[al] volumes cannot be praised too highly. The [first] of extant therapies consists of concise, balanc[ed] with due consideration of strengths and weak[nesses] strating that the author has not only read b[roadly] writings of their proponents. It is, moreover, [also] clusive and up-to-date, omitting, to my know[ledge] approach, a self-help program with somethi[ng] known as reevaluation counseling.

The detailed, play-by-play descriptions of [psychotherapy] therapy, illustrated by many transcripts of sess[ions] views, are obviously based on extensive and [clinical] experience. Within a broad analytical framew[ork] tive, flexible approach that uses a variety of [techniques] techniques when appropriate. Descriptions [of] group, and family therapies, although consc[iously] tailed, are lucid and authoritative. Various [special] matters, among them evaluation of psychotic [patients] of emergencies, and community mental hea[lth] attention.

All in all, if it were possible to write a de[finitive] of psychotherapy at the present juncture, this [would] its encyclopedic coverage and extensive b[ibliography] cluding texts, films, and tapes, it is an ind[ispensable] ence work, and its detailed, clinically astut[e] constructive therapy provides an excellent [guide] remains the most widely used American a[uthor] undoubtedly maintain the deservedly pre[eminent] established by the earlier editions.

JEROME D. [illegible]
Be[thesda]

The Anatomy of Hallucinations, *by Fred H[oyle] cago, Ill., Nelson-Hall, 1978, 226 pp., $16.9[5]*

This unusual, scholarly volume was wr[itten] roanatomist who was Papaz' last Ph.D. studen[t] studied under and worked with many fam[ous] whom he dedicates his book. As a scholar J[ohnson] references with extracts from these writings [and] he has read them all and digested their cont[ents]

The term "anatomy" is not meant to in[dicate] cal descriptions but mental *processes* that o[ccur] rological circuitry, about which little is [known] theless, investigators have determined that [hallucinations] are accompanied by subvocal physiological [activity] inner speech that usurp normal inner speech.

Johnson quotes and outlines a long list of [persons] history of our world whose hallucinations [were] creative or destructive, including Hitler, Muss[olini] anese war lords. Many who seem healthy s[uffer] vocal speech in the form of hallucinations—[their] own thoughts as if they were spoken. For a s[ample] son quotes the observations of others indicat[ing that] hallucinators can hear the silent auditory ha[llucinations]

others confined within the same institution and also suffering from schizophrenia. Picture two patients with blank faces and immobile lips talking and listening to each other on a nonverbal level! Not only schizophrenic patients but hysterical patients and depressed psychotic patients may hallucinate. This, in my experience, is hard to teach because students seem to define hallucinations as a necessary symptom of schizophrenia. Also, hallucinations are painful for some and pleasurable for others.

This volume must have taken years to compile, especially since the author is an anatomist and not a clinician. He has succeeded ably in a scholarly product. He reminds me of an examiner for the American Board of Psychiatry and Neurology who, some years ago, asked his examinees, "I am hallucinating—prove it!" Now the answer would be, "I am not, so I cannot hear you." This is an informative and scholarly book.

ROY R. GRINKER, SR., M.D.
Chicago, Ill.

The Psychiatric Halfway House: A Handbook of Theory and Practice, *by Richard D. Budson, M.D. Pittsburgh, Pa., University of Pittsburgh Press, 1978, 274 pp., $10.95.*

This is an uncommonly well-written, concise, but complete book. It is a pleasure to read. The author is correct in saying that it "approaches the ideal of a general textbook on the subject of halfway houses." In a detailed, logical, but succinct manner Dr. Budson shares his exceptionally broad experience in working with and developing halfway houses. While at Harvard Medical School he worked as a student volunteer with patients at the Metropolitan State Hospital. After finishing his psychiatric residency, he established a halfway house and later became Director of Community Residence Programs for the Commonwealth of Massachusetts. Thus, with equal authority he can discuss the relationships of patients in group meetings in the halfway house and the problems of developing state laws and regulations that promote high-quality community facilities while not throttling their development with bureaucratic regulations.

The first part of the book, titled The Development of an Ideology, discusses the impetus and need for the halfway house, its relationship to the community, and its many functions analogous to the family in the home and the community. The author also discusses moving out of the halfway house into nonsupervised living. "The Halfway House As a Family-Model Social System" is a chapter especially worth reading. The family has a responsibility for seeing that its members carry out tasks that are appropriate to their age, abilities, and position within the family. In mental hospitals, most tasks are the exclusive province of the staff and thus patients are deprived of the opportunity to assume responsibilities that would enhance their growth and development. In the halfway house, as in the family, there is appropriate sharing of tasks and responsibility. This is one of many striking differences between it and a mental hospital. The author also spells out the responsibilities of a halfway house and its inhabitants to the community and to themselves and shows how these are similar to family responsibilities. The halfway house must be part of the community in which it resides and must understand the laws and regulations of that community.

In "Legal Issues, Standards, and Regulations," the author states that "it is essential that standards not be so stringent as to compromise the essential programmatic characteristics of the community residence." For example, standards that require too many staff for around-the-clock service and too strict adherence to building codes designed for institutions can cause a program to be expensive, unworkable, or counterproductive in that it perpetuates the institutional system it was designed to replace. Perhaps the greatest challenge of our times is to set standards that are essential for the control and upgrading of human services in such a way as to allow these services to function in a humane and therapeutic manner. The author provides a discussion of the rules and regulations in Massachusetts and compares them with those of other states.

The short and to the point chapter "Community Entry and Zoning Requirements" is essential reading for anyone wanting to establish a halfway house. It contains good advice on how to avoid many of the pitfalls that have defeated the best efforts of those who want to help the mentally ill reintegrate into the community. The nitty gritty of the planning and operation of a community residence is discussed in a manner in which the pertinent details take precedence over wordy discourse. It is obvious that the writer knows what he is talking about. He not only shares his factual knowledge with the reader but also is able to communicate an affective experience in an objective and helpful way. This especially comes through in discussions of the group meetings of house members.

My only irritation with the book is that bad practices in large mental hospitals are blamed on the medical model. The fact that bad medicine may be practiced in some hospitals because of inadequate funds and inadequate staff is not necessarily the fault of the medical model. Some doctors might practice bad medicine, just as some authoritarian, custodial types of halfway houses might promote regression rather than social integration. However, my objection is a minor one and does not detract from the excellence of the presentation and the material. This book exemplifies the virtues of the old-fashioned idea that a book should be written by a single author who says what he needs to and stops.

ROBERT L. LEON, M.D.
San Antonio, Tex.

Epidemiological Approaches in Child Psychiatry, *edited by P.J. Graham. London, England, Academic Press, 1977, 397 pp., $19.50.*

This volume is welcome as an initial systematic attempt to examine and evaluate epidemiological approaches in child psychiatry. It is the first presentation in a single volume of data and issues in a field that is in its inception. Few large population studies have been done in child psychiatry. Meaningful epidemiological work requires the development of interview and questionnaire schedules for which validity and pertinence are both satisfactory. At this point, valid and reliable instruments for screening populations of children are scarce. The lack of standardized clinical assessment methods presents serious problems of comparison from one geographical area to another and from one period to another. A significant study of a representative population also requires the definition of the group sampled by the population, the demonstration that the screening methods have a low degree of false positives and false negatives, and the clarification of the pertinence of the findings by studying a subsample in depth.

This volume consists of a collection of papers given at a symposium held in October 1976. The participants came from six European countries and Canada, although the major representation was from England. In such a symposium one must expect that the individual contributions will be of uneven quality—and this is the case here. However, the contributors include a number of leading research workers in the field, among them Michael Rutter, who chaired the symposium, and Philip Graham, who edited the volume and summarized the implications for prevention.

The chapter by Rutter on methodological considerations and the one by Lorna Wing on the use of case registers are deceptively simple in their presentation. These chapters represent sophisticated and knowledgeable discussions of these basic issues for large-scale studies. Both the novice in the field and the expert will find these papers valuable.

The body of the volume is organized developmentally, with sections on early parenting, the preschool child, middle childhood, and adolescence. A wide range of topics is covered, including useful discussions of child abuse, the search for predictive features in "at-risk" babies, the behavioral screening of preschoolers, and the short-term outcome of problems in this age group. There are two interesting comparisons of psychiatric disorders in urban and rural settings: one in 6-year-olds in Denmark and the other in adolescents in Norway. A British study examines correlates of behavioral deviance in 8-year-olds, and a German study examines the correlates of delinquent behavior in youngsters under 14 years of age.

The final sections deal with areas of special interest. In a chapter on the psychiatric aspects of sensory disorders, Freeman, from Vancouver, Canada, gives an organized summation and critique of the confusion of terminology and data in this field. Bohman, from Sweden, presents a tightly written and well-organized overview of studies of adopted children, focusing on social and genetic influences on development. His discussion emphasizes the value of epidemiological data for programs of prevention.

The linkage between epidemiological research and prevention is emphasized by Graham in his final overview chapter. To quote from his summation,

> It will be clear that, although in some rather limited areas (mainly those of more purely medical concern) guidelines for preventive action can be laid down with some confidence, for most conditions, further research and investigation is necessary before this stage can be said to be reached. . . . What has so far been lacking in these endeavors is an epidemiological approach with examination of the impact of changes in social or educational policy, or planned intervention on the rates of disorders in a population.

This volume makes a significant contribution toward this goal.

STELLA CHESS, M.D.
New York, N.Y.

Models of Madness, *by Erica M. Bates, Ph.D. Lawrence, Mass., University of Queensland Press, 1978, 175 pp., $16.95.*

The author divides the book into two parts. In the first she deals with the popular stereotypes that make up the models of mental illness in modern Western society [illegible] effects of labeling people as mentally ill. In t[illegible] she discusses her research, in which she atte[illegible] out just how people in an urban Australian [illegible] mentally ill.

The first part of the book is really a histo[illegible] attitudes toward deviant behavior. The autho[illegible] development of the medical model as a reac[illegible] gious or "accursed" model. From this grew [illegible] el, modified by the inclusion of the "sick [illegible] people needing medical attention. The medica[illegible] includes community psychiatry, with attempts [illegible] psychiatry. The sociological model views [illegible] deviant social role, emphasizing that the s[illegible] rather than the actor is the critical variable [illegible] garding labeling and disposition.

The author then reviews the antipsychi[illegible] tional modes of Szasz and Laing, in which [illegible] change or modify deviant behavior is seen as [illegible] violating the patient's rights. The education[illegible] as the introduction of coping skills to the cli[illegible] an overview, the author sees four perspectiv[illegible] the goodness/badness dimension, the he[illegible] sion, the educated/miseducated dimension, a[illegible] equality/power dimension.

The second part of the book is the resear[illegible] of Australian urban populations as to the n[illegible] illness, the cause of mental illness, what s[illegible] about it, who should do it, and what are the [illegible] These attitudes are determined by a questi[illegible] thor provides numerical tabulations of the r[illegible] various questions.

The first part of the book is a good review [illegible] changes in the attitudes of psychiatrists tow[illegible] ward deviant behavior. The second part is [illegible] what modern Australian city dwellers th[illegible] behavior. The book is of moderate interest t[illegible] mental health professions. The research sho[illegible] to politicians, statesmen, and those who w[illegible] It reinforms us of the generally naïve attitudes [illegible] populace.

MAURICE J. B[illegible]
Ro[illegible]

International Encyclopedia of Psychiatry, Ps[illegible] analysis, and Neurology, 12 vols., *Benjamin [illegible] tor-in-chief. Division editors: Jules H. [illegible] Psychiatry; Joseph M. Notterman, PhD., [illegible] chology and Related Fields; Gordon F. De[illegible] George Stricker, Ph.D., Social, Developm[illegible] cal Psychology; Albert J. Solnit, M.D., [illegible] M.D., Psychoanalysis; and H. Houston Me[illegible] Abner Wolf, M.D., Neurology. New York, [illegible] trand Reinhold Co. for Aesculapius Publis[illegible] pp., $675.00.*

The task of reviewing an encyclopedia [illegible] dable and at worst futile. The venture unde[illegible] man is of heroic proportions. This set of vol[illegible] fields of psychiatry, psychology, psychoan[illegible] rology. To a surprising degree the encyclope[illegible] up to its name. This is both its strength and [illegible] tion. Virtually anything you wanted to know [illegible] to ask is to be found in this encyclopedia [illegible]

does not lend itself to leisurely reading, as does a good textbook. In my judgment it is exculsively a reference volume and a very complete one at that.

The editors have done a great deal of work in an attempt to unify the style so that the unavoidable differences between entries are minimized. All of the topics have a few bibliographical citations that can assist the reader in further explorations. No single topic is treated in excessive detail. The editors have placed stringent controls on the length of the entries in an effort to limit the size of the finished product. This was a necessary condition, but it leaves some of the topic treatments less than fully satisfying.

There are many virtues to be found in this encyclopedia, including the fact that it is a single source of much diverse information. The editors and authors selected to participate in this effort represent a remarkable array of talent never before brought together in a single undertaking. There is hardly a well-known figure who is not to be found in the pages of this encyclopedia. The information presented on the various topics is succinct and readily available. Nevertheless, it is unrealistic to expect that most practitioners will turn to such a set of volumes for assistance in their day-to-day educational needs. An encyclopedia is not likely to be used as a daily source of information. It has a special place and a special value. To the extent that any single encyclopedia can encompass these four fields, this effort has achieved that goal remarkably well.

ROBERT CANCRO, M.D.
New York, N.Y.

The Epidemiology of Heroin and Other Narcotics. NIDA Research Monograph 16, *edited by Joan Dunne Rittenhouse, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1977, 247 pp., no price listed.*

Robert DuPont, Director of the National Institute on Drug Abuse when this monograph was published, states in his foreword, "This document represents a quantum advance in the Institute's heroin epidemiology efforts and reinforces the Task Force's prediction that we will experience another decade of significant breakthroughs in this vital area." The details of the epidemiology of a relatively rare event, such as heroin addiction, are unlikely to be of great interest to many psychiatrists. However, some of the information contained in this book is fascinating and will have an impact on both treatment approaches and public policy.

Using her data on servicemen involved with heroin use in Viet Nam, Robins emphasizes the instability of the heroin addict population over time. Her follow-up studies indicate that only 2.8% of regular heroin users in Viet Nam resumed regular use of heroin within 3 years of their return home. That important information has done much to discredit the old myth, "Once an addict, always an addict." In this context she comments, "We have increasing evidence that many heroin users never become addicted at all, and that many of those who do become addicted recover rather quickly without any medical treatment."

Despite the above information, it was surprising to see Hunt's estimate that there were 4,350,000 active heroin users in the United States in 1974. Using the same data as Hunt—from arrests, treatment programs, and urinalyses of prospective employees—O'Donnell calculated a range of 815,625 to 13,050,000. Obviously there are problems in current methods of estimating prevalence figures. Some credence is given to the higher figure by the fact that in a Bedford-Stuyvesant survey, 21% of the men aged 20 to 24 reported that they used heroin (p. 203). In Lukoff's view, prevalence figures of this magnitude make the libertarian approach to heroin use, exemplified by Szasz, less tenable. Such extensive use impairs the socialization of many young men as they prepare for adult roles and removes a significant segment of the population from "the creative work of the community."

In another chapter Sells examines the data obtained from treatment programs through the Drug Abuse Reporting Program (DARP), which examined 43,943 admissions over a 4-year period. Since 1969 heroin use by whites appears to be increasing and use by blacks decreasing. There also appears to be increased multiple drug use and use by adolescents. One-third of those who use heroin daily have no illegal sources of support and are not in trouble with the law when they enter treatment. The data also indicate that treatment helps control illicit drug use and reduces crime. An interesting footnote (p. 143) reports that in Detroit a 1% increase in treatment program enrollment was associated with a .23% decrease in property crime, while a 1% increase in the price of heroin was accompanied by a .16% rise in crime.

McGlothlin, using data from his follow-up study of the California Civil Addict Program, reports positive as well as negative consequences of the use of heroin. Heavy alcohol and other drug use is reduced during periods of regular heroin use. When using heroin daily, 20% of his subjects reported becoming intoxicated with alcohol 2 or more times a week. The proportion rose to 51% when heroin use stopped.

In her summary the editor reminds the other authors that "measurement, which is the starting point of epidemiologic research, requires exquisite clarity of definition, differentiation, and precision." Although the current state of the art is still far from this goal, much has been accomplished. One conclusion emerges from the work reported in this volume: heroin use is not a homogeneous phenomenon. The problem has a varying impact on different individuals and communities and requires multivariate responses.

JOHN N. CHAPPEL, M.D.
Reno, Nev.

Dimensions of Personality, *edited by Harvey London and John E. Exner, Jr. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 599, pp., no price listed.*

This book consists of 13 chapters, each written at the request of the editors and devoted to research on a particular personality trait or dimension. It is a useful reference, bringing together in one place authoritative and generally well-written summaries of research on the major traits and providing, as was the editors' intent, "a sense of what has unfolded on the contemporary scene of personality research" (p. xv).

Each article begins with a discussion of the genesis of research on the trait under study and proceeds to a selective review of the experimental literature and to a summary. When possible, the scales that are used to measure the various traits are also included. It is interesting to note that clinical practice was the inspiration for only about half of the research described here (introversion-extroversion, trait anxiety, locus of control). Other concepts were developed during basic research in perception laboratories (field dependence-independence, repression-sensitization, sensation-

seeking) or were derived from the area of political science (authoritarianism, Machiavellianism).

For psychiatrists and psychiatric residents whose knowledge in the area of personality is largely theoretical, *Dimensions of Personality* can serve as an excellent and not overly technical introduction to experimental personality research, with only a few caveats. First, personality research is a diverse field, in which the study of variables of individual differences is only one major emphasis. Much personality research of interest to clinicians (e.g., in aggression, the emotions, sex roles, and attribution theory) is conducted under the banner of social psychology. Second, there is a great deal of controversy in the field of personality as to whether the trait approach represented in this book is the most fruitful or a major conceptual and methodological reorientation is needed. Even among those in favor of reorientation, disagreement as to the form it should take is widespread. Finally, Wilson's chapter on introversion-extroversion, although among the most clinically germane in the book, is misleading and biased. Its dismissal of psychoanalytic theory is not only arrogant but premature; the presentation of Eysenck's theory glosses over the extensive criticism that theory has provoked.

The research summarized in the individual chapters is impressive. Most of the concepts have been carefully defined and their utility in predicting and understanding particular domains of behavior well established. I was left, however, with an impression that the whole was somehow not equal to the sum of its parts. What is lacking in the book is what is lacking in personality research in general: a concern with synthesis and with the integration of disparate concepts through theorizing. Wittingly or not, the editors have created a book that depicts the current state of personality research in its structure as well as in its content.

PETER B. ZELDOW, PH.D.
Syracuse, N.Y.

Behavioral Treatment of Alcohol Problems, *by Mark B. Sobell and Linda C. Sobell. New York, N.Y., Plenum Press, 1978, 222 pp., $16.95.*

This book has been difficult to review because it concerns a critical analysis of the literature, arguments for and against, a complex research program, and a meticulous follow-up study regarding the feasibility of controlled nonproblem drinking as a goal of alcoholism therapy. The 1976 Rand report, Pattison's recent publications, and now the investigations of the Sobells take issue with the traditional belief that an alcoholic can never drink socially again. Obviously, there are a few alcoholics who regain control of their drinking, but whether this could ever be a safe or legitimate therapeutic goal is rather frightening to contemplate. I have had at least 15 patients who relapsed because of the Rand report. How many alcoholics read the Rand report and were able to resume nonproblem drinking is not known, so the concept of treatment directed toward changing pathological drinking into controlled drinking cannot be rejected out of hand.

The main body of this book is a report of a research project conducted at Patton State Hospital from 1969 to 1971. Individualized behavior therapy, consisting of electric shock avoidance, videotape replay, experimentally controlled consumption of alcohol, and individual therapy sessions directed toward problem-solving skills, especially the drinking problem, was given to 20 alcoholics w[illegible] achieving nonproblem drinking. Fourteen [illegible] were given the behavior therapy with the goal [illegible] and 36 subjects were not given the behavio[illegible] served as two control groups for both conti[illegible] and nondrinking comparisons.

An extensive and detailed monthly follow-[illegible] of 2 years was conducted on 69 of these 70 [illegible] results indicated that the behavior therapy g[illegible] attempting to learn nonproblem drinking beha[illegible] ter general outcome and longer periods of sob[illegible] behavior therapy group attempting abstinence[illegible] treated control groups. On the basis of these [illegible] thors imply that some alcoholics can be ta[illegible] their drinking to a nonproblem level. Who [illegible] might be or how many alcoholics would be su[illegible] unconventional treatment is not clear.

However, the authors suggest several [illegible] have found useful in predicting a successfu[illegible] ing outcome. These include a shorter drinking [illegible] arrests, fewer hospitalizations, a less im[illegible] stable personality, a less advanced or deteri[illegible] drinking, more resources and stronger suppo[illegible] higher intelligence. These characteristics w[illegible] apply to a better prognosis for any kind o[illegible] they are especially important if behavior t[illegible] goal of controlled drinking is attempted.

Certainly any therapist who has the [illegible] shape an alcoholic's drinking pattern to a n[illegible] could do well to read this book. The authors [illegible] their assessment, therapeutic, and evaluatio[illegible] sufficient detail to serve as a guideline in thi[illegible] troversial, method of treating alcoholism.

FREDERICK I[illegible]
S[illegible]

Alcoholism: A Comparison of Treatment and Study of the Influence of Marriage. Maudsley [illegible] *by Jim Orford, M.A., Dip. Psych., Ph.D.* [illegible] *wards, M.A., D.M., D.P.M. Oxford, Enstar* [illegible] *versity Press, 1978, 167 pp., no price listed*

This book is an expanded report of the re[illegible] wards presented at the National Council on [illegible] ference in Washington, D.C., in May 1976 [illegible] lished in the *Journal of Alcohol Studies* [illegible] marvelous example of the style of the sc[illegible] bane, diffident, examining many viewpoints [illegible] alternative hypotheses to counter his own [illegible] type of presentation lends itself to the reader [illegible] cepting some strong points of view that are [illegible] sented. Indeed, we could hope that more [illegible] would attempt to be as thorough and as cle[illegible] tion of their results and that they too would [illegible] ments such as this, which clearly dichotomi[illegible] ment variables while maintaining both respe[illegible] son as an individual and a clear scientific cl[illegible]

The research itself is a study in which [illegible] secutive randomized married alcoholic men [illegible] range alcoholism treatment of the kind consi[illegible] dard, and the other 50, after a thorough exp[illegible] that the responsibility for achieving the st[illegible] their own hands and could not be taken over [illegible] message was given in sympathetic and constr[illegible]

was explained that the husband would not be offered a further appointment at the clinic, but that the social worker would call each month to see the wife and collect news of her husband's progress.

The psychiatrist told both the treatment and the advice groups that the husband was suffering from alcoholism and advised that the treatment goal should be total abstinence, that the husband should continue or return to work, and that the husband and wife should attempt to make their marriage viable.

The results were startling over a 2-year follow-up in that there were no statistically significant differences in outcome between the treatment and the advice groups. Although they acknowledge that married alcoholics are a special classification, the authors suggest that perhaps long-stay inpatient treatment facilities are not as necessary as was once thought and that other provisions should be made for adopting, at least as a trial, the "advice" plan of management of the patient. Not a single man in the study was completely abstinent for the 2-year follow-up period.

An incidental but valuable aim of the study was the exploration of the alcoholic marriage by a number of observations and psychometric and historical explorations (of dominance-submissiveness, hostility, cohesion, and sexual activity, for instance). I would chide these alcoholism experts for pursuing their explorations in a vacuum that does not take into account the general psychiatric literature on marriage.

Of the marital variables studied, it was *cohesion* that most strongly related to outcome. Relatively cohesive couples were more than twice as likely to be in the good than in the bad outcome group at follow-up; relatively noncohesive couples were more than three times as likely to be in the "bad" group. The authors themselves point out the small number of couples involved and the need for replication.

The gnawing question that arises in the reader of alcohol literature is brought in by the relatively small degree of success (about one-third by their standards) that both treated and untreated clients achieved. There were also few men in the study who used Alcoholics Anonymous and who attributed any part of their success to AA. This contrasts with other follow-up statistics, e.g., those presented by the program of the U.S. Navy in which 84% of the patients over 25 years of age succeeded sufficiently to return to active duty (1).

I think we should not dismiss these discrepancies as being due to superior information gathering on the part of the Maudsley team. When we look at the two programs, we see other quite important variables that may influence results in such a complex field. The first, so well-known as to be banal, is the coercive value of a job-related referral to treatment (i.e., the Navy program). The second is more subtle, more open to scientific criticism, and yet a powerful force, i.e., the conviction on the part of those who provide treatment in the efficacy of a therapeutic mode. In the Navy program, the whole program is designed by people who believe absolutely that adaptation to the AA way of life is the single most important and necessary function to overcome alcoholism. In the Maudsley program, AA is one choice that is being studied, not advocated. Abstinence is another such variable. In fact, the "grapevine" in the London alcoholic community might be said to judge the Maudsley therapists justly or unjustly as leaning away both from abstinence and AA.

Under these circumstances I wonder if the lack of difference between outcomes of treatment and advice groups does not merely demonstrate the need for fervor on the part of those who provide treatment toward an outcome—a fervor that can be viewed by the client as ego supportive, motivating, and activating. Long stays in the Maudsley's neutral atmosphere made no difference, but there was not that much "business" to do. Long stays elsewhere might be valuable to overcome resistance to accepting a new way of life, to overcome suspicion and doubt of the "surrender" that AA demands, to openly accept the proferred "love" of the AA program, and to begin to see it and sobriety in practice.

The possibility of the efficacy of an "advice" strategy is in any case refreshing and supportive for many alcohol experts who only get one "chance at bat" with an alcoholic patient. I have seen many times the situation in which one extended and sympathetic interview with a patient or a patient and his or her spouse ending with clear recommendations has ushered in prolonged remission, either immediately or after an interval.

Dr. Edwards might take his own advice and look at the general psychiatric literature (2) in regard not only to marriage but to treatment. There is great interest in brief psychotherapy techniques—usually meaning a year and under for outpatient treatment—that call on the positive resources of the patient to actively seek improvement. It is my feeling that the exploration of such strategems in alcoholism as the "advice" group *can* lead to advances for some patients. However, a substantial and even major segment of the alcoholic population will still require inpatient indoctrination during which they learn about their disease and how to cope with it from those whose advice in imbued with fervor.

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The Care and Management of the Sick and Incompetent Physician, *by Robert C. Green, Jr., M.D., George J. Carroll, M.D., and William D. Buxton, M.D. Springfield, Ill., Charles C Thomas, 1978, 95 pp., $8.50.*

This small, timely book adds some much-needed hard data to the growing body of knowledge about physician illness. The experience of the Virginia State Board of Medicine provides the basis for these authors' study of the sick, incompetent, or unethical physician, and their conclusion is optimistic: well-considered intervention can effect a good prognosis. In addition to reviews of the serious subjects of drug addiction and alcoholism among physicians, the authors include chapters on maintaining professional competence by continuing education and on the subtle problems of aging. They point out that statistics about illness are still poor, that the profession as a whole still fails its sick members, and that adequate laws about reporting sick physicians are still needed. The medical profession must manage itself, or it will lose the right to do so. Our goal is to develop the most compassionate but effective ways of helping our sick colleagues, and these authors give us the benefit of their valuable experience.

ROBERT E. JONES, M.D.
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The Childhood Emotional Pattern and Corey Jones: A Psychoanalytical Biography, *by Leon J. Saul, M.D. New York, N.Y., Van Nostrand Reinhold Co., 1977, 336 pp., $13.95.*

This book, which is Dr. Saul's tenth, is obviously intended to illustrate the time-honored insight expressed in conventional terms as "the child is father to the man" and in psychoanalytic theory by the genetic or developmental point of view. The metapsychology of psychoanalysis consists of the set of essential assumptions on which its theory is based. It includes five points of view: the dynamic, the economic, the structural, the genetic, and the adaptive. The genetic point of view is that psychological phenomena begin with inborn traits and develop according to an "epigenetic groundplan," that the early forms of any such phenomena remain potentially active, and that, as the individual's life goes on, those earlier forms heavily influence subsequent ones (1).

The format of the present work is the life history of one individual, ostensibly based on a partial psychoanalytic reconstruction. There is no discussion of theory or technique and no reference to the literature. As a child, Corey Jones was cherished by his mother, father, sister, and one of his aunts; throughout his life he expected to be loved unconditionally, and he was. The bulk of the book's content concerns his libidinal ties. His apparently successful, nearly painless progress from beloved child to inhibited young adult to potent, successful lover and husband evokes Anna Freud's concept of normal developmental lines (2).

In fact, the book serves as an example of an aspect of the genetic point of view in psychoanalytic metapsychology that has recently been addressed by Abrams (3). He pointed out that the genetic basic assumptions include developmental propositions that tend to be overlooked in clinical work in favor of the emphasis on "antecedent determinism." In other words, there is an emphasis on determinism as opposed to "transformational processes" in development. Abrams has stated that we tend to confuse the genetic *point of view* with the genetic *approach*, which involves searching for antecedent causes in clinical psychoanalysis (3).

The transformational view of development is a reflection of the progressive (or regressive) potential in the human organism, and so it takes into account individuals' biological endowment (e.g., intellectual) as well as the effects of the process of growth and maturation on the outcome of personality formation.

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WILLIAM R. FLYNN, M.D.
Calabasas, Calif.

Cases of the Reincarnation Type, Vol. II: Ten Cases in Sri Lanka, *by Ian Stevenson, M.D. Charlottesville, Va., University Press of Virginia, 1978, 367 pp., $20.00.*

In reviewing an esoteric treatise of this kind, one must consider the integrity of the author as well as the nature of the subject. In this case the author demonstrates an admirable ability to treat a controversial subject in a controversial way. There is a difference. Controversy has a connotation implying polemics, disputation, and [illegible] novation has a positive connotation implying introduction of new ideas, renovation of old ideas, horizons, and breaking new ground. Dr. Stevenson, Professor of Psychiatry in the Division of Parapsychology, University of Virginia, clearly belongs in the [illegible] He therefore deserves our respectful attention and qualified agreement. It has been my personal [illegible] our profession welcomes innovative projects [illegible] are presented in a conservative, ethical, and [illegible] ner.

A mind without curiosity, it is said, is like [illegible] without a telescope. This book on reincarnation [illegible] ly stimulate such curiosity. For those readers [illegible] victims of the tenacity of tradition, it will be a [illegible] road to discovery, a source of intellectual [illegible] incentive to grant a latitude of opinion not [illegible] in precise science.

Despite the preposterous claims of certain [illegible] for hypnotic "regression," and despite the [illegible] that examples of reincarnation are most often [illegible] adherents of religions rooted in that belief [illegible] Buddhism in Stevenson's cases), certain [illegible] have rendered reincarnation a bit more scientifically [illegible] able if not yet scientifically proven. I refer [illegible] "immortality" of the germ plasm and its genetic [illegible] the provocative allegation, recently revived by [illegible] lian photography, that energy fields (auras, etc.) [illegible] may survive physical death, may retain elements [illegible] sciousness, may interact and reunite with [illegible] ent, and future energy fields, and may even [illegible] soul and spirit (1). It therefore seems not [illegible] suggest that reincarnation should not be ignored [illegible] not believed.

In this book we have a dispassionate, painstaking [illegible] cinating account by a respected psychiatrist [illegible] garded in parapsychological circles as a "[illegible] in the field. Of particular interest to psychiatrists [illegible] venson's suggestion that certain skills, phobias [illegible] behavior, confusion of gender identity, allergies [illegible] physical abnormalities may be related to [illegible] events in previous lives.

In addition to the 10 cases that make up the [illegible] book, the author includes an informative discussion [illegible] the Sinhalese belief in reincarnation and a glossary [illegible] and cultural terms essential to an understanding [illegible] veda Buddhism in the context of the case reports. Stevenson has written many articles on the subject [illegible] tion, and the present volume is an extension [illegible] 3). Interested researchers, as well as the general [illegible] look forward with interest and edification to [illegible] investigations and reports.

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STANLEY R. [illegible]

Mental Health in the Metropolis: The Midtown Manhattan Study, Book Two, revised, *by Leo Srole, Thomas S. Langner, Stanley T. Michael, Price Kirkpatrick, Marvin L. Opler, and Thomas A.C. Rennie; edited by Leo Srole and Anita Kassen Fischer. New York, N.Y., Harper Torchbooks (Harper & Row), 1977, 546 pp., $6.95 (paper).*

Books one and two of *Mental Health in the Metropolis* were originally published as volume 1 of the Midtown Manhattan Study (1). Dr. Srole has added new material, including four new chapters and two appendixes in book two and a new introduction in book one.

A pioneer work when it first appeared (1, 2) and now a classic of its genre, the Midtown Manhattan Study is a model for the collaboration of sociology and psychiatry. It is dedicated to the memory of Dr. Thomas Rennie, the founder and first director of the Midtown Manhattan Mental Health Study. Dr. Srole writes more like a novelist than a sociologist, and he refers to the great social realists and psychological novelists—Zola, Proust, Joyce, and Bellow.

Book one of the paperback version of the study, which is briefly summarized in book two, described the "psychosocial instruments" used in what the authors call their "exploratory surgery." Data comparing the Midtown Manhattan population with other general populations and findings on Manhattan's prime-of-life age groups, its men and women, and the various stages of marital status they represent are included.

Book two carries the study into the community. It cites home survey samples and compares rich, middle-class, and poor socioeconomic groups; immigrants and native-born groups; and the many national, ethnic, and religious groups—all in relation to mental health findings. It also discusses the metropolis versus the small town and the country and their respective "health-linked" qualities as human habitats. The documentation is extensive, and the charts are easy to read. References abound to the authors' predecessors, the Lynds (3), Warner and Low (4), Hollingshead and Redlich (5), and more contemporary workers such as Kenneth Kenniston.

A sample of just a few of the salient findings of this study should whet one's appetite for reading the entire work. Familiar now is the statement that the frequency of mental health impairment varies inversely with socioeconomic status. Using the four categories of well, mild symptom formation, moderate symptom formation, and impaired, the authors found that 34% of young people aged 20–29 whose parents were in the upper socioeconomic classes were well, compared with 21% of those whose parents were in the middle classes and only 13% of those whose parents were in the lower classes. Although the percentages were closer together at the impaired level, only 10% of the upper-class youth, compared with 17% of the middle class and 20% of the lower class, were judged severely impaired.

It was the large number of individuals found in the home survey samples who had symptoms of psychiatric illness that was so shocking when the study appeared 15 years ago, and it is still shocking. Whether or not poverty and other deprivations of socioeconomic origin have a profound effect on mental health is still being debated, but the studies of Srole and associates as well as the subsequent studies they have inspired remain as solid evidence.

An issue that Srole calls an ancient bias is the Rousseau-Jefferson idea that a big city like New York is "a cloacina of all the depravities of human nature." Srole brings us up to date on this issue in a chapter on the economic crisis of New York in 1975 and the anti-urban feeling of politicians, poets, and even sociologists (especially the "Chicago school," where Srole trained). Srole states that these groups are misled by nostalgic idealization of what Marcel Proust called "remembrance of things past." Srole cites Benjamin Rush, who said that cities are "pesilential to the morals, health and liberties of man." He then provides figures showing that although people living in the country lived longer than people in the city before the twentieth century, in 1960 the difference between city and country was insignificant. The Midtown Manhattan Study itself was used as evidence of the pathological environmental stress of the big city because of the large percentage of mentally ill people found in the home surveys. However, a parallel study of Stirling County in rural Nova Scotia, Canada (5), found that "half of the adults in Stirling County are *currently* [author's italics] suffering from some psychiatric disorder as defined by the American Psychiatric Association's Diagnostic and Statistical Manual (1952)." Srole also found that a New Zealand study showed that the psychotropic drug prescription rate per capita varied inversely with the size of the biggest cities.

As summary Dr. Srole offers "post hoc hypotheses" regarding the sociocultural factors that affect mental health adversely. First are the "multiple interwoven deprivations" imposed on children of indigent parents. Second are the "stigmatize-rejection mechanisms" rooted in society's rewarding and penalizing system for prestige or the lack of it. These are most extreme for blacks, but the poor, disabled, elderly, and nonmainstream population groups are also demeaned, and their self-esteem suffers. Third is the role of transition states such as disastrous life events.

The basic formulation of the authors is the following:

> Mental morbidity is in part an immediate manifestation of an impaired ego, damaged by an earlier malfunctioning family structure that, in turn, had been rendered defective, at least partially, by disruptive intrusions from, or defaults by mainstream community and national social networks.

We now call this phenomenon the biopsychosocial hypothesis of mental illness.

Finally, Dr. Srole answers some critics of the original work, proposes new research, and states that he is currently working on a restudy to check on the changes that have occurred since 1954. This classic work should be included in every residency training program as required reading. It is highly recommended to psychiatrists of all persuasions, including not only those who are more biologically grounded but also those who are more psychoanalytically oriented. The work provides a bridge between these groups.

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JOEL S. HANDLER, M.D.
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Handbook of Rational-Emotive Therapy, *by Albert Ellis and Russell Grieger. New York, N.Y., Springer Publishing Co., 1977, 433 pp., $22.50; $14.95 (paper).*

Although I was aware of Albert Ellis' reputation as a popular psychologist with a proclivity for placing his views before the general American public, I was unaware that he considered himself the founder of rational-emotive therapy, an approach to therapy that he views as unique and very much his discovery. I was furthermore unprepared for a style of presentation that is, at best, jaunty and unprofessional in tone and, at worst, disrespectful and ignorant, particularly regarding psychoanalysis and analytic psychotherapies, which are persistently harshly attacked. This is particularly ironic because it seems to me that rational-emotive therapy is derivative from dynamic forms of psychotherapy, representing the isolation and selection of one aspect of all effective psychotherapy rather than an original school of psychotherapy.

I do not wish in this review to appear as a defender of classical psychoanalysis as the exclusive source of effective psychotherapy. It seems important to me, however, to stress how often Ellis attributes a particular position or limitation in treatment technique to psychoanalysis when it is not in fact an aspect of psychoanalytic technique or theory. He then proceeds to present his "liberated" view as a welcome contrast. Although there is considerable room for disagreement within psychiatry, especially with regard to the practice of dynamic psychotherapy, there is no doubt that reading this handbook would bring all of us with a dynamic orientation closer together. Besides being out of touch with anything vaguely analytic that has occurred since 1910, Ellis and most of the other contributors to this book seem to have little knowledge of the general psychiatry literature.

Ellis as a writer is exceedingly self-indulgent. To illustrate, I quote, "Even the redoubtable Sigmund Freud occasionally saw fit to note, before he became anally fixated on the notion of having to bugger his mother, that emotional disturbance arises from what he called ideogenic sources, meaning from nutty and highly exaggerated ideas." This represents only the tip of the iceberg, stylistically speaking. In what may be one of the more offensive chapters in this rather astounding book, Ellis finds women frequently guilty of "love slobbism" and proceeds in what is a profoundly male chauvinistic way to devalue women's wishes for reliable dependency relationships in the interpersonal sphere.

If one is able to overcome Ellis' stylistic atrocities (it should be noted that chapters by other contributors are of a somewhat less offensive nature), one is still faced with the need for evaluating a questionable and superficial approach to psychotherapy. Unfortunately, Ellis and the other contributors continually point to increased symptomatic improvement as proof of the greater effectiveness of rational-emotive therapy as compared with other forms of psychotherapeutic treatment. Their naïveté in this regard is startling. The ability to alter subjective states of well-being by suggestion has been well established. Furthermore, modern dynamically oriented psychiatrists are aware that a variety of therapeutic techniques, ranging from anal[illegible] psychotherapy and from drugs to other s[illegible] ventions, may be successful in interrupting be[illegible] major illnesses.

I think it is of some importance, after stating [illegible] reservations about the quality of the book, to cl[illegible] authors mean by rational-emotive therapy. [illegible] easy task, as anyone inclined to read the book [illegible] soon find out. Perhaps the best summary com[illegible] himself:

> Given the position that people create their [illegible] ieties, guilts, depressions and angers, by the [illegible] beliefs they hold, the goal of therapy is to [illegible] client to give up these beliefs as well as the s[illegible] behaviors that often accompany them and to [illegible] their rational thinking and believing. [illegible] context of a direct verbally active and didac[illegible] the rational-emotive therapist makes full [illegible] Socratic method of disputing the client's [illegible] liefs in order to get him or her first to reco[illegible] then think them through, and finally reline[illegible] The therapist repeatedly and vigorously [illegible] clients think about their beliefs and insists [illegible] fer evidence to prove the things they have f[illegible] ply accepted.

Since most interpersonal or dynamic psychoth[illegible] reducing the influence of irrational beliefs, it is [illegible] what makes Ellis insist that he has discovered [illegible] ment entity.

As far as I can ascertain, the therapist's sta[illegible] emotive therapy is hostile toward the patient's [illegible] though all therapists often find it necessary to l[illegible] tively healthy neurotic patients to tolerate t[illegible] while treatment continues or to refrain from [illegible] peating a complaint in the service of continued [illegible] not think that the position of pitting oneself ag[illegible] tional in the patient is particularly helpful or just[illegible] unacceptable, however, is Ellis' disparaging at[illegible] the influence of the past relationships with pr[illegible] and about the importance of the transference [illegible] scious and symbolic meanings in the patient's [illegible] bal associations, and responses to the therapis[illegible] well-established aspects of dynamically orien[illegible] therapy are dismissed. I stress this fact becau[illegible] that there is some need for increased emphas[illegible] difficulties in the cognitive sphere. There may [illegible] tients whose treatment should be kept at a mor[illegible] level and for whom correction of educational [illegible] constricted world views, and a tendency to [illegible] tantly irrational ideas need to be addressed in [illegible] py. What is unsettling is to find a system [illegible] therapeutic thought that denies unconscious er[illegible] tors linked to the primary object relationship[illegible] the more superficial, cognitive difficulties.

Certainly naïveté is no crime. All of us w[illegible] psychiatric fields have areas of limitation in kn[illegible] this is different from elevating areas of limita[illegible] position of an asset, as Ellis tends to do. Fu[illegible] clinical example after clinical example, one get[illegible] sion of patients being assaulted by Ellis' pa[illegible] view, which appears to be harsh, hedonistic, an[illegible] For example, Ellis often shows patients that [illegible] ternal event that is causing a reaction but inte[illegible] tional thoughts. This results, at the clinical lev[illegible]

couraging patients to accept or ignore inconsiderate and abusive behavior by their friends or spouses. For Ellis it is the patient's assumptions that are the problem, i.e., assumptions that they should be treated well or that things should go right for them in life.

Working consistently with patients to help them reconstruct their histories and connect the past with the present, the unconscious with the conscious, is at the core of all dynamic psychotherapies. Part of this work includes attention to irrational ideas and attitudes. Unfortunately, this volume is written in such a way as to preclude its being a significant study of irrational ideas and how they can be managed in psychotherapy.

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Clinical Quantitative Methods, *by Richard W.J. Neufeld, M.D. New York, N.Y., Grune & Stratton, 1977, 184 pp., $14.50.*

What might be the goals of a statistical book for the use of psychiatrists? Initially, one would want help with understanding a literature that is becoming increasingly statistically complex, not only for the reader but also for the journal reviewer. Journal reviewers seem to need more and more help in understanding statistics, and frequently they need more help than they manage to get. A book that would serve this purpose would, on a very simple level, explain the purpose of a test, with examples from the clinical literature and various types of interpretations.

A second goal would be to show simple statistical designs for use in a case report with multiple quantitative observations or in small-sample research. This would aid the psychiatrist preparing something he or she finds interesting for the literature. Such a book in the nonparametric area is that of Siegel (1), which gives clear examples as well as purposes and methods of calculation and interpretation.

Finally, a third purpose of a statistics book for psychiatrists would be to provide a comprehensive resource in the field such as Winer's work (2). This would be used by the more sophisticated statistical researcher in checking assumptions, appropriate designs, statistics, and methodological procedures.

Clinical Quantitative Methods attacks the problem of single case design. Neufeld's approaches are not simple and therefore not of use to the ordinary psychiatrist. He does not cite some of the major works in this area. The common simple statistical procedures of chi square, t tests, and correlations are not presented. The areas that Neufeld does use to help readers and reviewers understand an increasingly complex literature are varied. He uses examples from such areas of interest to psychiatrists as schizophrenia and therapy evaluation as well as depression and evaluation of symptoms. Only a minority of the examples are from areas of somewhat less interest to the general psychiatrist. However, his examples are more complex than can be understood by the psychiatrist with a minimal background in statistics. Although journal reviewers seem to be more sophisticated in statistics than the average psychiatrist, I would doubt that many of them would be able to follow the procedures in this book if they were presented with a paper for evaluation.

Finally, Neufeld at no point attempts to present his book as a comprehensive reference book. This is wise, except that the title, *Clinical Quantitative Methods*, does not have a subtitle or suggest the area covered by the book. The introduction suggests that the emphasis is on the clinical part of the title and that some potentially useful areas of statistical analysis are presented. Again, however, it is not a comprehensive discussion of this area, nor does Neufeld attempt to point to other areas or books that might be useful to the person attempting to analyze data on patients or designing a study of clinical procedures or evaluation.

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DANIEL S.P. SCHUBERT, M.D., PH.D.
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The Alcoholic Marriage: Alternative Perspectives, *by Thomas J. Paolino, Jr., M.D., and Barbara S. McCrady, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 200 pp., $14.50.*

The authors state that this book was written because of their strong opinion that regardless of the theoretical perspective from which the alcoholic is conceptualized, his or her drinking and his or her marriage are inseparably related. With this as their objective, the authors examine the marriage of the alcoholic from four points of view: psychoanalytic, sociological, behavioral, and systems theory. Each perspective is reviewed in separate chapters on the basis of empirical evidence supporting or challenging the position in question. The chapters are divided by subtitles into topical components that offer a helpful outline to guide the reader through the authors' encyclopedic summation.

The writers offer their impression "based on the research evidence reviewed, that the behavioral perspective offers some of the most promising concepts and techniques developed to date." At the same time, they see the systems, psychoanalytic, and sociological concepts as offering ideas for the enrichment of treatment that would not be suggested by a strictly behavioral viewpoint.

As the basis of their extensive review of the literature, generously and selectively referenced in each chapter, the authors offer arguments that present a formidable challenge to the idea that people who are married to alcoholics tend to be psychologically disturbed. At the same time, they attempt to demonstrate how psychoanalytic theory has been "inadvertently abused, resulting in oversimplified theories about the alcoholic marriage." How well they may have succeeded in substantiating their position would appear to depend, to a considerable extent, on the professional sophistication of the reader.

The last section of the book, Treatment Implications, focuses on aspects pertaining to entering treatment and motivation for treatment, assessment of the alcoholic marriage, the delivery of treatment, and the therapeutic relationship. This section is worthy of perusal by sophisticate and novice seeking to come to therapeutic grips with the role of the spouse in the treatment course of alcoholics.

All in all, the authors present a virtuoso performance in their assessment of the four models of human behavior as applied to any aspect of normal or abnormal behavior. How-

ever, their exposition suffers from an important omission in that they fail to take advantage of their survey to comment on the possible extent to which the application of the different models might worsen the relationship between patient and spouse, even though such studied comments would at best yield only rough approximations. The book is thoughtful and does not make for easy reading. For those seriously concerned with psychiatric treatment, its reading and rereading should be rewarding because it offers a helpful guide in an area beset with complexities. I can recommend this book as a useful addition to one's working library.

ALBERT A. KURLAND, M.D.
Baltimore, Md.

Psychological Disorders of Children: A Handbook for Primary Care Physicians, *by Mark A. Stewart, M.D., and Ann Gath, D.M. Baltimore, Md., Williams & Wilkins Co., 1978, 160 pp., $9.95 (paper).*

Pedagogy is becoming a demanding taskmaster because of the current profusion of facts and varying clinical experiences. Selection of content has become as important as the manner of transmission. How should the old and new formulations of behavioral disturbances be correlated with each other? Can theory and practice drawn from experiences with adult disturbances be applied to children? Can the traditional medical model be made to fit the problems of children? Can theoretical sectarianism be avoided? Can psychological and biological determinants be integrated in a reasonable form of data selection and presentation? All of these questions are correctly raised in the short preface of this small book. Regrettably, the attempt to respond to these issues falls short of the intent of the authors to respond to them.

This book is presented as a handbook for primary care physicians, whom the authors identify as residents in psychiatry, pediatrics, and family practice, nurse practitioners, psychiatric nurses, medical students, and students in clinical psychology and other fields. In the face of their selection of so broad an audience, the authors assume a weighty responsibility to carefully choose, qualify, and quantify their subject matter. Nowhere do they meet this responsibility.

In their opening chapter, they question the validity of diagnosis in child psychiatry and then proceed to construct a book based on the specificity of diagnostic entities. In their next chapter, on infantile autism and schizophrenia, they devote 12 pages to infantile autism and dismiss the subject of childhood schizophrenia with less than 2 pages, which begin with the amazing statement that "true schizophrenia is probably rarer in children than autism." They justify this statement by quoting the isolated work of Makita reported in 1966 in the *Folia Psychiatrica et Neurologica Japonica* but completely ignore the vast literature on childhood schizophrenia in other major psychiatric publications. Anyone familiar with the complexity of the subject of infantile autism will be distressed both by the content and documentation of this section.

Uneven selection and presentation dominate the remaining chapters of the book. Nearly all of the sections reflect a combination of diverse bits of clinical work linked together by the authors' choice or inference. The result is a lack of clarity and the violation of stated purpose. The book does not achieve its goal of being "a handy introduction to child psychiatry," nor does it respond to the many questions raised in its own preface. In fact, this book [illegible] very questions it professes to answer. Its [illegible] from diagnostic entities to presentations of [illegible] tions. Conflicting attitudes between psycho[illegible] logical determinants are resolved by ignoring [illegible] of an extensive body of literature—usually in [illegible] cal area. Rarely do the authors point out [illegible] component to a physiological disturbance. [illegible] a simplistic representation of complex issues.

SOL NICH[illegible] M.D.
Ve[illegible] N.Y.

Children of Parting Parents, *by Lora Heims[illegible] New York, N.Y., Jason Aronson, 1978, 59[illegible] ed.*

The author is a clinical psychologist who [illegible] the staff of the MIT Psychiatric Service, Cam[illegible] and the Judge Baker Guidance Center in Bos[illegible] in the private practice of psychotherapy [illegible] adults. She reports material from more than [illegible] their parents whose problems had arisen [illegible] divorce, or death, with resulting emotional [illegible] children.

The book is divided into three sections. In [illegible] the author presents a wide range of underly[illegible] material presented reports on changing interact[illegible] men and women, changing attitudes toward c[illegible] changing role of the family in history, and cha[illegible] gy and in customs concerning marriage, div[illegible] care. In this first section the author describes [illegible] theories with reference to identification proc[illegible] ports on the various aspects of grief in childh[illegible] lescence.

In the second section of this book Dr. Tess[illegible] siderable clinical material, ranging from sing[illegible] therapy of several years' duration. In the th[illegible] author reports in detail about the therapy pro[illegible] suggestions not only for parents and profes[illegible] others in the child's social milieu.

The clinical material reveals clearly the au[illegible] sitivity, and professional competence. She [illegible] painful countertransference reactions that [illegible] dealing with grieving children and their paren[illegible] rich in clinical material. The case reports in[illegible] the material presented by the children, pare[illegible] parents but also the author's verbal responses [illegible] viduals, including her questions and interpret[illegible]

This book has an extensive bibliography [illegible] theoretical and clinical contributions. The pu[illegible] book at this time is of significance because [illegible] creasing rate of divorce, custody battles, [illegible] Many other writers have emphasized the se[illegible] traumata suffered by children and their paren[illegible] of divorce and remarriage. Dr. Tessman's [illegible] gives ample documentation to the needs of s[illegible] for professional guidance during and after [illegible] death of one parent. This book is highly rec[illegible] those in the mental health profession who de[illegible] who have lost a parent by divorce or death.

FRANK J. C[illegible] M.D.
New [illegible] N.Y.

This Month's Reviewers

JUDD MARMOR, M.D., is Franz Alexander Professor of Psychiatry, University of Southern California School of Medicine, Los Angeles, Calif.

JEROME D. FRANK, M.D., is Professor Emeritus of Psychiatry, Johns Hopkins University School of Medicine, Baltimore, Md.

ROY R. GRINKER, SR., M.D., is Chairman Emeritus, Department of Psychiatry, Michael Reese Hospital and Medical Center, and Professor of Psychiatry, Pritzker School of Medicine, University of Chicago, Chicago, Ill.

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MAURICE J. BARRY, JR., M.D., is Consultant in Adult Psychiatry, Mayo Clinic, Rochester, Minn.

ROBERT CANCRO, M.D., is Professor and Chairman, Department of Psychiatry, New York University Medical Center School of Medicine, New York, N.Y.

JOHN N. CHAPPEL, M.D., is Professor of Psychiatry, Department of Psychiatry and Behavioral Sciences, University of Nevada, Reno, Nev.

PETER B. ZELDOW, PH.D., is Assistant Professor of Psychiatry, Upstate Medical Center, and Staff Psychologist, Veterans Administration Hospital, Syracuse, N.Y.

FREDERICK LEMERE, M.D., is Clinical Professor of Psychiatry, University of Washington School of Medicine, Seattle, Wash.

FRANK A. SEIXAS, M.D., is Medical and Research Executive, National Council on Alcoholism, and Clinical Associate Professor of Public Health, Cornell University Medical College, New York, N.Y.

ROBERT E. JONES, M.D., is Clinical Professor of Psychiatry, Jefferson Medical College, Philadelphia, Pa.

WILLIAM R. FLYNN, M.D., is Medical Director, Woodview-Calabasas Hospital, Calabasas, Calif.

STANLEY R. DEAN, M.D., is Clinical Professor of Psychiatry, University of Miami School of Medicine, Miami, Fla., and University of Florida School of Medicine, Gainesville, Fla.

JOEL S. HANDLER, M.D., is Lecturer in Psychiatry, Northwestern University, Chicago, Ill.

HENRY J. FRIEDMAN, M.D., is Clinic Director, Outpatient Psychiatry, Tufts-New England Medical Center, Boston, Mass.

DANIEL S.P. SCHUBERT, M.D., PH.D., is Director of Research, Department of Psychiatry, Cleveland Metropolitan General Hospital, and Associate Professor, Case Western Reserve University School of Medicine, Cleveland, Ohio.

ALBERT A. KURLAND, M.D., is Research Associate, Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland School of Medicine, Baltimore, Md.

SOL NICHTERN, M.D., is Director of Psychiatric Services, Jewish Child Care Association of New York, New York, N.Y.

FRANK J. CURRAN, M.D., is Consulting Psychiatrist, St. Luke's Hospital, New York, N.Y.

Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Child in His Family: Vulnerable Children, Vol. 4, *edited by E. James Anthony, M.D., Cyrille Koupernik, M.D., and Colette Chiland, M.D., Ph.D. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 660 pp., $25.95.*

Theory and Problems of Adolescent Development, 2nd ed., *by David P. Ausubel, M.D., Ph.D., Raymond Montemayer, Ph.D., and Pergrouhi (Najarian) Svajian, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 541 pp., $19.50.*

Aging and Behavior: A Comprehensive Integration of Research Findings, 2nd ed., *by Jack Botwinick. New York, N.Y., Springer Publishing Co., 1978, 398 pp., $16.50.*

Controversy in Psychiatry, *edited by John Paul Brady, M.D., and H. Keith H. Brodie, M.D. Philadelphia, Pa., W.B. Saunders Co., 1978, 1,071 pp., $39.50.*

Language of Autistic Children, *by Don W. Churchill. Washington, D.C., V.H. Winston & Sons (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1978, 135 pp., $12.95.*

Chronic Hemodialysis as a Way of Life, *by J.S. Czaczkes, Ph.D., M.D., and A. Kaplan De-Nour, M.D. New York, N.Y., Brunner/Mazel, 1978, 223 pp., $15.00.*

The Bio-Medical Fix: Human Dimensions of Bio-Medical Technologies, *by David L. Ellison. Westport, Conn., Greenwood Press, 1978, 163 pp., $15.95.*

Album des Expressions du Visage, *by René Ermiane and Edmund Gergerian. Paris, France, Pensée Universelle, 1978, 216 pp., no price listed (paper).*

The Second Season: Life, Love, and Sex—Women in the Middle Years, *by Estelle Fuchs, Ph.D. Garden City, N.Y., Anchor Press (Doubleday & Co.), 1978, 280 pp., $8.95.*

Legal and Ethical Issues in Human Research and Treatment: Psychopharmacologic Considerations, *edited by Donald M. Gallant, M.D., and Robert Force, LL.M. New York, N.Y., SP Medical & Scientific Books, Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1978, 180 pp., $15.00.*

Progress in Sexology: Selected Papers from the Proceedings of the 1976 International Congress of Sexology, *edited by Robert Gemme and Connie Christine Wheeler. New York, N.Y., Plenum Press, 1978, 593 pp., $32.50.*

Psychoanalysis, Psychotherapy and the New England Medical Scene, 1894–1944, *by George E. Gifford, Jr. New York, N.Y., Science History Publications/USA (Neale Watson Academic Publications), 1978, 430 pp., $35.00.*

Undergraduate Medical Education and the Elective System: Experience with the Duke Curriculum, 1966–1975, *edited by James F. Gifford, Jr.; William G. Anlyan, M.D., Ewald W. Busse, M.D., and Thomas D. Kinney, M.D., supervising editors. Durham, N.C., Duke University Press, 1978, 243 pp., $9.75.*

A Systems Approach to Alcohol Treatment, *by Frederick B. Glaser, M.D., Stephanie W. Greenberg, M.A., and Morris Barrett, M.P.H. Toronto, Ont., Canada, Addiction Research Foundation, 1978, 303 pp., $14.95 (paper).*

Fish's Outline of Psychiatry for Students and Practitioners, 3rd ed., *edited by Max Hamilton. Bristol, England, John Wright & Sons, 1978, 269 pp., no price listed (paper).*

Public Relations in Psychiatry, *edited by M.T. Haslam, M.A., M.D., D.M.J. York, England, Society of Clinical Psychiatrists, 1976, 28 pp., 50 British pence (paper).*

Growing Pains: A Study of Teenage Distress, *by Edna M. Irwin. Totowa, N.J., Woburn Press, 1978, 304 pp., $14.00.*

Research Advances in Alcohol and Drug Problems, Vol. 4, *edited by Yedy Israel, Frederick B. Glaser, Harold Kalant, Robert E. Popham, Wolfgang Schmidt, and Reginald G. Smart. New York, N.Y., Plenum Press, 1978, 485 pp., $39.50.*

Stimulants: Handbook of Psychopharmacology, Vol. 11, *edited by Leslie L. Iversen, Susan D. Iversen, and Solomon H. Snyder. New York, N.Y., Plenum Press, 1978, 464 pp., $32.50.*

Affective Disorders: Drug Actions in Animals and Man. Handbook of Psychopharmacology, Vol. 14, *edited by Leslie L.*

Iversen, Susan D. Iversen, and Solomon H. Snyder. New York, N.Y., Plenum Press, 1978, 369 pp., $29.50.

Born to Win: Transactional Analysis with Gestalt Experiments, *by Muriel James and Dorothy Jongeward. New York, N.Y., New American Library, 1978, 314 pp., $2.50 (paper).*

The Listening Process, *by Robert Langs, M.D. New York, N.Y., Jason Aronson, 1978, 655 pp., $30.00.*

Organic Psychiatry: The Psychological Consequences of Cerebral Disorder, *by William Alwyn Lishman, B.Sc., D.P.M. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1978, 967 pp., $75.00.*

Progress in Experimental Personality Research, Vol. 8, *edited by Brendan A. Maher. New York, N.Y., Academic Press, 1978, 340 pp., $23.00.*

Find Your Perfect High, *by John Armstrong Marshall. Glendale, Calif., Happy Eye Enterprises, 1978, 189 pp., $5.95 (paper).*

Applied Dream Analysis: A Jungian Approach, *by Mary Ann Mattoon. New York, N.Y., Halsted Press (John Wiley & Sons), 1978, 240 pp., no price listed.*

Child Behavior Problems: An Empirical Approach to Management, *by Roger McAuley and Patricia McAuley. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 233 pp., $13.95.*

Signs of Stress: The Social Problems of Psychiatric Illness, *by J. Wallace McCulloch and Herschel A. Prins. Totowa, N.J., Woburn Press, 1978, 195 pp., $14.00.*

The Paranoid Process, *by W.W. Meissner, S.J., M.D. New York, N.Y., Jason Aronson, 1978, 840 pp., $30.00.*

Brain Dysfunction in Aggressive Criminals, *by Russell R. Monroe, M.D. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 211 pp., $17.95.*

Soul Force: Releasing the Power to Make the Rest of Your Life Work, *by Dr. Paul Olsen. New York, N.Y., M. Evans & Co., 1978, 199 pp., $8.95.*

The Art of Writing Successful R&D Proposals, *by Donald C. Orlich and Patricia Rend Orlich. Pleasantville, N.Y., Redgrave Publishing Co., 1978, 69 pp., no price listed (paper).*

The New Sex Education: The Sex Educator's Resource Book, *edited by Herbert A. Otto. Chicago, Ill., Association Press (Follett Publishing Co.), 1978, 364 pp., $14.95.*

Physician Compensation, *edited by David T. Pieroni. Germantown, Md., Aspen Systems Corp., 1978, 138 pp., no price listed (reprinted from Topics in Health Care Financing).*

Insanity: A Study of Major Psychiatric Disorders, *by Robert G. Priest with a chapter by Dr. J. Steinert. Totowa, N.J., Woburn Press, 1978, 388 pp., $14.00.*

The Prevention of Sexual Disorders: Issues and *edited by C. Brandon Qualls, John P. Wincze* [illegible] *Barlow. New York, N.Y., Plenum Press,* [illegible] *$14.95.*

Classic American Graffiti: Lexical Evidence fro [illegible] **raphy in Western North America,** *by Allen* [illegible] *Waukesha, Wis., Maledicta Press, 1978, 83 pp* [illegible] *ed (paper).*

A Developmental Approach to Problems of Ad [illegible] **vised ed.,** *edited by Eveoleen N. Rexford. Ne* [illegible] *International Universities Press, 1978, 336 pp* [illegible]

Patients, Practitioners, and Medical Care: Aspe [illegible] **Sociology, 2nd ed.,** *by David Robinson. Lond* [illegible] *William Heinemann Medical Books, 1978,* [illegible] *(paper).*

Karen Horney: Gentle Rebel of Psychoanalys [illegible] *Rubins. New York, N.Y., Dial Press (Dell Pub.* [illegible] *350 pp., $9.95.*

International Collaboration: Problems and Opp [illegible] **ited by Barbara Rutledge and E. Kaye Ful** [illegible] *Ont., Canada, Addiction Research Foundat* [illegible] *pp., $14.95 (paper).*

Autism: A Reappraisal of Concepts and Treatm [illegible] *Michael Rutter and Eric Schopler. New York* [illegible] *Press, 1978, 517 pp., $24.50.*

Manual of Neurologic Therapeutics with Essen [illegible] **sis,** *edited by Martin A. Samuels, M.D. B* [illegible] *Little, Brown and Co., 1978, 398 pp., $12.50 (s* [illegible]

A Professional's Guide to Working with the [illegible] **abled Child,** *by Selma Sapir and Bernice Wilso* [illegible] *N.Y., Brunner/Mazel, 1978, 228 pp., $13.50.*

Sociocultural Roots of Mental Illness: An Epide [illegible] **vey,** *by John J. Schwab, M.D., and Mary E.* [illegible] *New York, N.Y., Plenum Medical Book Co.,* [illegible] *$27.50.*

Pain Control in Dentistry: Diagnosis and Ma [illegible] *Samuel Seltzer, D.D.S. Philadelphia, Pa., J.* [illegible] *Co., 1978, 221 pp., $17.00.*

California Mental Health: The Struggle for [illegible] *E. Simmons. Sacramento, Calif., General* [illegible] *lications, 1978, 284 pp., no price listed (paper).*

The Psychopath in Society, *by Robert J. Smi* [illegible] *N.Y., Academic Press, 1978, 157 pp., $7.80.*

No Easy Answers: The Learning Disabled Chil [illegible] *Smith. Rockville, Md., National Institute of M* [illegible] *1978, 131 pp., no price listed (paper).*

The Human Mind Revisited: Essays in Honor of [illegible] **ninger,** *edited by Sydney Smith, Ph.D. New Y* [illegible] *ternational Universities Press, 1978, 492 pp.,* [illegible]

Minority Issues in Mental Health, *by William* [illegible] *Ann Kathleen Burlew, Myrtis Hall Mosley, a* [illegible]

Whitney. Reading, Mass., Addison-Wesley Publishing Co., 1978, 216 pp., $4.95 (paper).

Knowing Your Child Through His Handwriting and Drawings, *by Shirl Solomon. New York, N.Y., Crown Publishers, 1978, 174 pp., $8.95.*

Puberty and Adolescence, *by Barbara Baker Sommer. New York, N.Y., Oxford University Press, 1978, 253 pp., $8.95; $4.00 (paper).*

Psychosomatic Disorders in Childhood, *by Melitta Sperling, M.D.; edited by Otto E. Sperling, M.D. New York, N.Y., Jason Aronson, 1978, 406 pp., $20.00.*

Trance and Treatment: Clinical Uses of Hypnosis, *by Herbert Spiegel, M.D., and David Spiegel, M.D. New York, N.Y., Basic Books, 1978, 370 pp., $18.50.*

Mental Health in the Metropolis: The Midtown Manhattan Study, revised ed., *by Leo Srole, Thomas S. Langner, Stanley T. Michael, Price Kirkpatrick, Marvin K. Opler, and Thomas A.C. Rennie; edited by Leo Srole and Anita Kassen Fischer. New York, N.Y., New York University Press, 1978, 530 pp., $24.50.*

Psychiatry for Students, 5th ed., *by David Stafford-Clark, M.D., and Andrew C. Smith, M.B. Boston, Mass., Allen & Unwin, 1978, 326 pp., $16.25; $9.95 (paper).*

The Lesbian Couple, *by Donna M. Tanner. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 135 pp., $13.50.*

What Does the Ruling Class Do When It Rules? State Apparatuses and State Power Under Feudalism, Capitalism and Socialism, *by Göran Therborn. London, England, NLB (New York, N.Y., Schocken Books, distributor), 1978, 283 pp., $14.95.*

James Jackson Putnam: From Neurology to Psychoanalysis. A Study of the Reception and Promulgation of Freudian Psychoanalytic Theory in America, 1895–1918, *by Russell G. Vasile, M.D. Oceanside, N.Y., Dabor Science Publications, 1978, 115 pp., $14.95.*

Methoden der Klinischen Pharmakologie, *edited by Herausgegeben von Helmut and Paul Kuemmerle. Munich, Germany, Urban & Schwarzenberg, 1978, 783 pp., no price listed.*

Law and Psychiatry: Proceedings of an International Symposium Held at the Clarke Institute of Psychiatry, Toronto, Canada, February 1977, *edited by David N. Weisstub. New York, N.Y., Pergamon Press, 1978, 175 pp., no price listed.*

Elements of Psychopathology: The Mechanisms of Defense, *by Robert B. White, M.D., and Robert M. Gilliland, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1978, 173 pp., $14.25.*

Reasoning About Madness, *by J.K. Wing. New York, N.Y., Oxford University Press, 1978, 260 pp., $9.95.*

Interpersonal Psychoanalysis: New Directions, *edited by Earl G. Witenberg, M.D. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1978, 150 pp., $16.95.*

Group Therapy 1978: An Overview, *edited by Lewis R. Wolberg, M.D., Marvin L. Aronson, Ph.D., and Arlene R. Wolberg, M.S.S. New York, N.Y., Stratton Intercontinental Medical Book Corp., 1978, 205 pp., $19.75 (paper).*

Limits of Medicine: The Doctor's Job in the Coming Era, *edited by Stewart G. Wolf and Beatrice Bishop Berle. New York, N.Y., Plenum Press, 1978, 145 pp., $22.50.*

Early Child Care in Poland, *by Maria Ziemska. New York, N.Y., Gordon and Breach, 1978, 148 pp., $16.75.*

OFFICIAL ACTIONS

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in Los Angeles, Calif., April 17–18, 1978.

PSYCHIATRY

Adler, Kenneth E., M.D., Palm Desert, Calif.
Allen, James R., M.D., Tulsa, Okla.
Allport, Frederick W., M.D., APO New York, N.Y.
Alston, John F., M.D., Evergreen, Colo.
Altman, David B., M.D., Skokie, Ill.
Alvarez, Enrique, M.D., Goldsboro, N.C.
Amparo, Adonis C., M.D., Tiffin, Ohio
Anderson, Douglas J., M.D., Huntington Beach, Calif.
Aportela, Sinesio Z., M.D., Andrews AFB, Md.
Ardali, Cahit, M.D., Goshen, N.Y.
Armentano, Anthony J., M.D., Albany, N.Y.
Artal, Michael, M.D., Rancho Palos Verdes, Calif.
Auric, Robert, M.D., Ojai, Calif.
Axelrad, Allan D., M.D., Sacramento, Calif.

Banta, Richard G., M.D., Rockford, Ill.
Barley, Leonard V., Jr., M.D., San Antonio, Tex.
Battista, John R., M.D., Davis, Calif.
Bayardo, Jose Fernando, M.D., San Diego, Calif.
Beittel, Daniel C., M.D., San Rafael, Calif.
Bergmann, Barbara A., M.D., Freeland, Wash.
Berland, David I., M.D., Topeka, Kan.
Berman, Jonathan D., M.D., Sky Forest, Calif.
Bezcex, Patrick T., M.D., Santa Monica, Calif.
Bieri, Michael D., M.D., Des Peres, Mont.
Biernoff, Michael, M.D., Gallup, N.M.
Billings, Charles K., Jr., M.D., New Orleans, La.
Binder, Renee L., M.D., Mill Valley, Calif.
Bjorksten, Oliver J., M.D., Charleston, S. C.
Blevins, James W., M.D., Beaver Dam, Wis.
Blumenthal, Jerome B., M.D., La Canda, Calif.
Braid, Byron S., M.D., Junction City, Kan.
Briones, David F., M.D., El Paso, Tex.
Brovar, Alan, M.D., Santa Monica, Calif.
Brown, Paul H., M.D., Diamond Bar, Calif.
Buchanan, Robert W., M.D., Chicago, Ill.
Buerger, Helene E., M.D., Los Angeles, Calif.
Bullen, John A., M.D., Oklahoma City, Okla.
Bunes, Robert M., M.D., Santa Monica, Calif.
Burckhardt, William A. III, M.D., Madison, Wis.
Burger, Frances, M.D., Santa Monica, Calif.

Calhoun, William D., M.D., Huntsville, Ala.
Caligayahan-Belen, Nenita, M.D., Bryn Mawr, Calif.
Campbell, James E., M.D., Phoenix, Ariz.
Cannell, Mitchell B., M.D., San Diego, Calif.
Carder, Scott L., M.D., San Marino, Calif.
Carrick, John L., M.D., San Marcos, Tex.
Carrington, Stephens D., M.D., Sheridan, Wyo.
Castellani, Sam, M.D., Galveston, Tex.
Chagi, Darryl, M.D., Aptos, Calif.

Chambers, Aubrey P., M.D., Fort Smith, Ark.
Charry, Dana, M.D., Philadelphia, Pa.
Chesen, Eli S., M.D., Lincoln, Neb.
Chesluk, David J., M.D., Santa Cruz, Calif.
Chinn, Terrance A., M.D., Bellevue, Wash.
Chor, Philip N., M.D., Chicago, Ill.
Chua, Streamson T., M.D., Kingston, N.Y.
Clapp, William L., M.D., Denver, Colo.
Clifford, Daniel P., M.D., Los Angeles, Calif.
Cogbill, Tracy L., M.D., Los Angeles, Calif.
Coleman, Bernice E., M.D., Honolulu, Hawaii
Collins, William S., M.D., Winston-Salem, N.C.
Combs, Gene N., M.D., Lexington, Ky.
Conroe, Henry G., M.D., Chicago, Ill.
Cooper, Gale S., M.D., El Cajon, Calif.
Covington, Edward C., Jr., M.D., Overland Park, Kan.
Cowan, Judith R., M.D., Fayetteville, Ark.
Crockett, David S., M.D., Santa Cruz, Calif.
Crusen, James J., M.D., Denver, Colo.
Cummings, Robert M., M.D., San Diego, Calif.

Daehler, Robert T., M.D., Phoenix, Ariz.
Daigle, Bradley A., M.D., Sherman Oaks, Calif.
Daniec, Danuta A., M.D., Staunton, Va.
da Silva, J. Carlos, M.D., St. Louis, Mo.
David, Ronald S., M.D., Tucson, Ariz.
Davis, Campbell M., M.D., Bellingham, Wash.
Davis, William H., M.D., Crawfordsville, Ind.
Dean, William M., M.D., New Orleans, La.
Del Valle, Fernando, M.D., St. Louis, Mo.
De Silva, K. Himasiri, M.D., Wyoming, Okla.
De Silva, Sunil P., M.D., New York, N.Y.
Diamond, Ronald, M.D., Madison, Wis.
Dines, Franklin H., M.D., Los Angeles, Calif.
Doroski, Victor S., M.D., Solana Beach, Calif.
Drezner, Jeffrey L., M.D., Beverly Hills, Calif.
Duffourc, Rene C. III, M.D., New Orleans, La.
Duffy, Thomas M., M.D., Northbrook, Ill.

Eckert, Elke D., M.D., Minneapolis, Minn.
Edelman, Milton M., M.D., Tenafly, N.J.
Edelstein, David R., M.D., Chicago, Ill.
El-Mashhady, Afaf A., M.D., St. Louis, Mo.
Eltomi, Abdel M., M.D., St. Louis, Mo.
Estrader, Peter L., M.D., Savannah, Ga.
Ettinger, Alfred, M.D., Great Neck, N.Y.

Faguet, Robert A., M.D., Los Angeles, Calif.
Fair, Geraldine A., M.D., Auburn, Calif.
Fairley, Nora L., M.D., Portland, Ore.
Feinberg, Bernard, M.D., University City, Mo.
Fenn, Howard H., M.D., San Francisco, Calif.
Ferguson, James M., M.D., La Jolla, Calif.

Filice, Robert C., M.D., Naperville, Ill.
Fine, Martin, M.D., Chicago, Ill.
Fischer, Craig, M.D., Los Angeles, Calif.
Flavan, Mary Ann, M.D., Los Angeles, Calif.
Forkin, Daniel, M.D., San Francisco, Calif.
Fox, Charles S., M.D., Chicago, Ill.
Frank, Sidney W., M.D., Highland Park, Ill.
Franks, Darrell D., M.D., Louisville, Ky.
Franks, Ronald D., M.D., Denver, Colo.
Fredman, Gerald S., M.D., Albuquerque, N.M.
Freed, Marcia, M.D., Portland, Ore.
Freeland, H. Theodore, M.D., Oakland, Calif.
Freeman, Larry S., M.D., Sacramento, Calif.
French, Everett E., M.D., Helmuth, N.Y.
Friedmann, Claude T.H., M.D., Sherman Oaks, Calif.

Gabatin, Jose Q., M.D., APO San Francisco, Calif.
Gallaher, Marian E., M.D., Los Angeles, Calif.
Garcia-Ferrer, Eduardo A., M.D., Manchester, Mo.
Gatenby, Arthur E., M.D., Redding, Calif.
Gelfand, Ronald A., M.D., Los Angeles, Calif.
Gibfried, James P., M.D., Glendale, Calif.
Gober, Bruce A., M.D., Chicago, Ill.
Goethe, John W., M.D., New Orleans, La.
Goldstein, David M., M.D., Washington, D.C.
Goodman, Malka L., M.D., St. Paul, Minn.
Goodwin, Jean M., M.D., Albuquerque, N.M.
Gould, Brian S., M.D., San Francisco, Calif.
Grater, Simeon J., M.D., Mercer Island, Wash.
Graves, John S., M.D., Denver, Colo.
Gushin, Stephen R., M.D., New York, N.Y.

Hadley, June L., M.D., Chicago, Ill.
Haidar, Amer S., M.D., Beirut, Lebanon
Hale, Charles H., M.D., Seattle, Wash.
Hamill, William T., M.D., Redwood City, Calif.
Hammitt, Karleen B., M.D., Madison, Ind.
Hankins, Alton B., Jr., M.D., Hitchcock, Tex.
Hannan, Joseph E., Jr., M.D., Los Angeles, Calif.
Hanssmann, Dennett J., M.D., El Macero, Calif.
Haraszti, Joseph S., M.D., Park Ridge, Ill.
Hartstein, Norman B., M.D., Santa Monica, Calif.
Haws, John P., M.D., Boulder, Colo.
Heiman, Michael F., M.D., Stockton, Calif.
Henn, Ralph F., M.D., Columbus, Ohio
Heron, Geoffrey B., M.D., Denver, Colo.
Hicks, Philip S., M.D., San Francisco, Calif.
Higginbotham, Ronald, M.D., Palos Verdes, Calif.
Hill, Paul S., M.D., Temple, Tex.
Hilton, George Q., M.D., Dover, N.H.
Hoffman, Howard, M.D., San Francisco, Calif.
Hogrebe, James N., M.D., S. Laguna, Calif.
Holbrook, Thomas L., M.D., Hartland, Wis.
Hollingsworth, Charles E., M.D., Sherman Oaks, Calif.
Hong, Inpow, M.D., Nashville, Tenn.
Horton, John M., M.D., San Francisco, Calif.
Hsu, Jing, M.D., Honolulu, Hawaii
Hu, Jun-teh, M.D., Houston, Tex.
Huddleston, James E., Jr., M.D., Midland, Tex.

Jamieson, John, M.B., Fargo, N.D.
Janicak, Philip G., M.D., Maywood, Ill.
Jones, Edwin C., M.D., Springdale, Ark.

Kaldor, Bruce T., M.D., Sacramento, Calif.
Kaplan, Barnett, M.D., Seattle, Wash.
Katze, Jennifer A., M.D., Memphis, Tenn.
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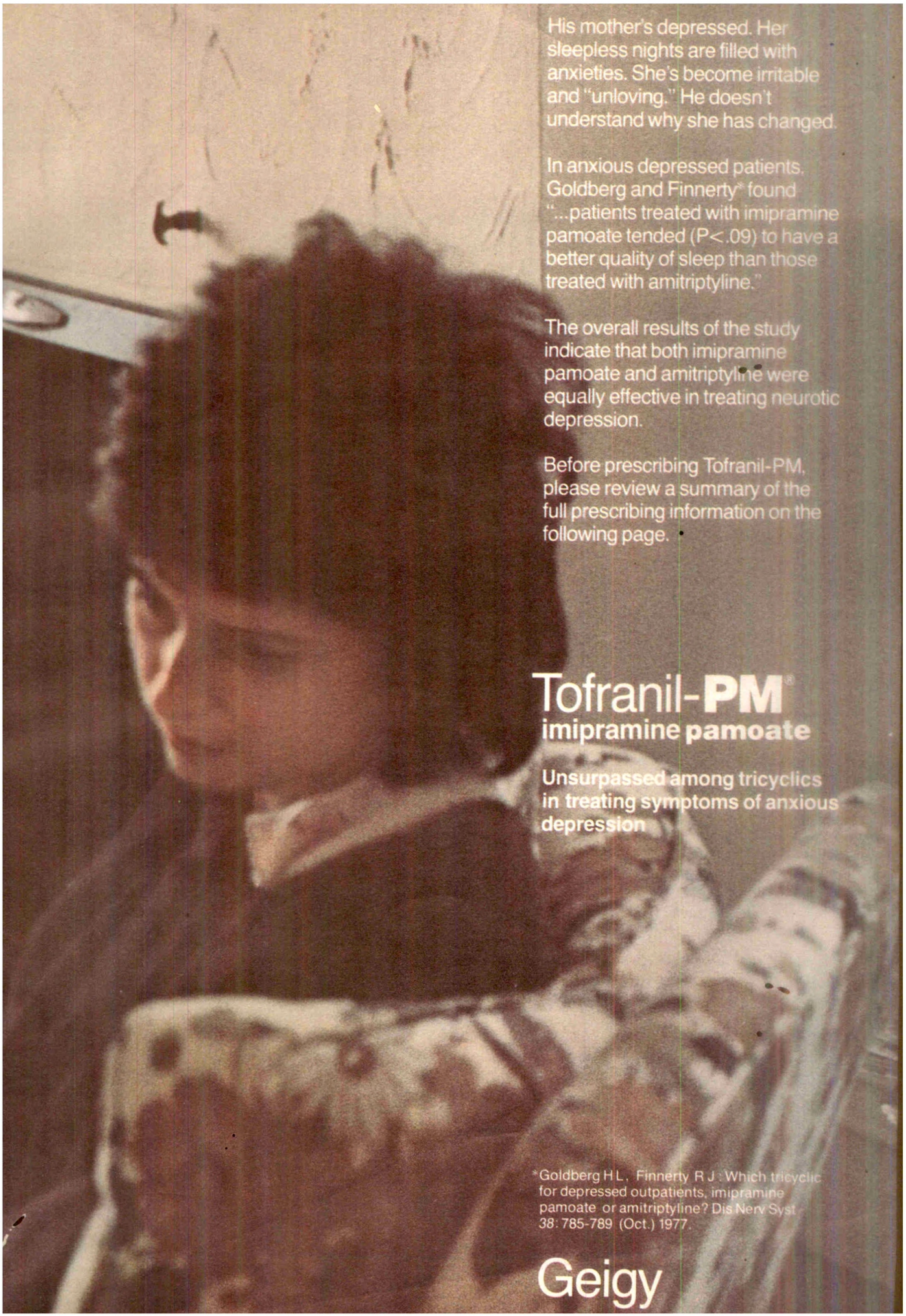
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Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)

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For complete details, including dosage and administration, please refer to the full prescribing information.

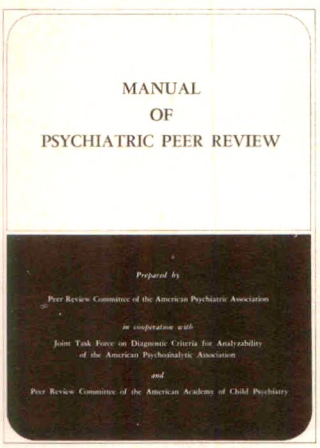
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MANUAL OF PSYCHIATRIC PEER REVIEW

by the Peer Review Committee of the American Psychiatric Association, Donald G. Langsley, M.D., Chairperson in cooperation with the Joint Task Force on Diagnostic Criteria for Analyzability of the American Psychoanalytic Association, William Offenkrantz, M.D., Chairperson, and the Peer Review Committee of the American Academy of Child Psychiatry. Larry B. Silver, M.D., Chairperson.



"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

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SLEEP

Objectively, the sleep laboratory proved relief of insomnia from the first through the 28th night.[1]

IMPROVEMENT IN SLEEP PARAMETERS OVER BASELINE DURING ADMINISTRATION OF DALMANE (flurazepam HCl)[2]
Mean Values of Averaged Nights 1-3, 12-14, 26-28

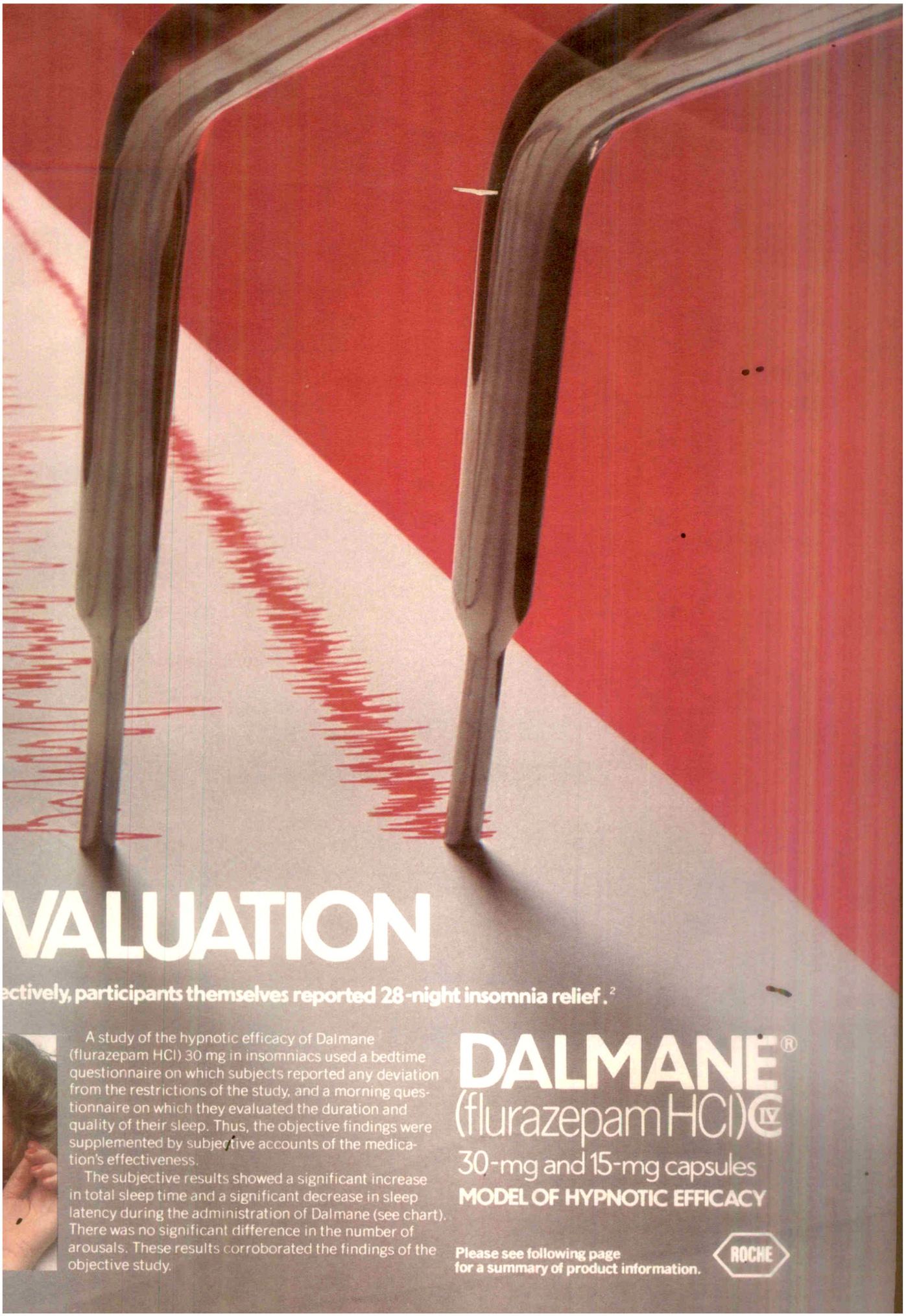
OBJECTIVE		SUBJECTIVE
62.5% less time	time required to fall asleep	45.6%† less time
23.3%† more time	total sleep time	18%† more time

Reductions in minutes required to fall asleep and increases in total sleep time were averaged and expressed as percentages.

A composite of six separate sleep laboratory studies of Dalmane®(flurazepam HCl) provides a comprehensive profile of hypnotic efficacy during short-, intermediate- and longer-term administration. Dalmane in 30 mg dosage was given to 23 insomnia patients for varying periods of time. Results showed that Dalmane was significantly effective in improving sleep induction and sleep maintenance. In addition, following discontinuation of Dalmane there was no sudden insomnia rebound.[1]

In another investigation of Dalmane 30 mg in five chronic insomniacs, the electro-physiologic evidence

eep time
me



VALUATION

ectively, participants themselves reported 28-night insomnia relief.[2]

A study of the hypnotic efficacy of Dalmane (flurazepam HCl) 30 mg in insomniacs used a bedtime questionnaire on which subjects reported any deviation from the restrictions of the study, and a morning questionnaire on which they evaluated the duration and quality of their sleep. Thus, the objective findings were supplemented by subjective accounts of the medication's effectiveness.

The subjective results showed a significant increase in total sleep time and a significant decrease in sleep latency during the administration of Dalmane (see chart). There was no significant difference in the number of arousals. These results corroborated the findings of the objective study.

DALMANE®
(flurazepam HCl) C IV
30-mg and 15-mg capsules
MODEL OF HYPNOTIC EFFICACY

Please see following page for a summary of product information.

ROCHE

28-NIGHT INSOMNIA RELIEF...
PROVED BY THE POLYGRAPH, CORROBORATED BY THE PATIENTS[2]

DALMANE®
(flurazepam HCl)

One 30-mg capsule *h.s.*—usual adult dosage
(15 mg may suffice in some patients).
One 15-mg capsule *h.s.*—recommended dosage for elderly or debilitated patients.

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Objective sleep laboratory data have shown effectiveness for at least 28 consecutive nights of administration. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit dosage to 15 mg to reduce risk of oversedation, dizziness, confusion and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies, or with impaired renal or hepatic function. Periodic blood counts and liver and kidney function tests are advised during repeated therapy.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect.
Adults: 30 mg usual dosage; 15 mg may suffice in some patients.
Elderly or debilitated patients: 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:
1. Kales A, *et al: Clin Pharmacol Ther* 19:576-583, May 1976
2. Dement WC, Guilleminault C, Zarcone V: Progress in clinical sleep research. Scientific exhibit at the American Medical Association, Atlantic City NJ, Jun 14-18, 1975

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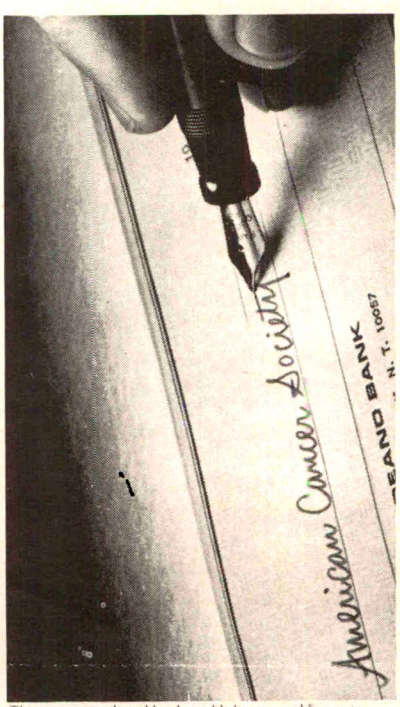
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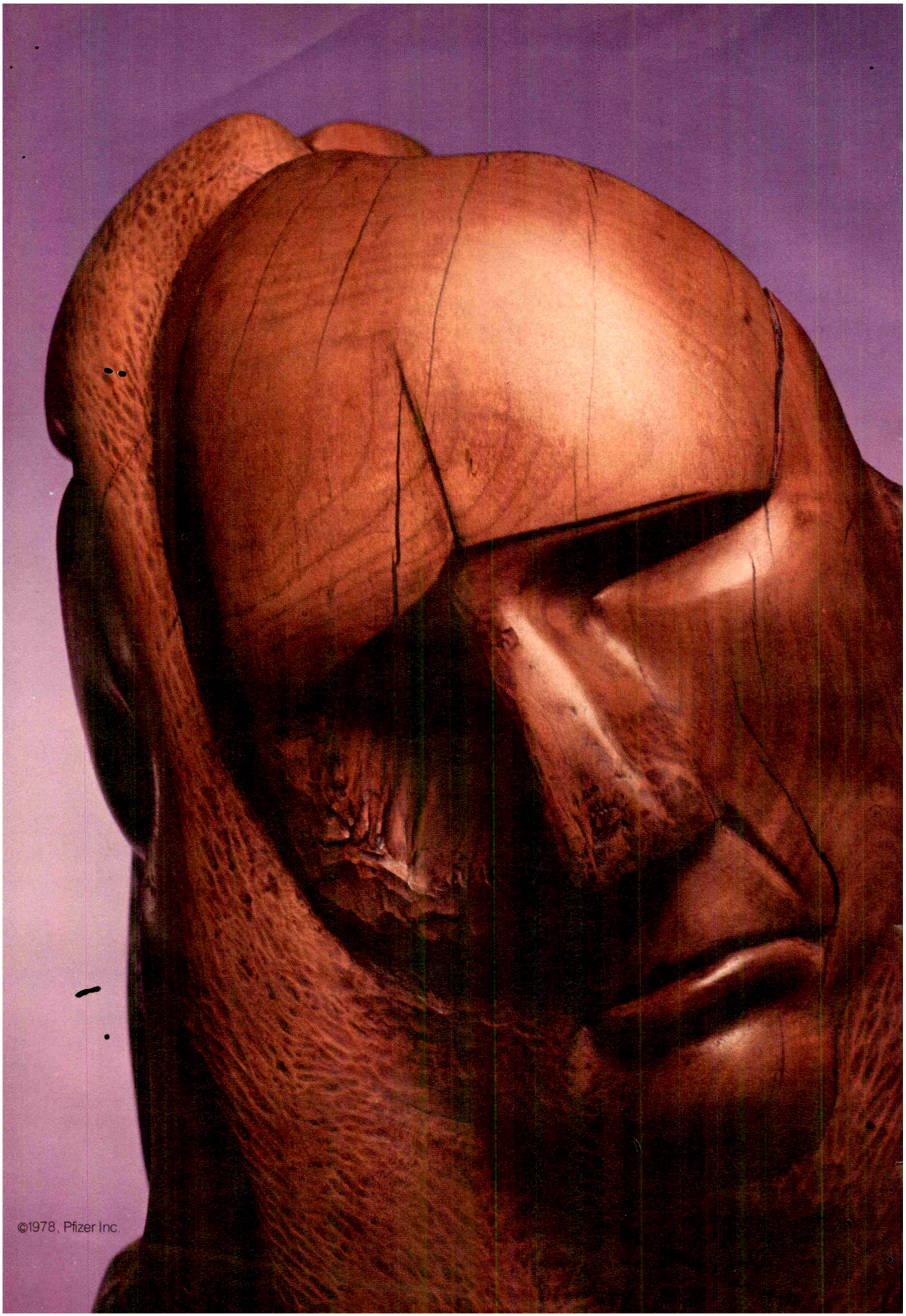
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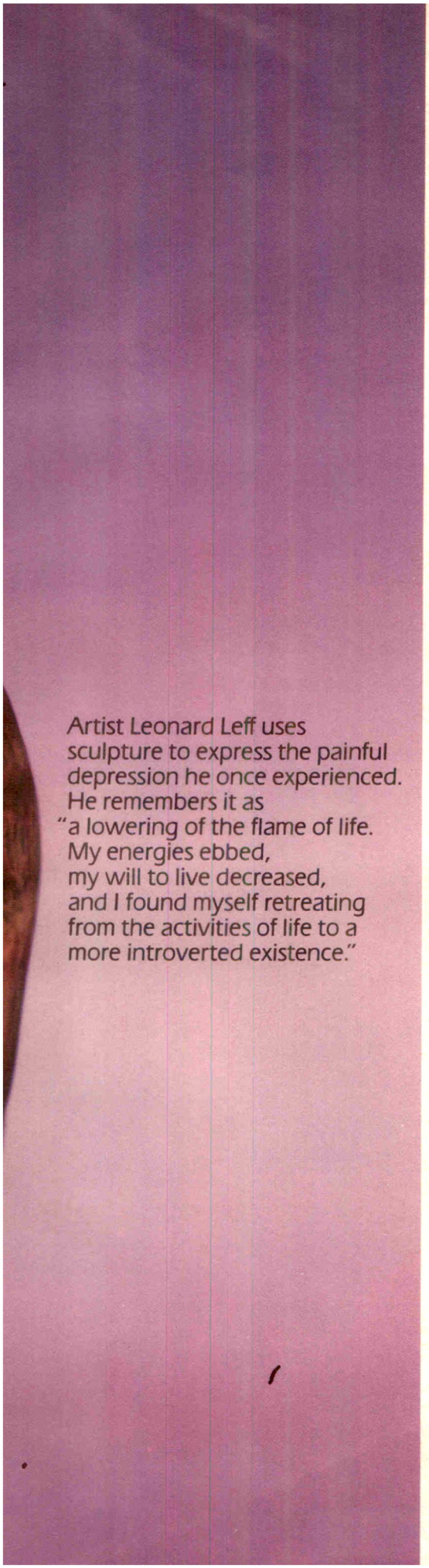
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

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† The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions

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which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

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Tranxene lets you manage anxiety in most patients without problem sedation. Any initial drowsiness is usually transient.

Calming without impairment

Patients not oversedated can function better on the job* or at home. By avoiding excessive sedation, Tranxene helps you calm patients without compromising their capacities.

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(CLORAZEPATE DIPOTASSIUM)

IV 4306CB

Capsules: 3.75, 7.5, 15 mg; Tablets: 11.25, 22.5 mg

*Of course patients should be cautioned against hazardous tasks requiring mental alertness. See overleaf for prescribing information.

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Tranxene® CIV

(CLORAZEPATE DIPOTASSIUM) 4306CB

CAPSULES,
SINGLE DOSE TABLETS

Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

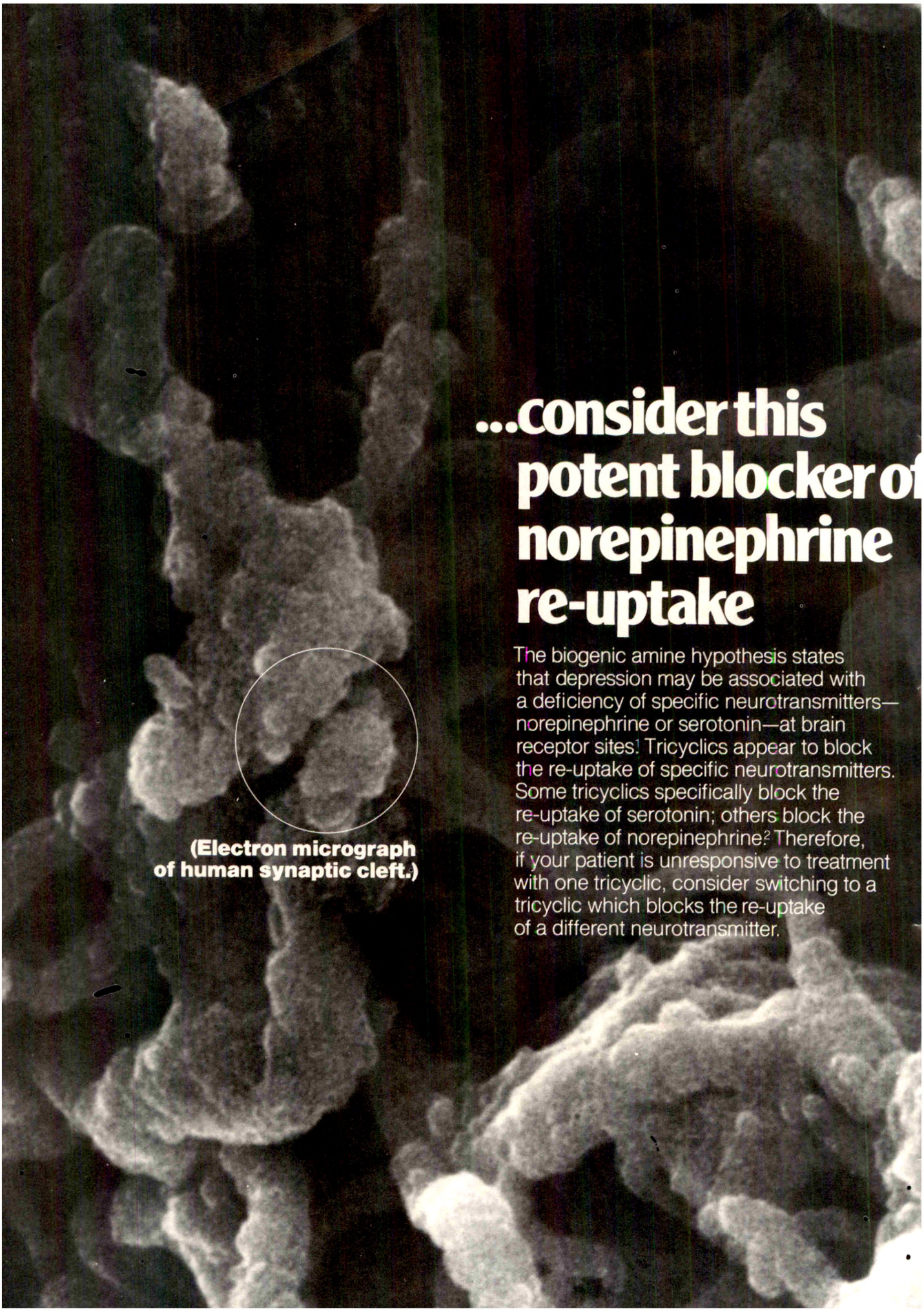
TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

8083272


When your patient is clinically depressed...
"...everything I saw was negative."



...consider this potent blocker of norepinephrine re-uptake

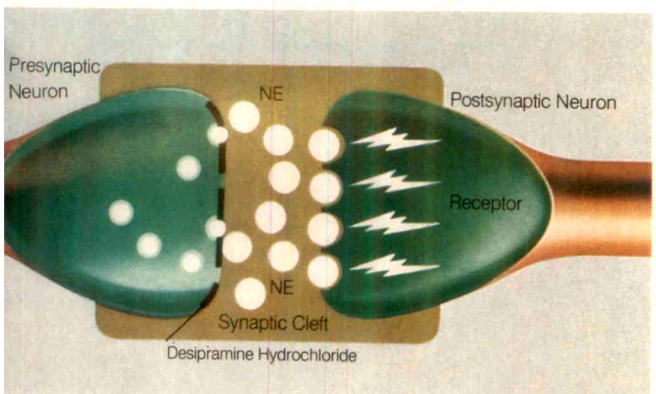
The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

(Electron micrograph of human synaptic cleft.)

Norpramin®
(desipramine hydrochloride tablets NF)
25, 50, 75, 100, 150 mg.

Potent blocker of norepinephrine re-uptake

According to current theory[1,3] some tricyclic antidepressants may fail in certain types of depression because they are not sufficiently potent blockers of norepinephrine re-uptake. Laboratory studies have shown that desipramine is the most potent inhibitor of norepinephrine re-uptake.[4] Norpramin may work in some patients when other tricyclic antidepressants do not.



Consider it now, consider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

References:

1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: **1.** Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. **2.** This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. **3.** USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. **4.** USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. **5.** The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. **6.** In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: **1.** It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. **2.** If serious adverse effects occur, dosage should be reduced or treatment should be altered. **3.** Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. **4.** The drug may cause exacerbation of psychosis in schizophrenic patients. **5.** Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. **6.** Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. **7.** Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. **8.** The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. **9.** This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. **10.** Both elevation and lowering of blood sugar levels have been reported. **11.** Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

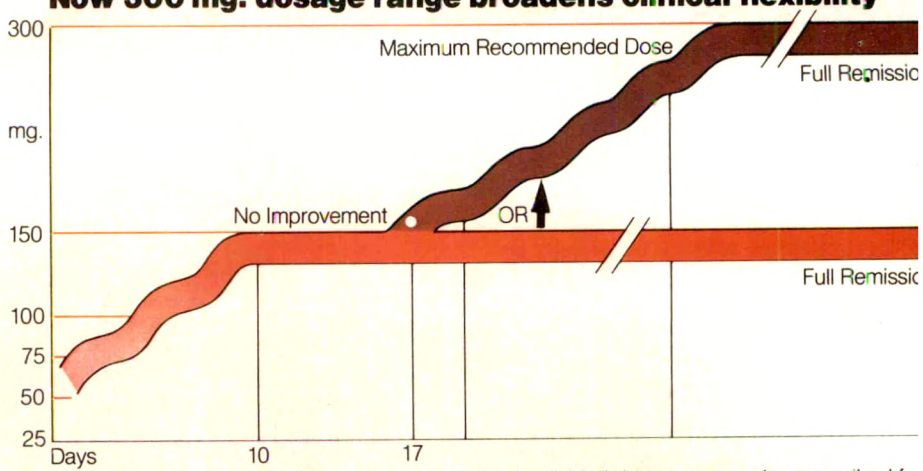
Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of April, 1978



A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed for Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

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8-3866 (Y546A

Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

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FJ:97:34

They thought Ed Sloan was just tired...

maybe he had narcolepsy

A narcoleptic can fall asleep almost anywhere, anytime. The results can be at least embarrassing... at most devastating.

Narcolepsy is often unrecognized

As a primary sleep abnormality narcolepsy is characterized by inappropriate sleepiness, inappropriate sleep episodes, and cataplexy (episodes of muscular weakness precipitated by strong emotion). These are often accompanied by hypnagogic hallucinations, automatic behavior and/or disturbed nocturnal sleep. Sleep apnea may be a severe life-threatening complication of narcolepsy.

It is important to recognize that electroencephalographic studies provide a means of distinguishing narcolepsy from normal sleep.

Besides its medical implications, narcolepsy may have drastic social consequences. "All too often, the patient with disabling hypersomnolence is ignored by physicians for years and even decades, with the consequent tragic toll on his ability to learn and to work."[1]

Ritalin can help in narcolepsy

For narcoleptic sleepiness and sleep episodes, the cited authorities[1,2] most frequently use Ritalin (methylphenidate).

References:

1. Dement WC, Carskadon MA, Guilleminault C, Zarcone VP: Narcolepsy: Diagnosis and Treatment. Primary Care 3:609-623, December 1976.

2. Daly DD, Yoss RE: Narcolepsy, in *Handbook of Clinical Neurology:* Vol 15, *The Epilepsies*, Magnus O, Lorentz De Haas AM (Eds.), Amsterdam, North-Holland Publishing Company, 1974, pp 836-852.

Ritalin®
(methylphenidate)

For the narcoleptics who can't stay awake

Please turn page for brief prescribing information.

CIBA

Ritalin® C II
(methylphenidate)

For the narcoleptics who can't stay awake



Ritalin® hydrochloride C II
(methylphenidate hydrochloride USP)
TABLETS

INDICATION
Narcolepsy.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

DOSAGE AND ADMINISTRATION
Adults
Administer orally in divided doses 2 or 3 times daily, preferably 30 to 45 minutes before meals. Dosage will depend upon indication and individual response.

Average dosage is 20 to 30 mg daily. Some patients may require 40 to 60 mg daily. In others, 10 to 15 mg daily will be adequate. The few patients who are unable to sleep if medication is taken late in the day should take the last dose before 6 p.m.

HOW SUPPLIED
Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500 and 1000.

C76-16 (Rev. 7/76) 665381

Consult complete product literature before prescribing.

174-81652

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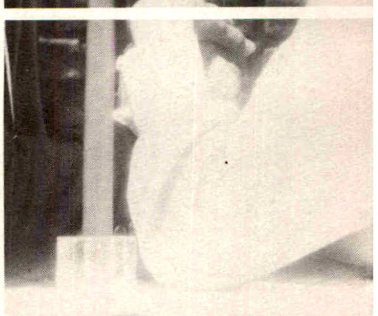


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five dosage strengths now available



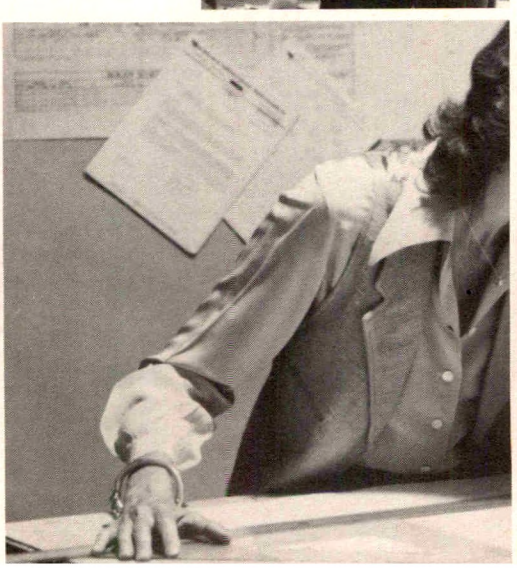
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for initiating therapy
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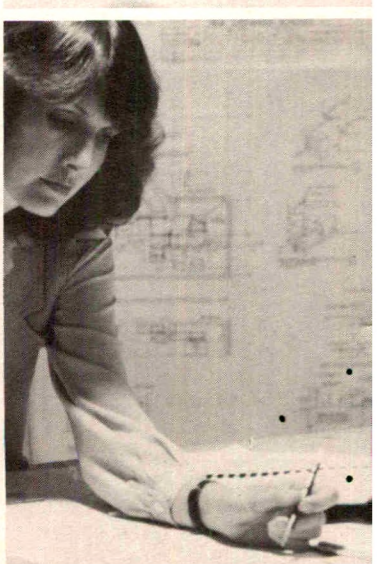




NEW unit-of-use package
bottles of 60 for 30 days of therapy, where appropriate







When TRIAVIL is part of the program, you may anticipate these therapeutic benefits for many patients:

Rapid antianxiety effect
The tranquilizer component can alleviate symptoms of anxiety and agitation within a few days. Hypnotic effects from the tranquilizer component appear to be minimal, particularly in patients permitted to remain active.

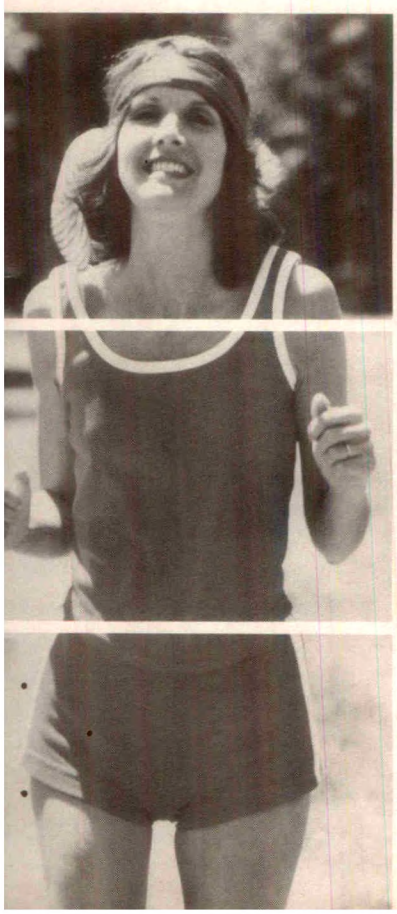
Highly effective antidepressant action
The antidepressant component can help relieve symptoms of depression such as poor concentration and feelings of hopelessness as well as early morning awakening; adequate relief of symptoms may take a few weeks or even longer.

...to aid the psychotherapeutic process
Patients often become more accessible and cooperative; symptomatic relief may enable the patient to function more effectively in his daily activities.

Treatment with TRIAVIL—a balanced view:
TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. TRIAVIL may impair mental and/or physical abilities required for performance of hazardous tasks such as operating machinery or driving. The drug may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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Please see following page for a brief summary of prescribing information.





For a wide range of patients with marked agitation and depression

dual-action Triavil®
containing perphenazine and amitriptyline HCl

now offers more dosage strengths than any other formulation containing a tranquilizer and an antidepressant

dual-action Triavil®

containing perphenazine and amitriptyline HCl

Available:
TRIAVIL® 2-25: Each tablet contains
2 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 2-10: Each tablet contains
2 mg perphenazine and 10 mg amitriptyline HCl.
TRIAVIL® 4-50: Each tablet contains
4 mg perphenazine and 50 mg amitriptyline HCl.
TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides. There is sufficient experimental evidence to conclude that chronic administration of antipsychotic drugs which increase prolactin secretion has the potential to induce mammary neoplasms in rodents under the appropriate conditions. There are recognized differences in the physiological role of prolactin between rodents and humans. Since there are, at present, no adequate epidemiological studies, the relevance to human mammary cancer risk from prolonged exposure to perphenazine and other antipsychotic drugs is not known.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.
Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long-term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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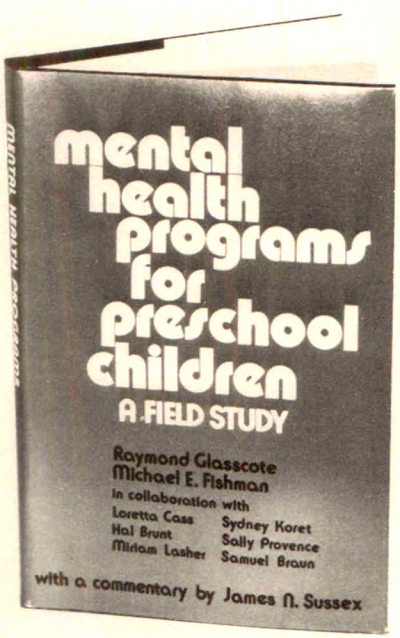
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A Timely New Book from The Joint Information Service

This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

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FLEXIBLE DOSAGE TO MEET THE INTENSITY OF ANXIETY

□ for clinically manifest neurotic or psychoneurotic anxiety, and anxiety associated with somatic disease†

Approximate Milligram Equivalency*

AZENE™ Clorazepate monopotassium	Diazepam	Chlordi-azepoxide	Oxazepam
3.25 mg	2 mg	5 mg	10 mg
6.5 mg	5 mg	10 mg	15 mg
13.0 mg	10 mg	25 mg	30 mg

*Based upon available information from manufacturers' package inserts.

□ an acceptable sedative profile

Drowsiness may occur at initiation of treatment, and with dosage increments.

Brief Summary of Prescribing Information

CONTRAINDICATIONS AZENE™ (clorazepate monopotassium) is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS AZENE™ is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on AZENE™ should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since AZENE™ (clorazepate monopotassium) has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, AZENE™ is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence: Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepates. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Tolerance developed within 3 to 9 days in dogs. Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam and meprobamate) during the first trimester of pregnancy has been suggested in several studies. AZENE™ (clorazepate monopotassium) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to

†The effectiveness of AZENE™ in long-term use, that is, more than four months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.



become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

AZENE™ (clorazepate monopotassium) should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Periodic blood counts and liver function tests are advisable for patients on AZENE™ for prolonged periods. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS The side effect most frequently reported with the use of benzodiazepines is drowsiness. Less commonly reported side effects are: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects include insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit. Decrease in systolic blood pressure has been observed with clorazepates.

DRUG INTERACTIONS If AZENE™ (clorazepate monopotassium) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that clorazepates prolong the sleeping time after hexobarbital or after ethyl alcohol, increase the inhibitory effects of chlorpromazine, but do not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If AZENE™ is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

HOW SUPPLIED AZENE™ (clorazepate monopotassium) is supplied as 3.25 mg and 6.5 mg capsules in bottles of 100 and 500, and as 13 mg capsules in bottles of 100.

6092-1 BS

Manufactured by
Abbott Laboratories
North Chicago, Illinois 60064 for

Endo Laboratories, Inc.
Subsidiary of the DuPont Company
Garden City, New York 11530

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EDO-33-A3/8

ASSISTANT SUPERINTENDENT FOR PROFESSIONAL SERVICES

New Hampshire Hospital, Concord, N.H., an 800 bed JCAH-Accredited psychiatric hospital, is seeking an Assistant Superintendent for Professional Services to be responsible for the direction and coordination of clinical programs for the treatment, care and management of mentally ill patients. New Hampshire Hospital, the only public psychiatric hospital in N.H., is located in Concord, a city of approximately 30,000 people, 75 minutes north of Boston, MA. Salary to $41,690. Possibility of facutly appointment to major university. Housing and other fringe benefits are available. Candidate must be a physician-psychiatrist licensed or eligible for licensure as an M.D. in New Hampshire and certified or eligible for certification by the American Board of Neurology and Psychiatry.

Interested applicants should contact C. Clifton DuBois, Ph.D., Superintendent at New Hampshire Hospital, 105 Pleasant Street, Concord, New Hampshire 03301. Telephone (603) 271-2205.

LIAISON and INPATIENT PSYCHIATRISTS WANTED

Bd. cert. or elig. required. Chillicothe VA Hospital is a 1000-bed hosp. with large aftercare program. JCAH approved. OSMA approved cont. med. ed. prog. for AMA Phy. Recognition Award. Hosp. located in southern Ohio natural recreation and scenic area, 45-min. from Columbus. Financial assistance in moving. Equal Employment Opportunity Employer. Write Chief of Staff, VA Hospital, Chillicothe, OH 45601 or call 614-773-1141, ext. 202.

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Immediate opening for Assistant Director in Department of Psychiatry in 375 bed general teaching hospital in Fort Worth, Texas. Teaching and patient care programs are being developed through affiliation with Department of Psychiatry of Southwestern Medical School at Dallas.

Psychiatrist will assist Director to provide direct patient care, teaching rotating residents, and clinically supervise acute and intermediate treatment programs in two units totaling 58 beds. Therapeutic milieu is eclectic with emphasis on interpersonal, humanistic and psychodynamic approaches. Multidisciplinary professional staff includes four masters level therapists. Many services locally available for patient referrals including outpatient clinic, partial hospitalization, alcohol treatment, vocational development centers, and others.

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Faculty appointment and remuneration will be based on clinical and academic experience.

Please forward curriculum vitae with your initial response to:

Director, Department of Psychiatry
Tarrant County Hospital District
1500 South Main Street
Fort Worth, Texas 76104

MEDICAL AND PSYCHIATRIC DIRECTOR THE BROWN SCHOOLS

We are looking for a board certified child psychiatrist to provide leadership for the medical and clinical staffs of our three JCAH accredited psychiatric facilities, our halfway houses, and our two group homes. Our present program has a capacity of over 525 residents, and expansion is planned into other states in the near future.

Applicants for this position should be experienced in the field of residential treatment and be capable in the areas of administrative duties and responsibilities. Salary is open based on qualifications and excellent fringe benefits are provided. Qualified applicants should send a curriculum vita to:

William Porcher, M.D.
Chairman — Search Committee
P.O. Box 4008
Austin, Texas 78765

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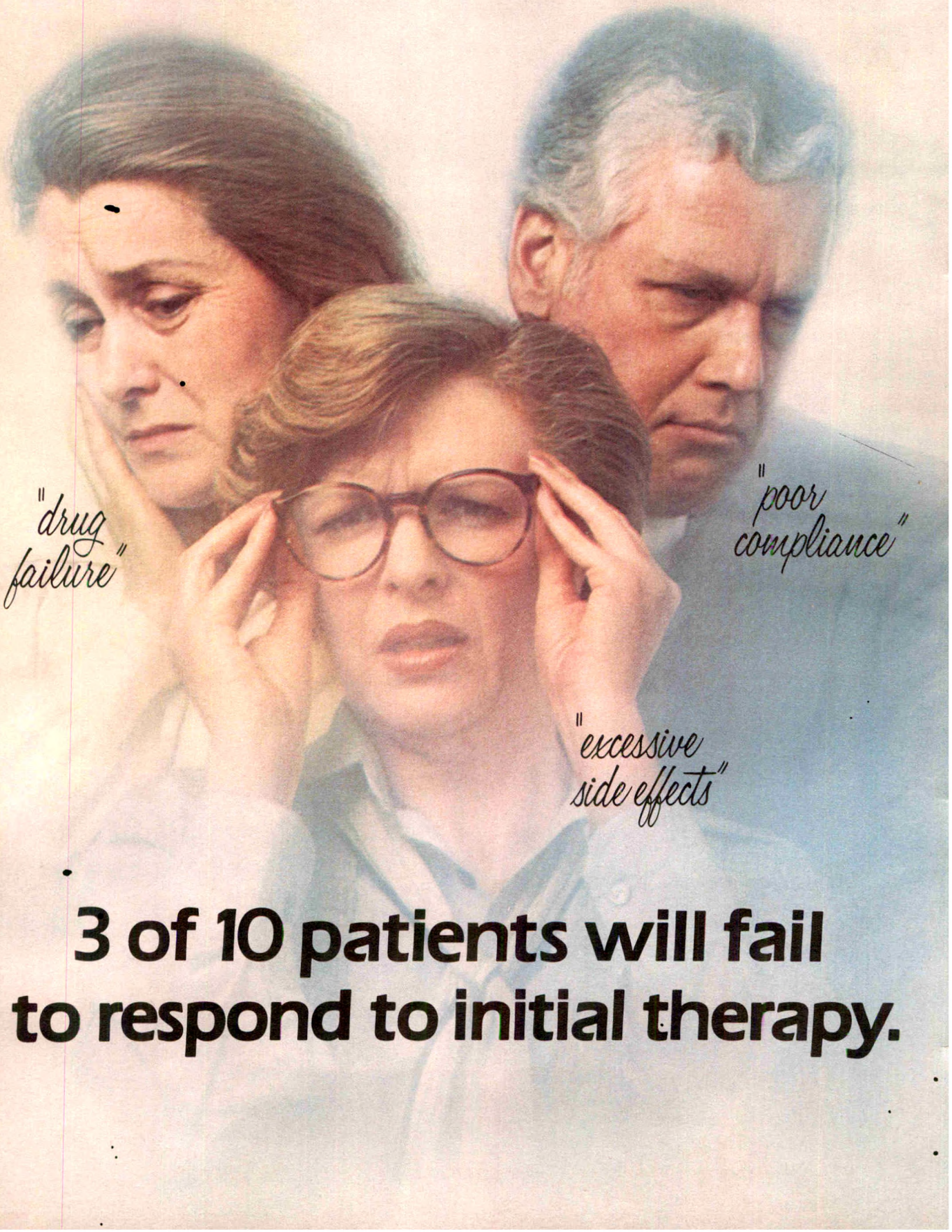
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In depression...



3 of 10 patients will fail to respond to initial therapy.

make

PERTOFRANE® (desipramine HCl NF)

your first choice for the 3 of 10 patients who don't respond.

because of:

- **CLINICAL EFFECTIVENESS** even when "most patients had a history of several depressive episodes, hospitalizations, ECT or poor response to numerous other drugs."[1]

- **LOW SIDE EFFECTS PROFILE** with less sedating properties than amitriptyline or doxepin[2] and less anticholinergic activity.[3]

- **ENHANCED PATIENT ACCEPTANCE** through a flexible dosage schedule plus rapid onset of action.

PERTOFRANE® when they don't respond.
(desipramine HCl NF)

USV LABORATORIES
USV Laboratories Inc.
Manati, P.R. 00701

Please see next page for brief summary of prescribing information and references.

PERTOFRANE®
(desipramine HCl NF)

Indication: For relief of mental depression. **Contraindications:** Do not use MAO inhibitors concomitantly or within 2 weeks of the use of this drug. Hyperpyretic crises or severe convulsive seizures may occur with such combinations; potentiation of adverse reactions can be serious or even fatal. When substituting Pertofrane (desipramine HCl) in patients receiving an MAO inhibitor, allow an interval of at least 14 days. Initial dosage in such patients should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated following recent myocardial infarction and in patients with a known hypersensitivity to tricyclic antidepressants. **Warnings:** Activation of psychosis may occasionally be observed in schizophrenic patients. Due to atropine-like effects and sympathomimetic potentiation, use only with the greatest care in patients with narrow-angle glaucoma or urethral or ureteral spasm. Do not use in patients with the following conditions unless the need outweighs the risk: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (may lower seizure threshold). This drug may block the action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents. Hypertensive episodes have been observed during surgery. The concurrent use of other central nervous system drugs or alcohol may potentiate adverse effects. Since many such drugs may be used during surgery, desipramine should be discontinued prior to elective procedures. The potentiation resulting from excessive use of alcohol may increase the danger inherent in a suicide attempt or overdosage. Caution patients on the possibility of impaired ability to operate a motor vehicle or dangerous machinery. Do not use in women who are or may become pregnant, or in children under 12 years of age, unless the clinical situation warrants the potential risk. Because of increased sensitivity to the drug, use lower than normal dosage in adolescent and geriatric patients. **Precautions:** Potentially suicidal patients require careful supervision and protective measures during therapy. Prescriptions should be limited to small quantities. Discontinuation of the drug may be necessary in the presence of increased agitation and anxiety shifting to hypomanic or manic excitement. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents). Prescribe cautiously in hyperthyroid patients and in those receiving thyroid medications; transient cardiac arrhythmias have occurred in rare instances. Periodic blood and liver studies should supplement careful clinical observations in all patients undergoing extended courses of therapy. **Adverse Reactions:** The following have been reported: *Nervous System:* dizziness, drowsiness, insomnia, headache, disturbed visual accommodation, tremor, unsteadiness, tinnitus, paresthesias, changes in EEG patterns, epileptiform seizures, mild extrapyramidal activity, falling and neuromuscular incoordination. A confusional state (with such symptoms as hallucinations and disorientation), particularly in older patients and at higher dosage, may require discontinuation of the drug. *Gastrointestinal Tract:* anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea. *Skin:* skin rashes (including photosensitization), perspiration and flushing sensations. *Liver:* rare cases of transient jaundice (apparently of an obstructive nature) and liver damage. If jaundice or abnormalities in liver function tests occur, discontinue the drug and investigate. *Blood Elements:* bone-marrow depression, agranulocytosis, thrombocytopenia and purpura. If these occur, discontinue the drug. Transient eosinophilia has been observed. *Cardiovascular System:* orthostatic hypotension and tachycardia. Carefully supervise patients requiring concomitant vasodilating therapy, particularly during initial phases. *Genitourinary System:* urinary frequency or retention and impotence. *Endocrine System:* occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement, and decreased libido and estrogenic effect. *Sensitivity:* urticaria and rare instances of drug fever and cross-sensitivity with imipramine. **Dosage:** All patients except geriatric and adolescent: 75 to 150 mg./day in divided doses or as single daily dose. Dosage may be increased up to 200 mg. daily. Geriatric and adolescent patients should usually be started with lower dosage (25 to 50 mg. daily) and may not tolerate higher doses. Dosage may be increased up to 100 mg. daily. Lower maintenance dosages should be continued for at least 2 months after obtaining a satisfactory response. Therapy may be given in divided doses or as a single daily dose. **How Supplied:** 25 mg. capsules (pink), bottles of 100 and 1000. Also, 50 mg. capsules (maroon and pink), bottles of 100 and 1000.

References:

1. Barringer, T.J.: Desipramine (Pertofrane) in the treatment of depression, Psychosomatics, Third Symposium: Anxiety and Depression 6:326, 1965. 2. Bondurant, W.W. III and Ford, H.: Depression and manic depression in Current Therapy 1976 (Conn, H.F., ed.), Philadelphia, Saunders, 1976, p. 841. 3. Ayd, F.J., Jr.: Central anticholinergic activity and tricyclic antidepressant efficacy, Int. Drug Ther. Newsletter 10:21, 1975.

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Prescribing Information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats Oral Dose —
20 mg/kg/day — 10 days, no adverse effect
40 mg/kg/day — 10 days, no adverse effect

Pregnant Mice Oral Dose —
20 mg/kg/day — 10 days, slight increase resorptions
40 mg/kg/day — 10 days, slight increase resorptions

Pregnant Rabbits Oral Dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50 to 75 mg/day.
- Increase to 100 mg/day in three or four days.
- Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.
- An increase to 225 mg/day may be required in patients with severe symptomatology.
- Some chronic, treatment-resistant patients may require up to 400 mg/day; (however, the long-term safety of 400 mg/day has not been established).

Elderly or debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild — 5 mg to 15 mg three or four times a day.
2. Moderate — 10 mg to 25 mg three or four times a day.
3. Severe — 225 mg/day may be required.

Dosage may be administered once a day.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

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Vol. 135, No. 12 December 1978

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THE AMERICAN JOURNAL OF PSYCHIATRY

In this issue

Genetic Counseling for Psychiatric Patients and Their Families

By Ming T. Tsuang

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Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: The possibility of extrapyramidal reactions from 'Thorazine' may confuse the diagnosis of Reye's syndrome or other encephalopathy. Therefore, avoid use in children or adolescents with suspected Reye's syndrome.

Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or obscure diagnosis of conditions such as intestinal obstruction, brain tumor, and Reye's syndrome (see Warnings). Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.



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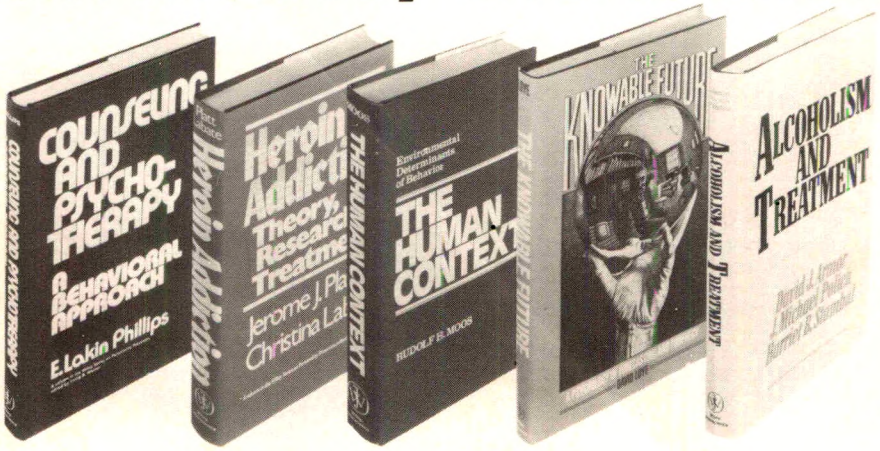
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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26

2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968

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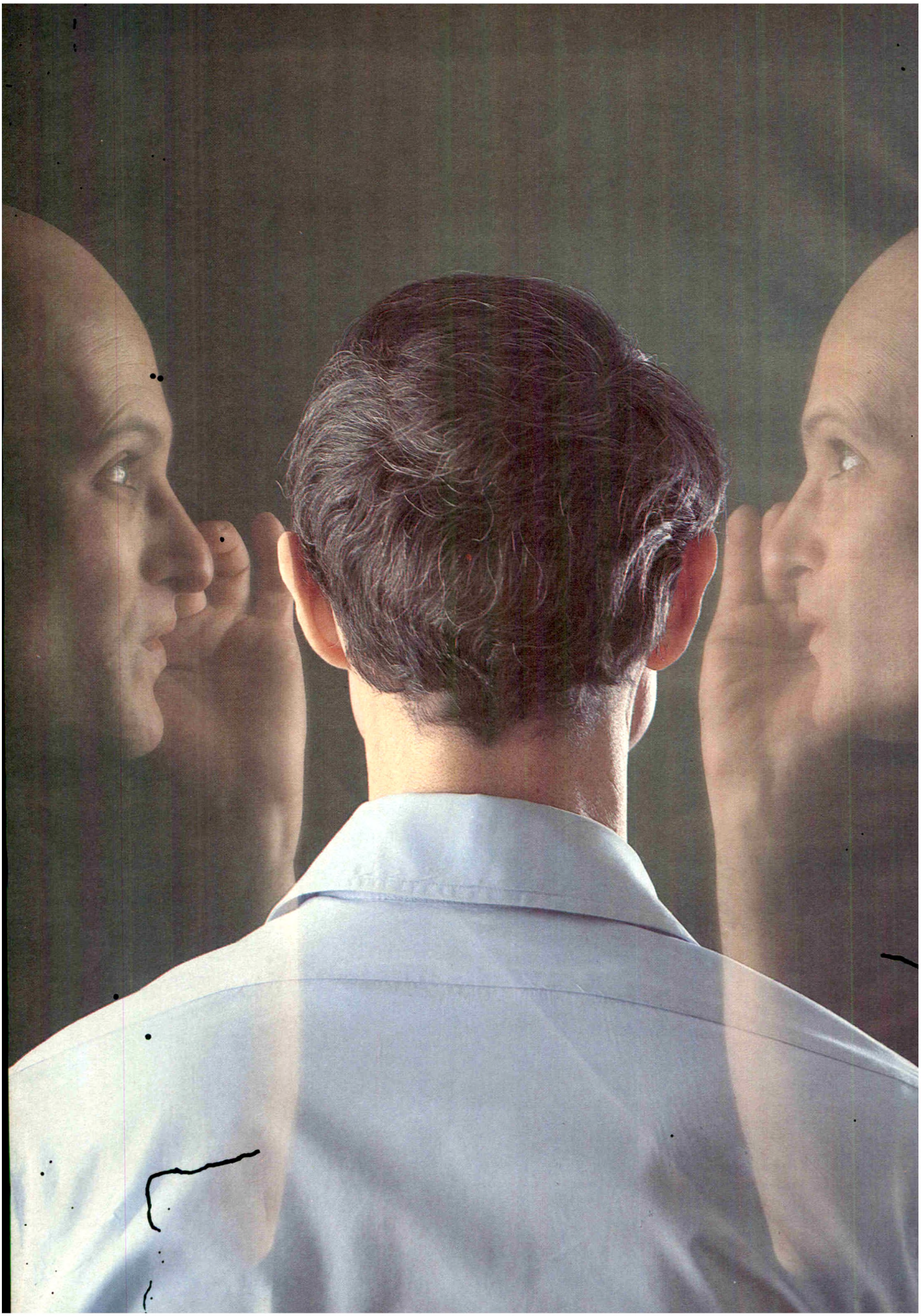
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Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane (thiothixene). Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudo-parkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leukocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg, and in 30 ml (1 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, and 5 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

References. 1. Stotsky BA: Relative efficacy of parenteral haloperidol and thiothixene for the emergency treatment of acutely excited and agitated patients. *Dis Nerv Syst* 38:967-973, 1977. 2. Bressler B, Friedel RO: A comparison between chlorpromazine and thiothixene in a Veterans Administration hospital population. *Psychosomatics* 12:275-277, 1971. 3. Denber HCB, Turns D: Double blind comparison of thiothixene and trifluoperazine in acute schizophrenia. *Psychosomatics* 13:100-104, 1972. 4. Sterlin C, Ban TA, Lehman HE, Saxena BM: The place of thiothixene in the treatment of schizophrenic patients. *Can Psychiatr Assoc J* 15:3-4, 1970. 5. Brauzer B, Goldstein BJ: Comparative effects of intramuscular thiothixene and trifluoperazine in psychotic patients. *J Clin Pharmacol* 8:400-403, 1968. 6. Engelhardt DM, Rudorfer L: The chronic schizophrenic outpatient in an urban community: Social and vocational adjustment. Presented as a Scientific Exhibit at the 130th Annual Meeting, American Psychiatric Association, Toronto, Canada, May 2-6, 1977. 7. Itil TM, Unverdi C, Wohlrabe J, Larsen V, Levitt J: Drug therapy of psychosis associated with organic brain syndrome, presented as a scientific exhibit at the American Public Health Association Centennial, Atlantic City, New Jersey, November 12-16, 1972. 8. Dillenkoffer RL, et al: Electrocardiographic evaluations of schizophrenic patients. Presented as a Scientific Exhibit at the 125th Annual Meeting of the American Psychiatric Association, Dallas, Texas, May 1-4, 1972. 9. Goldstein B, Weiner D, Banas F: Clinical evaluation of thiothixene in chronic ambulatory schizophrenic patients, in Lehmann HE, Ban TA (eds): *Modern Problems in Pharmacopsychiatry*. Basel, S Karger, 1969, vol 2, pp 45-52.

For additional information on Navane, please consult your Roerig representative or write to: Roerig Medical Department, 235 East 42nd Street, New York, NY 10017.

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- not a phenothiazine

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NO incidence	Tolerance development Hepatotoxic changes Ophthalmological changes Excessive weight gain Impotence Decreased libido Skin pigmentation Thyroid function changes Persistent tardive dyskinesia
LOW incidence	Sedation Adverse drug interactions Hypotension Hematological changes Lactation Amenorrhea Autonomic changes Skin rash Nonpersistent symptoms of tardive dyskinesia
MODERATE incidence	Extrapyramidal reactions

*This table compiled from clinical and field reports, according to the best available information.

Further advantages with MOBAN®

The pharmacological effect from a single oral dose persists for 24-36 hours, permitting *h.s.* dosage, once the daily dosage level is established.

Several clinicians have reported that obese patients on MOBAN® frequently experienced a marked reduction in weight toward normal weight.

How to use MOBAN®

The usual starting dose is 50-75 mg/day. You can increase to 100 mg/day in 3-4 days. Titrate individually up or down depending on patient response. Some chronic, treatment-resistant patients may require an increase to 400 mg/day. (However, the long-term safety of 400 mg/day has not been established.) Please refer to complete prescribing information for further dosage instructions.

For complete prescribing information, please see next page.

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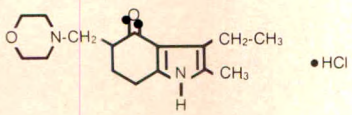
MOBAN®

molindone HCl

5 mg	10 mg	25 mg
Endo	Endo	Endo

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN® (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN® being excreted in urine and feces.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in the pregnant patient have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN® Caution should be exercised where increased activity may be harmful.

MOBAN® does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN® has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological

Extrapyramidal Reactions

Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia

Motor restlessness may occur early.

Parkinson Syndrome

Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome

Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System

Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological

There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN® may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects

Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with MOBAN® therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN®.

Cardiovascular

Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological

Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN® (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN®.

Skin

Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN® usage alone.

MOBAN® (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN® is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia

Although rarely reported with MOBAN® (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50-75 mg/day.

—Increase to 100 mg/day in 3 or 4 days.

—Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.

—An increase to 225 mg/day may be required in patients with severe symptomatology.

—Some chronic, treatment resistant patients, may require up to 400 mg/day (however, the long term safety of 400 mg/day has not been established).

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

Dosages may be administered once a day.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN® in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN® in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles of 100 and 1000 with potencies and colors as follows: 5 mg orange, 10 mg lavender, 25 mg light green.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

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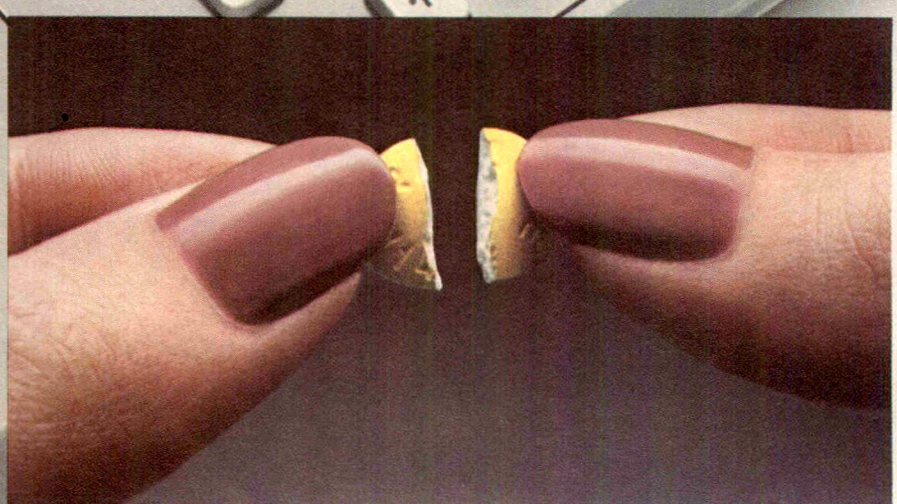
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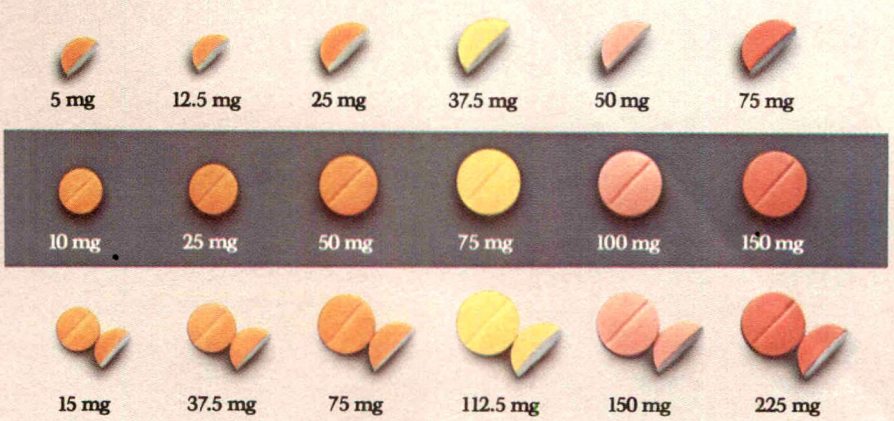
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Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

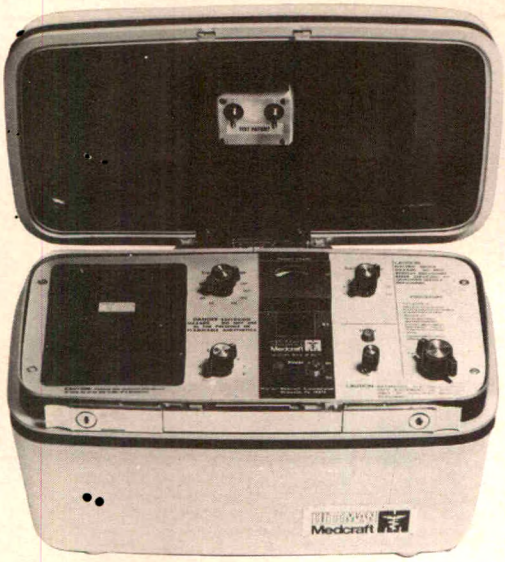
Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Supplied: *Scored Tablets:* 10, 25, 50, 75, 100, 150 mg.

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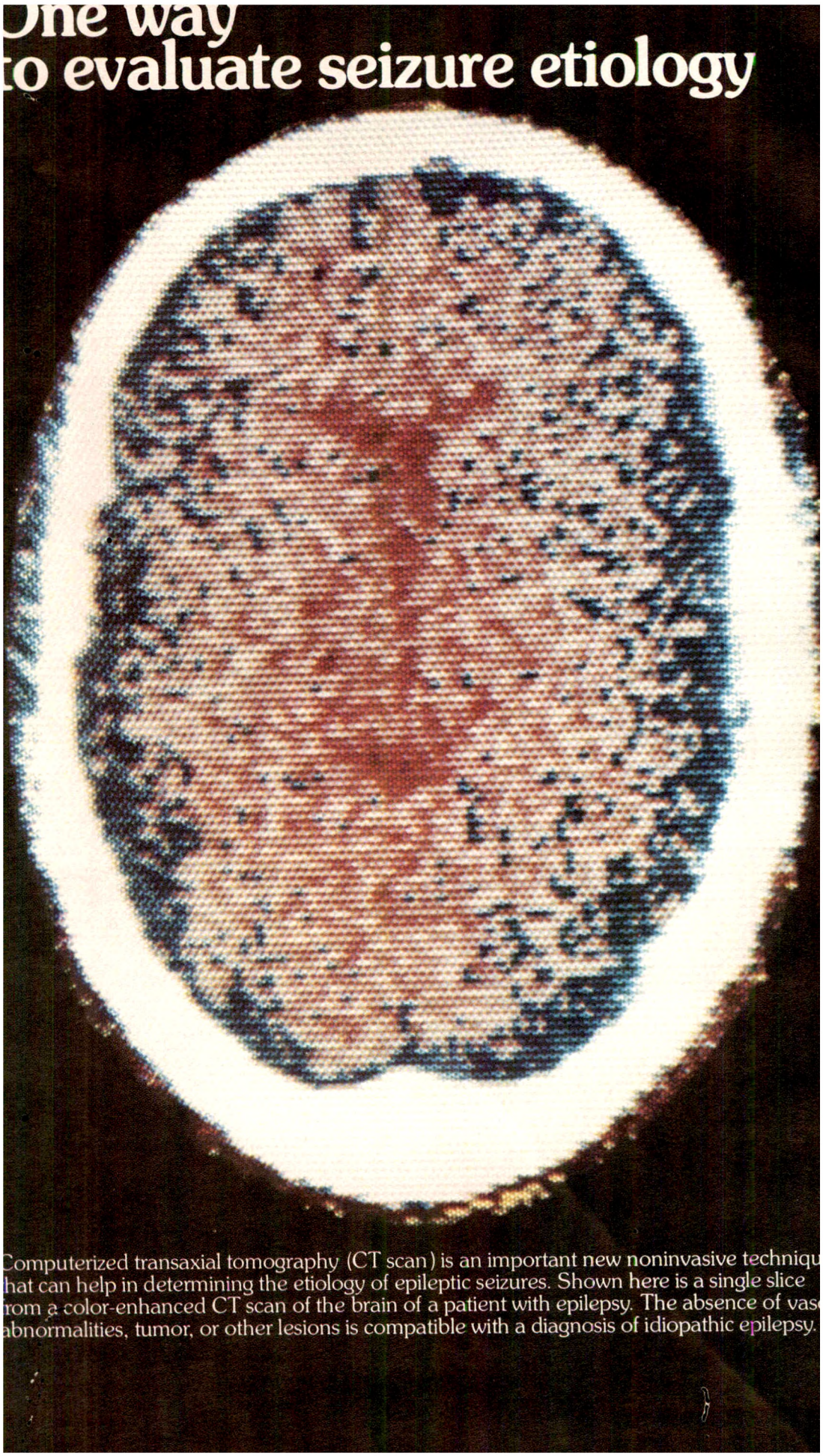
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Computerized transaxial tomography (CT scan) is an important new noninvasive techniqu
that can help in determining the etiology of epileptic seizures. Shown here is a single slice
from a color-enhanced CT scan of the brain of a patient with epilepsy. The absence of vas
abnormalities, tumor, or other lesions is compatible with a diagnosis of idiopathic epilepsy.

A proven way to start seizure control

MYSOLINE®
(primidone)

Tablets 50 mg, 250 mg
Suspension 250 mg/5 ml

A single entity with multiple anticonvulsant properties

Primidone itself has anticonvulsant activity. In addition, its major metabolite, phenylethylmalonamide (PEMA), exerts an anticonvulsant effect of its own and also enhances the anticonvulsant action of the second metabolite, phenobarbital. In patients refractory to phenytoin and phenobarbital, primidone may prove effective as the sole therapy.[1]

Multiple indications—grand mal, psychomotor, and focal epileptic seizures

In grand mal, MYSOLINE is comparable in efficacy to phenytoin and phenobarbital.[2,3] In psychomotor seizures, phenobarbital is rarely effective.[1] In focal seizures, phenytoin used alone is not indicated[4]—and, according to Forster,[3] MYSOLINE is markedly more effective than phenobarbital, which should dispel the misconception that MYSOLINE is "just a phenobarbital." (See chart below.)

Efficacy ratings* of anticonvulsants in epilepsy

Agent	Grand Mal	Psychomotor	Focal
Mysoline	++++	++++	++++
Hydantoinates (phenytoin)	++++	+++	+++
Barbiturates (phenobarbital)	++	++	++

* Relative effectiveness, ++++ being most effective.
Adapted from Forster, F. M.[3]

Consider it first for major motor epilepsies

MYSOLINE® (primidone) Ayerst®

See next page for Brief Summary and references.

Start seizure control with MYSOLINE® (primidone)

Initial dose in children over 8 years of age and adults: 250 mg daily at bedtime

BRIEF SUMMARY
(For full prescribing information, see package circular.)
MYSOLINE®
Brand of **PRIMIDONE** *Anticonvulsant*

ACTIONS: MYSOLINE raises electro- or chemoshock seizure thresholds or alters seizure patterns in experimental animals. The mechanism(s) of primidone's antiepileptic action is not known.

Primidone *per se* has anticonvulsant activity as do its two metabolites, phenobarbital and phenylethylmalonamide (PEMA). In addition to its anticonvulsant activity, PEMA potentiates that of phenobarbital in experimental animals.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several weeks before it can be assessed.

Usage in pregnancy: The effects of MYSOLINE in human pregnancy and nursing infants are unknown.

Recent reports suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Data are more extensive with respect to diphenylhydantoin and phenobarbital, but these are also the most commonly prescribed anticonvulsants; less systematic or anecdotal reports suggest a possible similar association with the use of all known anticonvulsant drugs.

The reports suggesting an elevated incidence of birth defects in children of drug-treated epileptic women cannot be regarded as adequate to prove a definite cause and effect relationship. There are intrinsic methodologic problems in obtaining adequate data on drug teratogenicity in humans; the possibility also exists that other factors, *e.g.*, genetic factors or the epileptic condition itself, may be more important than drug therapy in leading to birth defects. The great majority of mothers on anticonvulsant medication deliver normal infants. It is important to note that anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and threat to life. In individual cases where the severity and frequency of the seizure disorder are such that the removal of medication does not pose a serious threat to the patient, discontinuation of the drug may be considered prior to and during pregnancy, although it cannot be said with any confidence that even minor seizures do not pose some hazard to the developing embryo or fetus.

The prescribing physician will wish to weigh these considerations in treating or counseling epileptic women of childbearing potential.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

PRECAUTIONS: The total daily dosage should not exceed 2 g. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 g per day. The initial dose is 250 mg. Increments of 250 mg are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 g. A typical dosage schedule for the introduction of MYSOLINE is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg daily at bedtime	*2nd Week* 250 mg b.i.d.
3rd Week 250 mg t.i.d.	*4th Week* 250 mg q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg, with gradual weekly increases of 125 mg a day, to a daily total usually between 500 mg and 750 mg.

In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances: for initiation of combination therapy; during "transfer" therapy; for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.).

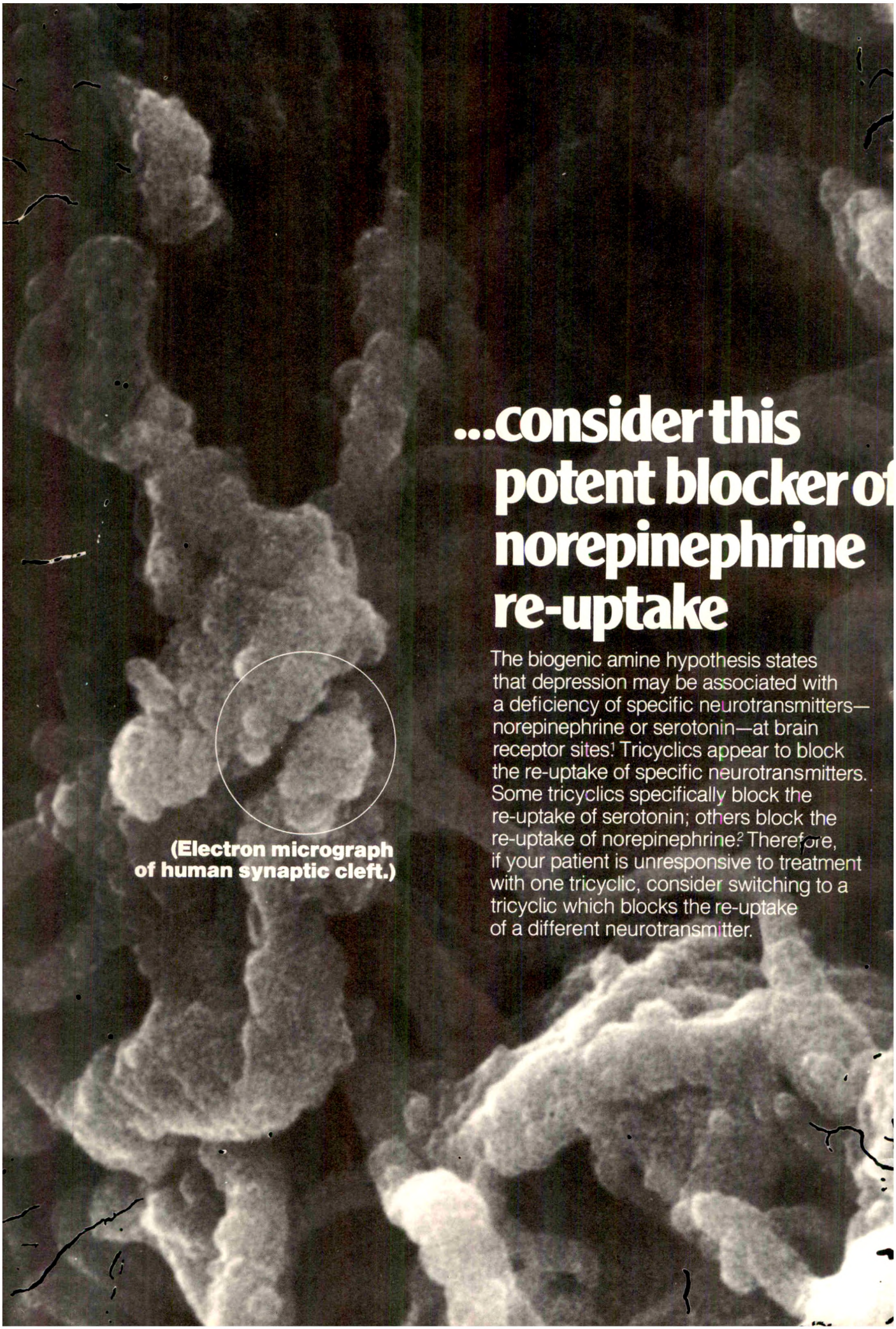
HOW SUPPLIED: MYSOLINE Tablets—No. 430—Each tablet contains 250 mg of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg of primidone (scored), in bottles of 100 and 500.
MYSOLINE Suspension—No. 3850—Each 5 ml (teaspoonful) contains 250 mg of primidone, in bottles of 8 fluidounces.

References: 1. Woodbury, D. M., and Fingl, E., in Goodman, L. S., and Gilman, A. (eds.): The Pharmacological Basis of Therapeutics, ed. 5, New York, Macmillan Publishing Co., Inc., 1975, p. 222. **2.** Rodin, E., in Conn, H. F. (ed.): Current Therapy 1977, Philadelphia, W. B. Saunders Company, 1977, p. 716. **3.** Forster, F. M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. **4.** Physicians' Desk Reference, ed. 31, Oradell, N.J., Medical Economics Co., 1977, p. 1175.

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When your patient is clinically depressed...
"...everything I saw was negative."



...consider this potent blocker of norepinephrine re-uptake

The biogenic amine hypothesis states that depression may be associated with a deficiency of specific neurotransmitters—norepinephrine or serotonin—at brain receptor sites.[1] Tricyclics appear to block the re-uptake of specific neurotransmitters. Some tricyclics specifically block the re-uptake of serotonin; others block the re-uptake of norepinephrine.[2] Therefore, if your patient is unresponsive to treatment with one tricyclic, consider switching to a tricyclic which blocks the re-uptake of a different neurotransmitter.

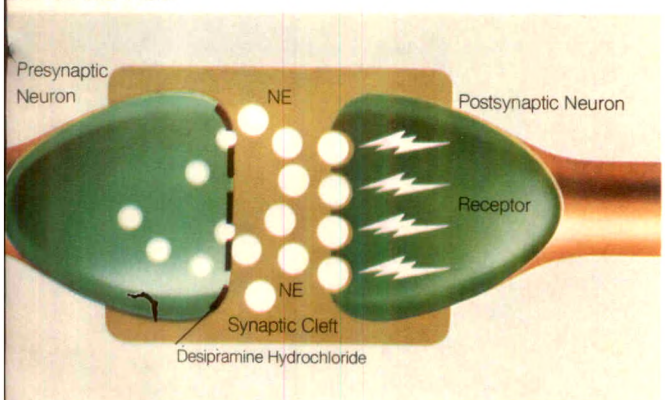
(Electron micrograph of human synaptic cleft.)

Norpramin®
(desipramine hydrochloride tablets NF)

25, 50, 75, 100, 150 mg.

Potent blocker of norepinephrine re-uptake

According to current theory[1,3] some tricyclic antidepressants may fail in certain types of depression because they are not sufficiently potent blockers of norepinephrine re-uptake. Laboratory studies have shown that desipramine is the most potent inhibitor of norepinephrine re-uptake.[4] Norpramin may work in some patients when other tricyclic antidepressants do not.



Consider it now, consider it next.

Less anticholinergic activity

(See Warnings section.)

Studies in animals[5] and in normal human subjects[6,7] have shown that desipramine has significantly less anticholinergic activity than amitriptyline or doxepin. The single bond side chain is associated with lower anticholinergic activity than these other leading antidepressants. This may mean:

- less dry mouth
- less blurred vision
- less urinary retention

Helps patients remain calm but active

Norpramin does not usually inhibit normal activity although patients should be cautioned as to driving or operating machinery if drowsiness occurs.

In addition...

Begins to improve sleep patterns within one week in some patients[8]

- Norpramin helps relieve the sleep disturbances that often accompany depression.
- As the depression is relieved, the difficulty in falling asleep, restlessness, and early morning awakening diminish.

References:

1. Maas, J.W.: Biogenic amines and depression. Biochemical and pharmacological separation of two types of depression. Arch. Gen. Psychiat. 32:1357-1361, 1975. **2.** Mendels, J., Stern, S., and Frazer, A.: Dis. Nerv. Syst. 37:3, 1976. **3.** Davis, J.M.: Central biogenic amines and theories of depression and mania. In, Phenomenology and Treatment of Depression, W.E. Fann, I. Karacan, A.D. Pokorny, and R.L. Williams, Ed., New York, Spectrum Publications, Inc., 1977, pp. 17-32. **4.** Ross, S.B. and Renyi, A.L.: Tricyclic antidepressant agents. 1. Comparison of the inhibition of the uptake of ^{3}H-noradrenaline and ^{14}C-5-hydroxytryptamine in slices and crude synaptosome preparations of the midbrain-hypothalamus region of the rat brain. Acta Pharmacol. (Kobenhavn) 36:382-394, 1975. **5.** Snyder, S.H. and Yamamura, H.I.: Antidepressants and the muscarinic acetylcholine receptor. Arch. Gen. Psychiat. 34:236-239, 1977. **6.** Blackwell, B., Stefopoulos, A., Enders, P., Kuzma, R., and Adolphe, A.: The anticholinergic activity of two tricyclic antidepressants. Amer. J. Psychiat., 135:722-724, June 1978. **7.** Peterson, G.R., Hostetler, R.M., Blackwell, B., Kuzma, R., and Adolphe, A.B.: Effects of the tricyclic antidepressants desipramine and doxepin on anticholinergic and CNS activity in non-depressed volunteers. Neuroscience Abstr. 3:447, 1977. **8.** Zung, W.W.K.: The pharmacology of disordered sleep. A laboratory approach. J.A.M.A. 211:1532-1534, 1970.

(For prescribing information see next page.)

Merrell

Norpramin®
(desipramine hydrochloride tablets NF)

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

MECHANISM OF ACTION: Available evidence suggests that many depressions have a biochemical basis in the form of a relative deficiency of neurotransmitters such as norepinephrine and serotonin. Norepinephrine deficiency may be associated with relatively low urinary 3-methoxy-4-hydroxyphenyl glycol (MHPG) levels, while serotonin deficiencies may be associated with low spinal fluid levels of 5-hydroxyindolacetic acid.

While the precise mechanism of action of the tricyclic antidepressants is unknown, a leading theory suggests that they restore normal levels of neurotransmitters by blocking the re-uptake of these substances from the synapse in the central nervous system.

Evidence indicates that the secondary amine tricyclic antidepressants, including Norpramin, may have greater activity in blocking the re-uptake of norepinephrine. Tertiary amine tricyclic antidepressants, such as amitriptyline, may have greater effect on serotonin re-uptake.

Norpramin (desipramine hydrochloride) is not a monoamine oxidase (MAO) inhibitor and does not act primarily as a central nervous system stimulant. It has been found in some studies to have a more rapid onset of action than imipramine. Earliest therapeutic effects may occasionally be seen in 2 to 5 days, but full treatment benefit usually requires 2 to 3 weeks to obtain.

INDICATIONS: Norpramin (desipramine hydrochloride) is indicated for relief of symptoms in various depressive syndromes, especially endogenous depression.

CONTRAINDICATIONS: Desipramine hydrochloride should not be given in conjunction with, or within 2 weeks of, treatment with an MAO inhibitor drug; hyperpyretic crises, severe convulsions, and death have occurred in patients taking MAO inhibitors and tricyclic antidepressants. When Norpramin (desipramine hydrochloride) is substituted for an MAO inhibitor, at least 2 weeks should elapse between treatments. Norpramin should then be started cautiously and should be increased gradually.

The drug is contraindicated in the acute recovery period following myocardial infarction. It should not be used in those who have shown prior hypersensitivity to the drug. Cross sensitivity between this and other dibenzazepines is a possibility.

WARNINGS: 1. Extreme caution should be used when this drug is given in the following situations: a. In patients with cardiovascular disease, because of the possibility of conduction defects, arrhythmias, tachycardias, strokes, and acute myocardial infarction. b. In patients with a history of urinary retention or glaucoma, because of the anticholinergic properties of the drug. c. In patients with thyroid disease or those taking thyroid medication, because of the possibility of cardiovascular toxicity, including arrhythmias. d. In patients with a history of seizure disorder, because this drug has been shown to lower the seizure threshold. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. USE IN PREGNANCY: Safe use of desipramine hydrochloride during pregnancy and lactation has not been established; therefore, if it is to be given to pregnant patients, nursing mothers, or women of childbearing potential, the possible benefits must be weighed against the possible hazards to mother and child. Animal reproductive studies have been inconclusive. 4. USE IN CHILDREN: Norpramin (desipramine hydrochloride) is not recommended for use in children since safety and effectiveness in the pediatric age group have not been established. 5. The patient should be cautioned that this drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. 6. In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

PRECAUTIONS: 1. It is important that this drug be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with this class of drug. Ordinary prudence requires that children not have access to this drug or to potent drugs of any kind; if possible this drug should be dispensed in containers with child-resistant safety closures. Storage of this drug in the home must be supervised responsibly. 2. If serious adverse effects occur, dosage should be reduced or treatment should be altered. 3. Norpramin (desipramine hydrochloride) therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates. 4. The drug may cause exacerbation of psychosis in schizophrenic patients. 5. Close supervision and careful adjustment of dosage are required when this drug is given concomitantly with anticholinergic or sympathomimetic drugs. 6. Patients should be warned that while taking this drug their response to alcoholic beverages may be exaggerated. 7. Clinical experience in the concurrent administration of ECT and antidepressant drugs is limited. Thus, if such treatment is essential, the possibility of increased risk relative to benefits should be considered. 8. The sedative effects of Norpramin and benzodiazepines (*e.g.*, chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. 9. This drug should be discontinued as soon as possible prior to elective surgery because of the possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients taking desipramine hydrochloride. 10. Both elevation and lowering of blood sugar levels have been reported. 11. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathologic neutrophil depression.

ADVERSE REACTIONS: Note: Included in the following listing are a few adverse reactions that have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when Norpramin (desipramine hydrochloride) is given.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurologic: numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus.

Anticholinergic: dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis, increased intraocular pressure; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs.

Hematologic: bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia.

Gastrointestinal: anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue.

Endocrine: gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

DOSAGE AND ADMINISTRATION: Not recommended for use in children. Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients compared to hospitalized patients, who are closely supervised. Dosage should be initiated at a low level and increased according to clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a period of time and should be at the lowest dose that will maintain remission.

Usual Adult Dose: The usual adult dose is 100 to 200 mg. per day. In more severely ill patients, dosage may be further increased gradually to 300 mg./day if necessary. Dosages above 300 mg./day are not recommended.

Dosage should be initiated at a lower level and increased according to tolerance and clinical response.

Treatment of patients requiring as much as 300 mg. should generally be initiated in hospitals, where regular visits by the physician, skilled nursing care, and frequent electrocardiograms (ECG's) are available.

The best available evidence of impending toxicity from very high doses of Norpramin is prolongation of the QRS or QT intervals on the ECG. Prolongation of the PR interval is also significant, but less closely correlated with plasma levels. Clinical symptoms of intolerance, especially drowsiness, dizziness, and postural hypotension, should also alert the physician to the need for reduction in dosage. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

Adolescent and Geriatric Dose: The usual adolescent and geriatric dose is 25 to 100 mg. daily.

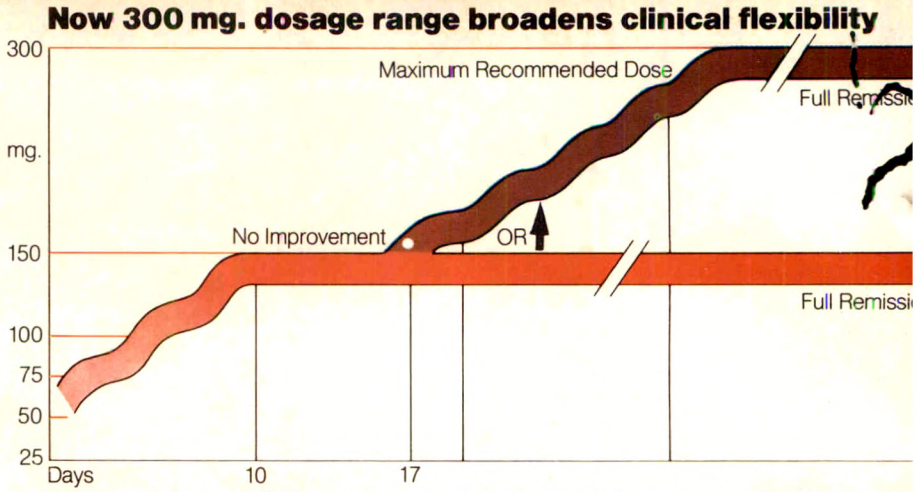
Dosage should be initiated at a lower level and increased according to tolerance and clinical response to a usual maximum of 100 mg. daily. In more severely ill patients, dosage may be further increased to 150 mg./day. Doses above 150 mg./day are not recommended in these age groups.

Initial therapy may be administered in divided doses or a single daily dose.

Maintenance therapy may be given on a once-daily schedule for patient convenience and compliance.

OVERDOSAGE: See prescribing information for a discussion of symptoms and treatment of overdose.

Product Information as of April, 1978

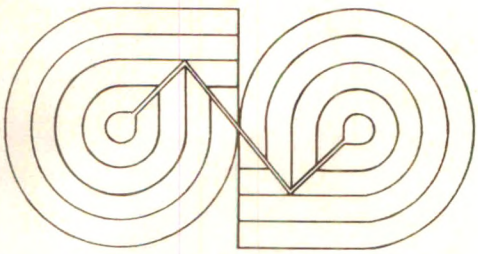


A maximum dosage of 300 mg. a day, as a single or divided dose, may now be prescribed fo Norpramin. Since inadequate dosage is thought to be one of the major causes of treatment failure, the new therapeutic range should prove clinically helpful. Plasma desipramine measurement would constitute the optimal guide to dosage monitoring.

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The Departments of Reproductive Medicine and Psychiatry; Divisions of Urology and Plastic Surgery; and the Office of Continuing Education in the Health Sciences, University of California San Diego School of Medicine will sponsor the Sixth International Gender Dysphoria Symposium at the Hotel Del Coronado, Coronado, California, February 21-25, 1979. Presentations will be made in psychological, physical and endocrinologic diagnosis and management of Gender Dysphoria. This program is designed for the professional in the medical, social and psychological disciplines that evaluate the problems of patients with a variety of trans-gender states. Internationally renowned experts in the field will present invited papers. The course is designed to promote the widest possible audience participation and discussion of each paper, and the attendance is therefore limited. The proceedings of the symposium will be published. The symposium will be accredited by the American Medical Association, and the associations of the various disciplines involved. All professionals are invited to attend.

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Infrequent akathisia and other disabling extrapyramidal effects...

The rapidly rising readmission rate among discharged psychotic patients is mainly due to noncompliance with antipsychotic drug therapy. And this, in turn, may be largely attributed to disabling extrapyramidal side effects, notably akathisia.[1]

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Extrapyramidal Effects of Selected Antipsychotic Agents*[2]

DRUG	EXTRAPYRAMIDAL EFFECTS
Chlorpromazine	Moderate
Perphenazine	High
Prochlorperazine	High
Fluphenazine	High
Acetophenazine	Moderate
Trifluoperazine	High
Chlorprothixene	Moderate
Thiothixene	Moderate
Haloperidol	High
MELLARIL® (thioridazine)	Low

*Based on antipsychotic dosage ranges

References

1. Van Putten T: The rising rehospitalization rate of psychiatric patients. Scientific Exhibit, American Psychiatric Association, 130th Annual Meeting, Toronto, Canada, May 2-6, 1977.

2. Byck R: Drugs and the treatment of psychiatric disorders, in Goodman LS, Gilman A (eds): *The Pharmacological Basis of Therapeutics*, ed. 5. New York, Macmillan Publishing Co, Inc, 1975, pp 170-171.

Mellaril® [thioridazine]

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CONCENTRATE: 100 mg per ml

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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

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Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.
Store and dispense Concentrate below 86°F; use tight, amber glass bottle. Just prior to administration, Concentrate may be diluted with distilled water, acidified tap water, or suitable juices; preparation and storage of bulk dilutions is not recommended.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.
It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Genetic Counseling for Psychiatric Patients and Their Famili

BY MING T. TSUANG, M.D., PH.D.

The author presents an overview of genetic counseling for psychiatric patients and their families, including a brief summary of contemporary research into the evidence of genetic factors, the familial risks, and the possible modes of inheritance for major psychiatric disorders. The purpose is to review and condense the available literature pertaining to psychiatric genetic counseling for the benefit of psychiatrists and counselors in their day-to-day practice and for the general "consumer." It is hoped that giving the general public access to such information will help to reduce the suffering commonly brought about by the stigmatization of mental disorders.

"Is mental illness inherited?" Doctors are frequently asked such a question by concerned patients. Can the question be answered? No, not any more than one could say whether physical illness in general is inherited. Just as there are many kinds of physical illness, some inherited and some not, so there are many kinds of mental disorder, ranging from the most debilitating psychoses to states bordering on normality. Within the wide range of mental disorders, some, such as Huntington's chorea, have a clear genetic basis. Others, such as schizophrenia, affective disorder, and even alcoholism, have genetic components but also show the influences of environment. Still others, like the neuroses, are of uncertain cause y[illegible] volve a genetic predisposition, since th[illegible] tically run in families. "What pa[illegible] would you like to know about?" may [illegible] best reply.

This paper will present an overview o[illegible] seling for psychiatric disorders, inclu[illegible] of research findings in the genetics of [illegible] orders, which doctors may find useful [illegible] to patients or their families.

IS PSYCHIATRIC GENETIC COUNSE[illegible] LEGITIMATE?

Many professionals in the health scie[illegible] felt that psychiatric disorders are not [illegible] genetic counseling because of their unk[illegible] If we are not sure what gene defects [illegible] psychiatric disorders, how can we esti[illegible] any certainty?

This paper summarizes data from tw[illegible] adoption studies that give evidence of [illegible] for many of the major psychiatric illnes[illegible] knowing that there is a genetic basis for [illegible] substitute for knowing how the gene[illegible] works. However, some physical disea[illegible] logies that are no more certain than the [illegible] chiatric disorders are commonly the s[illegible] netic counseling.

Diabetes mellitus is a useful example [illegible] many parallels that can be drawn betwe[illegible] ophrenia, genetically speaking (1, 2). [illegible] and genetic factors appear to be at w[illegible] ease, but definite causes are unknown. [illegible] may in fact have several genetically [illegible] Neither disorder is likely to reveal itse[illegible] ly, each disorder has early onset form[illegible] be genetically distinct from its late on[illegible]

Such problems make counseling m[illegible] they do not preclude it. Doctors have [illegible]

This paper was written at the invitation of the Editor.

Dr. Tsuang is Professor of Psychiatry, Department of Psychiatry, University of Iowa College of Medicine, and Chief Staff Psychiatrist, East Ward Inpatient Service, University of Iowa Psychiatric Hospital, 500 Newton Rd., Iowa City, Iowa 52242.

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practical means for estimating the risk of recurrence for ambiguously inherited disorders. Chiefly, they have access to pools of data from large epidemiologic studies that allow them to make reasonably accurate empirical risk estimates. Furthermore, advancements in genetics and statistical methodology have provided theoretical mathematical models into which many family data can be fit. Finally, doctors can serve patients and their families beyond offering risk estimates: they can dispel mistaken notions about psychiatric disorders, calm needless anxiety, and help those at risk to draw up a rational plan of action based on the best available information.

BENEFITS OF PSYCHIATRIC GENETIC COUNSELING

Misunderstandings of genetic disorders, mental illness, and genetic counseling can easily create certain anxieties that informed and humane psychiatric genetic counseling can do much to alleviate. In particular, counseling can correct the following common but mistaken beliefs:

1. If a disorder is genetic it inevitably occurs in those who possess the harmful genes. In psychiatric disorders especially, a 100% genetic influence is by far the exception rather than the rule. Genetic factors are variable, and environmental variables may play a role even when genetic factors are present. Only Huntington's chorea seems to obey strictly genetic rules. For other psychiatric disorders, the relationship between environmental and genetic influences is different in each case, and both types of factors have different impacts on different individuals. Some people may be carriers of damaging genes and yet experience no symptoms of the disorder themselves.

2. If a disorder is genetic it is untreatable. Even though some psychiatric disorders, such as dementia, often follow a continuously deteriorating course, other disorders frequently respond to treatment. For example, phenylketonuria, a rare genetic disorder that appears during the first year of life, can often be controlled by early supervision of the infant's diet. The course of manic-depressive disease can often be influenced positively by treatment with lithium carbonate (3, 4).

3. Genetic counseling is associated with eugenics and genocide. This belief, perhaps based on memories of the horrors of Nazi Germany, is unjustifiable. Contemporary genetic counseling is as removed from such atrocities in method and purpose as the peacetime nuclear reactor is from the atom bomb. Contemporary genetic counseling is noncoercive, stressing the autonomy of the individual in decision-making. Genetic counselors seek to distribute genetic information to those who request it and to aid counselees in forming their own resolutions based on the information they receive. Doctors will sometimes find it necessary to raise the level of anxiety in counselees who take the implications of mental illness too casually. At other times, the doctor may be able to relieve anxiety in the overly pessimistic patient or relative. Both functions serve the broadest purpose of genetic counseling: to reduce human suffering due to genetically transmitted disorders.

By correcting mistaken notions about genetic disorders and genetic counseling, the doctor may help counselees attain realistic estimates of the risks they confront. He or she may convince counselees to disregard the pseudo-scientific judgments of well-meaning relatives or friends and to reserve judgment until the diagnosis of the disorder is firmly established and the available facts are known. Some disorders may then call for careful family planning, while others may not raise such serious considerations.

The need for counseling for all genetic disorders is great because recent social changes and advancements in medical research have greatly increased the options for prospective parents who wish to circumvent the recurrence of serious genetic disorders. The public now has access to effective means of prenatal diagnosis, such as amniocentesis, which can detect chromosomal abnormalities or the sex of a child before birth. Effective means of contraception, sterilization, and legal abortion are available for those who wish to prevent high-risk births, and artificial insemination or adoption permits parents to bypass serious risk factors in building a family.

These developments have been popular subjects in the public media in recent years. Consequently, the public shows a new sophistication in scientific knowledge. Some may still be asking, "Is mental illness inherited?" while others may be inquiring whether they have a chromosomal aberration, and if so, what its origin is, whether it is transmissible, how it alters biochemical processes, whether it could be the result of drugs, or whether parental age is a factor in its transmission.

However, new knowledge can and often does create new confusions. Social and scientific developments offer power to the citizen to grant or prevent the creation of life, but new power brings concomitant responsibilities. When information is partially communicated or wrongly interpreted, it can bring about irresponsibility, prejudice, new forms of suffering, and greater legal, moral, and emotional burdens than ever before.

Here the contribution of the genetic counselor becomes plain. He becomes one of the arbitrators in the exercise of power. He has access to medical facts, probability models, gene frequency data, and empirical risk data. He has some idea of the course, outcome, and prognosis of a wide range of diseases. He has access to other medical, psychiatric, and legal specialists who can extend his expertise. He can calm, inform, encourage, advise. Equally important, he can withhold information where scientific uncertainties exist or where too much information would create confusion.

The psychiatric genetic counselor, as arbitrator, is a

catalyst in the prevention of suffering that results from severe genetically influenced mental disorders. He can interpret often puzzling and ambiguous developments in psychiatric research to genetically vulnerable families to help them make decisions about marriage or parenthood. At the same time he can inform those who need to know that some predictions can be made with reasonable accuracy on the basis of genetic evidence and that "not all mental illness is inherited."

WHO SEEKS PSYCHIATRIC GENETIC COUNSELING?

Three groups of people most typically seek professional advice concerning the genetics of psychiatric disorders: 1) psychiatric patients, 2) families of psychiatric patients, and 3) prospective spouses.

Patients usually seek counsel in the early stages of illness when they first become aware that the illness seriously affects their material prosperity, social adjustment, or family life. At that time they may be seriously concerned about the implications of the disorder for their descendants. In certain disorders that cause impairment of self-awareness, e.g., chronic deteriorating schizophrenia or late-stage Huntington's chorea, the patient is not likely to volunteer for genetic counseling. When self-awareness is retained to some degree—as in affective disorder, alcoholism, or neurosis—the patient is more likely to be counseled.

Families will most often approach a doctor for counseling when many incidences of illness have occurred among blood relatives or when there have been a few relatives with particularly severe or chronic mental illnesses. In cases of affective disorder, patients and their families are often counseled together. By the time a family seeks genetic counseling, it may already have experienced a remarkable degree of psychosocial and financial distress due to the presence of illness.

A prospective spouse will often seek counseling when psychiatric illness has become apparent in one or many of the partner's relatives. The prospective spouse may then want counsel concerning the genetic prospects for both the future partner and their potential offspring. The advice of the genetic counselor at this stage may indirectly serve an important, even crucial, function as marriage counsel.

THE STAGES OF COUNSELING

Genetic counseling may be defined as the process of communicating information and advice to patients and/or their relatives to help them deal with problems connected with the occurrence or possibility of recurrence of a genetically caused or influenced disorder. Various schematizations of this process can be found in the literature of genetic counseling (5–8), but genetic counseling usually includes seven basic stages, described below.

Diagnosis

The first step in the process must be ... agnosis of the disorder. This implies ob... cross-sectional picture based on the tota... sentation and a longitudinal view based ... clinical course, in addition to a comple... tory. Counseling under a mistaken d... more dangerous than counseling without ... the hereditary mechanism at work.

The standard diagnostic manual curre... the United States is the 2nd edition of th... *and Statistical Manual of Mental Disord...* published by the American Psychiatric A... 1968 (9). Because certain ambiguities of ... peared in *DSM-II*, APA is now revising ... and will soon publish *DSM-III*. For resea... more strictly defined criteria are available ... though these do not cover all diagnostic ... mental disorders. The physician must ... available means to make sure a disorder ... diagnosed before undertaking counsel...

A number of factors may complicate t... obtaining an accurate diagnosis, especial... ric disorders. Diagnostic criteria may dif... to clinic or from region to region. Furthe... cal expertise required to obtain an accu... may not be located within easy reach of t... Certain disorders that look alike may actu... ogeneous. Sometimes, purely enviro... can cause illnesses that closely resemble ... influenced disorders. Other disorders ... cally related but have no apparent rese... cause of variable ages of onset, idios... course, "variable expressivity," etc. In ... difficulties, the importance of obtaining a ... agnosis before proceeding with counseli... overemphasized.

Family History

The second stage of counseling involves ... a complete and accurate pedigree. If the ... still uncertain at this stage, the family his... veal the most probable one. It may also ... mode of inheritance of the disease. The fa... includes such information as parental age, ... ground, occurrence of abortions, stillbirths, ... deaths, as well as the ages, sexes, and hea... siblings and children. Primary attention is g... occurrence of the disorder in first-degree ... the family history also records the hea... blood relatives, such as grandparents, un... and cousins.

Risk of Recurrence

After the pedigree has been carefully ... physician must estimate, if possible, the ris... rence of the disorder. Risk refers to the ... that a given trait or disorder will occur in ... of an affected person. Murphy and Chase ...

guished three categories of information that can be used to arrive at an estimate of risk.

1. "Empirical information" consists of estimates based on data collected in controlled research. Such studies may provide the counselor with information about the rate of certain gene mutations in the population, the average age of onset of a disorder, or the estimated risk of recurrence in first-degree relatives (parents, siblings, children).

2. "Modular information" is based on a rational understanding of the mode of inheritance of a certain trait. Thus, while empirical information allows one to reason backward from known estimates to possible genetic factors, modular information permits more precise estimates based on known or reasonably certain genetic factors.

3. "Particular information" is all of the data contained in the family history of the person or persons whose genes are in question. This category is further divided into "posterior information" (i.e., information about all persons whose genetic makeup could be influenced by the person in question, such as children, grandchildren, etc.) and "anterior information" (i.e., all information on members of the pedigree who could not possibly have been influenced by the patient's genotype, such as parents, uncles, nieces, stepsons, etc.). Using such information, the doctor may be able to calculate "exact" risk, when the mode of inheritance and other crucial factors are known, or "empirical" risk, when the estimates must be based on average frequencies of a given disease among various degrees of relatives of people known to have the disease.

Evaluation of the Counselee

Before the doctor can effectively communicate the risk estimates to counselees and help them weigh the risk against the potential burden of the disorder, he or she must understand their intentions, intellectual capabilities, and states of mind. Do they seek information, advice, or both? Do they want advice for their own offspring or someone else's? How sophisticated is their acquaintance with genetic facts and principles? What is their general level of intellect? Can they make rational decisions? What is their present emotional state? Finally, how do they perceive the risk/burden ratio in their situation? Humane counseling must accommodate the specialized knowledge of the physician to the needs, intentions, personalities, and mental states of the counselees.

The Risk/Burden Ratio

The doctor must next help the counselees to weigh the estimated risk of recurrence against the burden of the disorder, even though they may tend to be concerned only with burden. Although risk can often be calculated with reasonable accuracy, there is no precise way to estimate burden. Burden is the expected cost to a family of a recurrence of disorder. It must be reckoned not only in terms of financial loss but also in terms of human effort, pain, loss of life, embarrassment, childlessness, grief, and many other intangibles (8).

Obviously, burden will be considered relatively low when the disorder is so severe that it is rapidly fatal or when it is long-lasting but does little to impair normal functioning over the course of a lifetime. The burden will be greatest when an individual suffers from severe impairment requiring elaborate supervision over a long period of time. Frequently, parents will take high risks if they perceive that the burden will be low but will "play safe" when the perceived burden is very great.

Forming a Plan of Action

Once the risk/burden ratio is understood, the doctor's responsibility is to assist counselees in forming a reasonable plan of action for deciding among their options. If the decision involves family planning, the decision whether or not to have children cannot and should not be made by the doctor. The decision properly belongs to parents themselves. However, the doctor's role must go beyond mere stating of facts. He is better able than the parents to predict consequences, but he should do so with care. He may also explain his own preferences for certain courses of action. Finally, however, he must commit all responsibility for the decision to the parents, without pressuring or rushing them.

This stage of counseling requires that the physician assess the counselees' understanding of what they have been told. He may ask them to repeat to him what they understand, or he may summarize in writing the main points that arose during the counseling session.

Follow-Up

The final stage of genetic counseling must be follow-up. Follow-up within a reasonable period of time permits continued assessment of the counselees' understanding. It may also provide new data leading to a more exact prognosis, e.g., a subsequent birth or other follow-up information may specifically indicate a certain mode of inheritance. Such information may permit a revision of the risk estimates for greater accuracy.

METHODOLOGY IN PSYCHIATRIC GENETIC STUDIES

The most effective means for studying the presence of genetic factors in psychiatric disorders, in increasing order of precision, are family, twin, and adoption studies. These are the most widely published studies; however, recent advances in medical and genetic technology have also led to research on linkage or association between some disorders and known genetic markers (12–15). The finding of association between certain genes and certain disorders could provide practical means to determine the presence of genetic predispositions for those disorders. The finding of linked genes could implicate a major gene to the exclusion of

a polygenic mode of inheritance. However, since the evidence is inconclusive at present, I will not go into further detail concerning linkage and association.

Family and Twin Studies

Family studies are useful in providing reasonable risk estimates for various disorders among different classes of relatives of affected patients. They may suggest the presence of genetic factors by showing the concentration of various illnesses within families, but genetic factors cannot be distinguished from environmental ones on the basis of family studies.

Twin studies involve a comparison of two types of twin pairs: monozygotic (MZ) and dizygotic (DZ). Monozygotic twins can be expected to be 100% genetically identical, whereas dizygotic twins can be expected to be about 50% genetically identical, like other siblings. Since MZ twins have identical genes, any differences between them must be due to nongenetic factors. On the other hand, it cannot be assumed that all similarity between twin partners (their "concordance" for a given condition) is due only to genetic factors. Their similarities may also result from a largely identical environment.

Twin studies attempt to test whether genetic factors are present for certain conditions by comparing the MZ twin concordance rate for a given condition with the DZ concordance rate for the same condition. Environmental effects are assumed to be approximately equal for the two types of twins. Thus, in a representative sample of twins, if the MZ concordance rate is significantly higher than the DZ concordance rate, there is strong evidence that the condition in question has been influenced by genetic factors, although the twin study cannot completely disentangle genetic from environmental influences.

Adoption Studies

Adoption studies achieve the best results in distinguishing genetic from environmental influences. They study persons who were separated from their biological parents at birth and raised with biologically unrelated adoptive parents. By controlling for the environmental factor in this way, one can separate the interacting influences of genes and environment in the causation of given disorders. If the adoptee is significantly more like his biological relatives than his adoptive relatives, a genetic factor is assumed to be present. Adoption studies have four basic designs: 1) the adoptee study design, 2) the adoptees' parents study design, 3) the adoptees' family study design, and 4) the "crossfostering" design (16).

The adoptee study investigates the rate of disorder in the adopted-away children of ill parents. The adoptees' parents and family studies investigate the rate of disorder in the parents and relatives, respectively, of ill adoptees. The crossfostering study investigates the rate of disorder in children born to normal parents but raised in the homes of disordered adoptive parents. Occasionally adoption and twin studies are combined in an investigation of MZ [illegible] raised apart.

GENETIC FACTORS IN SPECIFIC PSY[illegible] DISORDERS

This section offers a brief outline of e[illegible] presence of genetic factors in several m[illegible] ric disorders. Because of space limitatio[illegible] attempted to mention all pertinent rese[illegible] scribe any one study in full detail, althoug[illegible] extensive literature has been weighed in [illegible] sults germane to this discussion.

Schizophrenia

Twin study data from investigations [illegible] ferent countries have shown that MZ tw[illegible] significantly higher concordance rates fo[illegible] nia than DZ twin pairs (MZ pooled rate [illegible] pooled rate=13.7%) (17). These studies [illegible] that as the genetic relationship between w[illegible] more similar—from opposite-sex DZ to [illegible] to MZ—the intrapair concordance rises [illegible] (18). The fact that even MZ concordance [illegible] than 50% suggests that environmental [illegible] netic factors play a significant role in the [illegible] of schizophrenia.

Adoptee studies have shown signifi[illegible] rates not only of schizophrenia but also o[illegible] psychiatric disorders (e.g., personality d[illegible] social personality, and mental deficiency) [illegible] away children of schizophrenics than [illegible] adoptees (19, 20).

Several adoptees' family studies have fou[illegible] cantly higher rates of schizophrenia among bi[illegible] cal relatives of schizophrenic adoptees than [illegible] ther their adoptive families or the biological [illegible] tive families of controls (21, 22). These studies [illegible] twin study evidence in showing the presence o[illegible] factors in schizophrenia. A crossfostering [illegible] adoptees raised by schizophrenic parents fou[illegible] 4.8% rate of schizophrenia compared with [illegible] cantly higher rate (19.7%) among adopted-a[illegible] spring of schizophrenic biological parents (2[illegible] finding suggests that schizophrenia in a par[illegible] not promote the development of schizophren[illegible] child if the child is not genetically predisp[illegible] schizophrenia.

Affective Disorder

A pool of seven major twin studies (see refere[illegible] for a summary) shows an overall MZ twin concor[illegible] rate of 76% for affective disorder, compared w[illegible] overall rate of 19% for DZ twins. Studies of MZ [illegible] reared apart (25) show a 67% concordance rate, [illegible] is statistically similar to the overall concordan[illegible] MZ twins. Twin studies of affective disorders [illegible] been summarized by Gershon and associates (26

significant difference between MZ and DZ concordance rates indicates a strong genetic factor in affective disorder.

Mendlewicz and Rainer (27) recently conducted a study of adoptees' parents in Belgium that strongly supports twin studies in showing the importance of genetic factors in affective disorders. When the biological and adoptive parents of 29 bipolar adoptees were compared, the former showed significantly higher rates for all psychopathology (40% versus 16%), particularly affective disorders. Further, the rate of affective disorders in these biological parents (31%) was not statistically different from the rate of affective disorders among parents of 37 bipolar nonadoptees (26%). This study, in which a rigorous research design was used to control for environmental factors, offers strong support for previous studies implicating genetic factors in bipolar illness. A very recent adoptee study (28) also reported a significantly increased incidence of depression in adoptees who had affectively disordered biological parents.

Presenile Dementias

From a counseling perspective the most important of the dementias are the presenile dementias—Huntington's chorea, Alzheimer's disease, and Pick's disease. Because these are slowly deteriorative conditions with usual onset in middle age, they impose a greater overall burden than forms with later onset, such as senile dementia. Usually, the genes are transmitted to offspring before the disease manifests itself in the parents. Family studies of Huntington's chorea show a clear pattern of autosomal dominant inheritance with age-dependent penetrance. Some kindreds in which Alzheimer's and Pick's diseases appear also show autosomal dominant inheritance, but most cases of these illnesses are of uncertain heredity (29).

Alcoholism

Although environmental factors are very important in alcoholism, twin and adoption studies have shown that genetic factors also seem to be present (30, 31). A well-designed twin study by Kaij (32) examined concordance according to the severity of alcohol abuse. In the two categories of greatest severity, DZ twins showed a high concordance rate (66.7%), but MZ concordance rates were significantly higher (84.5%) (32). In the same study, concordance for alcohol abuse increased with increasing severity of abuse in MZ but not in DZ pairs. These findings give evidence of genetic factors in alcoholism. However, major adoption studies of alcoholism have supported genetic influence only in the case of male adoptees. A Danish adoptee study of alcoholism (33, 34) found a significantly higher rate of alcoholism among the adopted-away sons of alcoholic biological fathers than among control adoptees. Adopted-away daughters of alcoholic biological fathers show no higher rates of alcoholic abuse than control adoptees (35).

Sociopathy

Sociopathy by definition involves environmental factors, but twin and adoptive studies suggest the presence of genetic factors as well (36, 37). Twin studies of psychopathy (38, 39) show MZ concordance rates ranging from 36% to 100% and DZ concordance rates of up to 54%. These high rates point strongly toward the existence of both genetic and environmental factors in sociopathy.

An adoptee study found a rate of 13% for antisocial personality among the adopted-away offspring of female legal offenders compared with a base rate for antisocial personality among all adoptees of about 2.3% (40). A recent adoptee study of antisocial behavior by Cadoret (37) found a significant correlation between the diagnosis of antisocial personality or behavior in an adoptee and the same diagnosis in a biological parent. Both studies used the adoptee design to control for environmental influences and suggest that genetic factors are present in antisocial personality.

Neuroses

Certain types of personalities seem more prone than others to anxiety in response to environmental stress. It is supposed, therefore, that the many, many genes which regulate the processes of personality development and physiological response, although their individual effect is small, may have a combined influence in creating a predisposition to neurosis in some people. Evidence for such an influence has been provided by twin studies, which have generally found much higher concordance rates for neurosis among MZ than DZ twin pairs. A major study of neurosis and personality disorder in 146 twin pairs by Slater and Shields (41) found that in 47% of the MZ pairs both members received a psychiatric diagnosis compared with only 24% in DZ pairs. The same diagnosis was given to both members in 29% of the MZ pairs but only 4% of the DZ pairs. The ratios between MZ and DZ concordance rates in the study suggested that genetic influence was specific to anxiety neurosis and personality disorder, in marked contrast to reactive depressions and other forms of neurosis (41, 42).

RISK ESTIMATES FOR SPECIFIC PSYCHIATRIC DISORDERS

Except in the case of Huntington's chorea, where the genetic mechanism is known, doctors must rely on empirical data to calculate risk estimates for various psychiatric disorders. The doctor must distinguish between exact estimates, based on known genetic mechanisms, and empirical estimates, based on observed frequencies of disorder, using the latter only when they would clearly be of benefit to patients or their relatives.

Empirical risk estimates are established through family studies, which investigate the frequency of dis-

TABLE 1
Morbidity Risk of Schizophrenia*

Relatives of Schizophrenics	Risk (%)
First-degree	
Parents	4.4
Siblings	8.5
Neither parent schizophrenic	8.2
One parent schizophrenic	13.8
Children	12.3
Both parents schizophrenic	36.6
Second-degree	
Uncles and aunts	2.0
Nephews and nieces	2.2
Grandchildren	2.8
Half-siblings	3.2
Third-degree	
First cousins	2.9
General population**	0.86

Based on Slater and Cowie (44); data mainly derived from pooled data of Zerbin-Rüdin (43), with only cases of definite schizophrenia counted.
Compiled from 19 investigations in six countries (43).

orders among various classes of relatives. When risk estimates among relatives of affected patients are higher than the general population risk for a given disorder and when risk estimates are higher among near relatives than among distant relatives, risk figures offer suggestive but not conclusive evidence for genetic factors in a given disorder.

Empirical estimates must be viewed with caution, since many factors may call their accuracy or usefulness into question. Observed frequencies may be based on epidemiologic studies that contain sampling biases. Various studies offer widely differing estimates that may reflect cultural differences. Finally, the empirical risk estimate may be irrelevant or even misleading. It would be a disservice, for instance, to quote an empirical risk figure of 15% to a patient whose family history for a given disorder shows dominant inheritance with complete penetrance that would set the exact risk for siblings and offspring at 50%, as could be the case in Alzheimer's or Pick's disease.

The following is a brief summary of available risk data for specific psychiatric disorders.

Schizophrenia

Risk figures pooled from numerous studies (43, 44) are presented in table 1. The general population risk, compiled from 19 investigations in 6 countries, is 0.86%. The risk rates among first-degree relatives of schizophrenics are roughly 10 times the general population risk and nearly 3 times the risk for more distant relatives. Since most of these data are drawn from classical European studies, they reflect empirical risk estimates for process schizophrenia, the usually severe, deteriorating form of the disorder. If milder syndromes such as borderline, acute, latent, or schizo-affective schizophrenia were included in the criteria; as is more commonly done by investigators in the United States, the total number of schizophrenics diagnosed would be much greater, and risk estimates would be different from those in table 1. Unfortu[illegible] risk estimates for relatives of borderl[illegible] ics have not yet been obtained becaus[illegible] the current definition of borderline sc[illegible]

Affective Disorder

The age-adjusted risk for manic-d[illegible] sis in the general population is 1.[illegible] 2.5% for females, as estimated in a[illegible] lation study (45). Other studies hav[illegible] rates among first-degree relatives o[illegible] fective disorder of approximately 10.5[illegible] (46). The higher risk figures reflect [illegible] containing only cases of bipolar a[illegible] Winokur and Clayton (47) have estima[illegible] for siblings of an affected person [illegible] when one parent is affected, and wh[illegible] fected the risk rises to 43%.

Presenile Dementia

Since Huntington's chorea is trans[illegible] autosomal dominant gene with age-dep[illegible] trance, the unmodified risk to siblings [illegible] affected patients is 50%. Beginning fr[illegible] counselor can estimate the probability [illegible] will develop the illness at a given age [illegible] onset can be considered normally dist[illegible] the person passes through the age of ris[illegible] veloping the illness, the risk estimates [illegible] ly lowered on the basis of reasonably a[illegible] onset data.

It is estimated that the general po[illegible] Alzheimer's and Pick's diseases toget[illegible] mately 0.1% (48). For parents of affec[illegible] risk is 15%; for all siblings, the rate is 5% [illegible] least one parent is affected, 2.5% whe[illegible] is affected). For those families in which [illegible] dominant pattern of inheritance appea[illegible] quite clear: 50% for the siblings and off[illegible] fected cases and 25% for their second-deg[illegible] (nieces, nephews, grandchildren).

Alcoholism

A survey of studies in five different cou[illegible] ed risk rates for alcoholism in the general [illegible] 3%–5% for males and 0.1%–1% for fem[illegible] higher rate for males is consistent in studi[illegible] ism and is seen again in the risk estimates [illegible] of alcoholics. Data from six studies of chr[illegible] ics show risk figures of 0.4%–8.2% for mot[illegible] holics and 2.8%–10.3% for sisters. The ris[illegible] relatives are roughly 5 times higher than [illegible] males—11.4%–32.6% for fathers of alco[illegible] 12.2%–27.7% for brothers (1). A study [illegible] and associates (49), which used personal [illegible] with family members to ascertain familial [illegible] coholism, found even higher rates amon[illegible] lings—46% for brothers of male alcoholics [illegible] sisters of male alcoholics. The brothers of fe[illegible]

holics also had a high risk rate of 50%, while the rate for sisters of female alcoholics was only 8%.

Neurosis

A few family studies of anxiety neuroses have shown that the risk to parents and siblings of an affected person is approximately 15% (44).

HOW ARE SPECIFIC PSYCHIATRIC DISORDERS INHERITED?

Doctors would benefit greatly from having clear modular information that would permit them to replace empirical risk figures with more exact risk estimates for specifically diagnosed disorders. Unfortunately, the modes of inheritance for most major psychiatric disorders are not yet clearly understood. In this state of uncertainty, researchers have interpreted the available data to formulate theories about the possible genetic modes of transmission for many disorders. The complex arguments for and against such theories are beyond the scope of this discussion. Instead, I will present a brief outline of genetic models that have been proposed for certain major psychiatric disorders. Definitions and more complete discussions of basic genetic principles can be found in numerous textbooks on human genetics and genetic counseling (1, 5, 8, 50).

Schizophrenia

Theoretical models for the inheritance of schizophrenia may be grouped in three classes (51–53): monogenic, polygenic, and genetic heterogeneity.

Monogenic models in general postulate a single specific schizophrenic gene that may cause the disease to be manifested either in homozygotes only or in both homozygotes and heterozygotes (54–61). A recessive gene model is not widely advocated. Most monogenic models posit a dominant gene with a modified effect. The modification may result from other genes at other loci, from reduced penetrance (penetrance rates have been estimated at 6%–13%), or from intermediate effects, where schizophrenia is manifested in 100% of homozygotes but only in about 25% of heterozygotes. Polygenic theories include both "continuous phenotypic variation" and "quasi-continuous variation" models (51, 62–66). Both single-dominant-gene theories and polygenic theories fit the available data well enough that for practical purposes the doctor may adopt the one that best suits his or her purposes in counseling (44).

Genetic heterogeneity theories propose that schizophrenia is not a single disease entity but a variety of diseases that probably have different etiologies and modes of transmission (67). Schizophrenia may first be divided into organic (symptomatic) and idiopathic (true) categories. The former involves a clear precipitating factor such as brain damage or drug abuse, and the latter involves no clearly precipitating factors and shows a different pattern of clinical symptoms and family history. Idiopathic schizophrenia may be further divided into typical and atypical (68–70). Within typical schizophrenia, clinical and family differences may point to a distinction between paranoid and nonparanoid types (71–73). Within nonparanoid schizophrenia, a distinction between simple, catatonic, and hebephrenic subtypes may be possible (74). Within atypical schizophrenia, it may be possible to distinguish an affective type, a schizophrenic type, and an undifferentiated type (75). Some cases of schizophrenia, it has been postulated, may be due to rare genes that are specific for the individual subtypes of schizophrenia (68).

Affective Disorder

The exact mode of transmission for affective disorder is not yet known, but several genetic theories have been proposed, chiefly genetic heterogeneity, X-linked dominant, autosomal dominant, and polygenic models.

Numerous studies have suggested that bipolar affective disorder is genetically distinct from unipolar depression (47, 76, 77). Other studies have offered additional evidence for a bipolar-unipolar distinction by showing that bipolar patients as a group respond better to treatment with lithium carbonate than unipolar patients (3, 4). Some studies suggest that unipolar depression itself may be comprised of two genetically distinct types: 1) depressive spectrum disease, which occurs mostly in females under 40 years, and 2) pure depressive disease, which occurs mostly in males over age 40 (78). This hypothesis is not yet sufficiently tested to warrant general acceptance.

Many studies (12, 14, 79), including the recent adoption study by Mendlewicz and Rainer (27), have found evidence for an X-linked mode of transmission for bipolar affective disorders. The evidence for linkage is usually an absence of ill father-ill son pairs. Additional support for the hypothesis has come from studies linking bipolar affective disorder with known genetic markers such as color-blindness and the X_g^a blood group. However, numerous studies have contradicted an X-linked hypothesis by finding ill father-ill son pairs (13, 76, 80–86). Information on X-linkage is not very useful for counselees, since it is not yet possible to determine clinically which cases of bipolar affective disorder are sex-linked. Until the question of X-linkage is resolved, doctors should not counsel as though bipolar affective disorder were a sex-linked disorder, since doing so might result in a serious counseling error.

Other genetic theories for the transmission of affective disorder have invoked either a single autosomal dominant gene of approximately 30% penetrance (77, 81, 82) or a polygenic model with quasi-continuous variation (57, 87–89). Certain statistical techniques such as threshold model analysis (90, 91) and segregation analysis (92) have recently been devised for the purpose of determining whether single major genes or groups of genes are responsible for genetic transmis-

sion. However, conclusive results have not yet been obtained using such models.

Presenile Dementia

The genetic mechanism in Huntington's chorea has already been discussed. Autosomal dominant kindreds also appear in a small minority of cases of Pick's and Alzheimer's diseases (29, 93). In families where an autosomal dominant gene is not implicated for Pick's and Alzheimer's disease, the inheritance appears to be polygenic, but the exact mode is not certain (48).

Alcoholism

The predominance of males in all samples of alcoholics leads naturally to the suspicion of X-linked recessive transmission for alcoholism. If alcoholism were X-linked, it might be expected that the sons of daughters of alcoholics would show a higher incidence of alcoholism than the sons of the sons of alcoholics. However, a study specifically designed to test this hypothesis found no evidence to support it (94). In the study, the risk of alcoholism for all grandchildren of alcoholics was 43%. Such findings do not fit recessive gene models but could be interpreted as fitting a dominant gene model.

Sociopathy

All major adoption studies of sociopathy point to the influence of both environmental and genetic factors in the transmission of antisocial personality, criminality, and psychopathy (95–97). Data from a St. Louis family study of sociopathy fit a multifactorial model of disease transmission well enough to suggest that the model may be acceptable as a description of the mode of inheritance in sociopathy (98). However, the relative importance of genetic and environmental factors in sociopathy is not yet clearly understood. The same researchers have found evidence in family study data to suggest an overlap between sociopathy and Briquet's syndrome (hysteria), indicating that the two may be manifestations of a single process rather than independent disorders (99). Chromosomal abnormalities have been implicated in some studies of criminality. Males with an extra X chromosome (Klinefelter's syndrome) or an extra Y chromosome seem to be at almost 20 times the normal risk for incarceration, although the chromosomal abnormality itself cannot at present be proven responsible for the increased risk (100).

CONCLUSIONS

I have discussed the psychiatric disorders for which genetic counseling is most commonly sought, the evidence for genetic factors in each, risk rates based on empirical data, and possible modes of inheritance. Many other psychiatric disorders are known, but an account of all of them lies outside the scope of this paper. For further information, the reader is referred to standard textbooks on human genet[illegible] counseling (5, 44, 50). Many forms of [illegible] tion show autosomal dominant, autosom[illegible] or X-linked patterns of transmission. Ty[illegible] other psychiatric disorders that may fo[illegible] models, have been usefully catalogu[illegible] (101).

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The Incidence of Psychosurgery in the United States, 1971–1973

BY JOHN DONNELLY, M.D.

As part of the work of the APA Task Force on Psychosurgery, the author surveyed 1,901 neurosurgeons on how many psychosurgical procedures they performed in 1971, 1972, and 1973. A total of 1,481 questionnaires were returned; 195 neurosurgeons indicated they had performed a total of 476 procedures for intractable pain and 1,039 procedures for psychiatric conditions. Excluding the procedures reported by 4 neurosurgeons who performed more than 20 psychosurgical operations in any one of the three years, the number of psychosurgical operations is 575 for the three years, which the author suggests should allay fears about the abuse of psychosurgery.

IN THE POLITICAL TURMOIL during the decade of the troubles in the cities and of the Viet Nam war, the then somewhat obscure and declining use of neurosurgical treatment of psychiatric disorders was elevated to a position, one might say, of social prominence. Activist psychiatrists and others made repeated and highly publicized accusations that perhaps thousands of individuals were being subjected to such operations needlessly and for purely experimental purposes, as well as for political, social control, racist, and other reasons. Psychosurgery became a torch of activist psychiatrists in the United States.

In the emotional ferment of those times, the scientific value of objective evaluation was typically absent in the hierarchy of priorities of many psychiatrists, whether activist or not. It became fashionable, indeed *de rigueur*, for even nonactivists to issue statements disclaiming any support for its use—and incidentally implying opposition to it.

Within the American Psychiatric Association itself, the issue of psychosurgery became for activists a *cause célèbre*. In 1973 the Task Force on Psychosurgery was established under the aegis of the Council on Emerging Issues to study all aspects of the subject and to submit its findings and recommendations regarding the use of psychosurgery. In October 1976 the task force report was submitted to the APA Board of Trustees, who decided to refer the report back to the Council on Emerging Issues—without recommendation.

SURVEY OF PSYCHOSURGERY

As part of the work of the Task Force on Psychosurgery, the author undertook a survey of the use of neurological surgery for the treatment of psychiatric conditions by neurosurgeons in the United States. A search of the literature revealed only one recent study of the extent to which psychosurgery was performed, but this drew only a 31.5% response from the neurosurgeons surveyed (1).

In constructing the survey questionnaire, a number of difficult decisions were necessary. The first was how to keep the questionnaire as simple as possible and demand relatively little work from the recipients in order to obtain as great and as accurate a response as possible.

A significant question also arose immediately with respect to the inclusion of intractable pain as a psychiatric condition; surgery for this condition is not regarded as psychosurgery by neurosurgeons, especially when the pain is caused by conditions such as malignancies. Yet in some cases intractable pain cannot be demonstrated to be due to physical diseases and is known to occur as a psychogenically determined symptom.

Another problem was that most of the publicized charges repeatedly referred to "lobotomy." This particular technique, the procedure used when psychosurgery was introduced in 1939, has long been superseded by methods more accurate and more limited in their destruction of brain tissue. It was desirable, therefore, to obtain a picture of current procedures, of the specific conditions for which psychosurgery is performed, etc.

The one-page questionnaire (appendix 1) was numbered so that the returns could be followed up. Respondents were given the option of not signing their names, and all were promised that their identity would be kept completely secret. A very large number did, however, sign the form.

METHOD

Questionnaires, which covered the years 1971, 1972, and 1973, were sent on December 31, 1973, to all sen-

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

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TABLE 1
Neurosurgery for Intractable Pain and Psychosurgery in 1971, 1972, and 1973

Neurosurgical Procedure	Number of Neurosurgeons	Number of Procedures: Surgery for Intractable Pain
Surgery for intractable pain only	66	314
Psychosurgery only	77	
Surgery for intractable pain and psychosurgery		
Specified by respondent	23	74
Allocated by author	19	88
Impossible to assign, allocated to psychosurgery	10	
Total	195	476

ior and active U.S. and Canadian members (N=1,450) on the 1973 membership list of the American Association of Neurological Surgeons. The Congress of Neurological Surgeons furnished the mailing list of its members, and questionnaires were sent to those who were not also on the membership list of the AANS (N=451). In response to this combined mailing 1,065 replies were received.

On April 30, 1974, 837 questionnaires were again sent to the neurosurgeons who had not responded. From this mailing 416 returns were received, so that out of the 1,901 neurosurgeons contacted 1,481 replied and from 420 there was still no response.

In order to ascertain where psychosurgery was carried out, every member hospital of the National Association of Private Psychiatric Hospitals was sent a questionnaire. Of the 151 hospitals involved, 139 replied. Of the 12 not responding, on examination of the size, type of patient served, and reputation, it was judged that no psychosurgery would have been conducted in this group of hospitals.

Consideration was given to sending questionnaires to community general hospitals, state hospitals, Veterans Administration hospitals, etc., but it was concluded that the responses were most likely to be sparse and the statistics derived not significant enough to reach any valid conclusions.

RESULTS

There was a 78% rate of return, an exceptionally high percentage for a questionnaire study, which probably reflects the degree of neurosurgeons' interest in a controversial issue involving their special area of professional concern. Of the 1,481 questionnaires that were returned, 18 were returned by the postal service as unforwardable and 35 had been sent to neurosurgeons who were retired or deceased; 1,231 neurosurgeons indicated that they had not performed surgery for the treatment of psychiatric disorders. Of the 197 who had, 2 responses were eliminated, the first because the number of procedures was not listed and the second because the 7 operations reported had been performed outside North America. The breakdown of the remaining 195 responses is shown

Analysis of the responses indicating for both intractable pain and psych demonstrated that they could be su sets. The first set included 23 response number of procedures performed for and those for the treatment of psychi The second set was composed of 19 re tained additional information so that ment could be exercised in allocating for intractable pain and those for ps making this allocation, considerato comments noted on the questionnai surgical procedures reported, and treated, etc. The numbers arrived at sion to assign to psychosurgery rathe for intractable pain those procedu purpose was uncertain.

The third set, which presented great cluded the returns from 10 neurosurge was not possible to distinguish specif cedures for pain from those for psych In order to avoid the charge of bias r surgery, all such returns, covering were placed in the psychosurgery cat doubtedly true that this group, the e least a significant number of procedure conditions other than psychiatric.

Of the 110 neurosurgeons in the Ju performed psychosurgery in 1971, 19 specified having performed a total of the 635 procedures performed by the o ther determined to be psychosurgery o as such, yielding a total of 972 proce year. There were 18 Canadian neu specified having done a total of 61 psy cedures and 1 whose 6 procedures were total of 67 procedures or 23 per year.

A total of 420 questionnaires were n though the reasons may vary, it is like jority of nonrespondents had little inte surgery. Nevertheless, assuming the s formance by them as by the respondent 276 procedures would give a correcte operations. Because only a handful of

TABLE 2
Number of Psychosurgical Procedures Performed by Individual Neurosurgeons in the United States and Canada in 1971, 1972, and 1973

Procedures Performed	1971						1972					
	Number of Neurosurgeons			Number of Procedures			Number of Neurosurgeons			Number of Procedures		
	U.S.	Canada	Total	U.S.	Canada	Total	U.S.	Canada	Total	U.S.	Canada	Total
1	36	9	45	36	9	45	27	12	39	27	12	39
2	19	5	24	38	10	48	18	3	21	36	6	42
3	5	1	6	15	3	18	5	0	5	15	0	15
4	2	0	2	8	0	8	5	1	6	20	4	24
5	1	0	1	5	0	5	5	1	6	25	5	30
6–10	5	0	5	40	0	40	4	0	4	32	0	32
11–15	4	0	4	56	0	56	3	0	3	35	0	35
16–20	0	0	0	0	0	0	1	0	1	20	0	20
21–40	3	0	3	110	0	110	2	0	2	68	0	68
41–60	0	0	0	0	0	0	0	0	0	0	0	0
61–80	0	0	0	0	0	0	1	0	1	65	0	65
Over 80	0	0	0	0	0	0	0	0	0	0	0	0
Total	75	15	90	308	22	330	71	17	88	343	27	370

were Canadians, the corrected number was allocated to neurosurgeons in the United States, yielding a total of 1,248 procedures or 416 psychosurgical operations per year in the United States.

It may be of interest to compare the incidence of psychosurgery in the United States and Canada with that in other countries. According to the report of the National Commission on the Protection of Human Subjects of Biomedical and Behavioral Research (2), it is estimated that between 200 and 250 such operations were performed annually between 1973 and 1975 in the United Kingdom and approximately 83 in Australia. Based on population, the rate in the United States is half that in the United Kingdom and one-third that in Australia.

For a variety of reasons, including accusations that had been leveled about the abuse of psychosurgery, but more specifically to determine the distribution of psychosurgery, the frequency of operations by individual neurosurgeons was obtained by a breakdown of the number of procedures by year (table 2). The total of the numbers of neurosurgeons, of course, includes some operating in one, two, or three years or any combination of years. It is extremely interesting to note that a relatively small proportion of the neurosurgeons responding reported having carried out psychosurgical procedures in all three years.

Also of interest are the figures resulting from removing just 4 neurosurgeons who carried out more than 20 procedures in any one of the three years. In 1971 72 neurosurgeons in the United States performed 198 procedures, in 1972 68 performed 210 procedures, and in 1973 57 carried out 167 psychosurgical procedures, yielding a total of 575 psychosurgical operations carried out by those U.S. neurosurgeons who performed fewer than 20 procedures in any one of the three years. It will be noted that there was a decline in 1973 in the number of neurosurgeons and of the procedures they performed. This decline from 1971 to 1973 was, without doubt, the consequence of the notoriety that was generated by adverse publicity regarding psychosurgery.

The statistics on psychosurgical procedures performed by individual neurosurgeons are of interest because the question has been raised whether infrequency of operation is related to proficiency and to results. On the other hand, it has been argued that increased frequency is related to lack of discriminating selection of patients. Of the U.S. neurosurgeons who performed psychosurgery in 1971, 1972, and 1973, there were 62, 55, and 49 neurosurgeons in the respective years who performed 4 or fewer operations. There were 13, 16, and 10 neurosurgeons performing 5 or more procedures in those years.

It should be remembered that the number of procedures listed as psychosurgical is undoubtedly far greater than the number actually performed for the treatment of psychiatric disorders because of the many unspecified neurosurgical procedures designated psychosurgery for the reasons stated in this section (see table 1).

Nonrespondents

With regard to the 420 questionnaires not returned, there were a few letters from neurosurgeons of varying persuasions, giving their reasons for not responding—including more than one offering the advice that a prepaid, self-addressed envelope might produce a greater response. Some neurosurgeons who were opposed to psychosurgery wrote explaining their positions and giving names of neurosurgeons who were not members of their organizations and who performed psychosurgery. These were followed up.

Study of the names and affiliations of nonrespondents revealed that with two exceptions none had published clinical reports on psychosurgery or had received publicity in the numerous newspaper articles on psychosurgery making headlines at that time. Of

1973					
Number of Neurosurgeons			Number of Procedures		
U.S.	Canada	Total	U.S.	Canada	Total
28	8	36	28	8	36
10	3	13	20	6	26
8	0	8	24	0	24
3	1	4	12	4	16
1	0	1	5	0	5
3	0	3	26	0	26
4	0	4	52	0	52
0	0	0	0	0	0
1	0	1	22	0	22
0	0	0	0	0	0
0	0	0	0	0	0
1	0	1	132	0	132
59	12	71	321	18	339

the two exceptions, one was known not to have been recently active in operating on psychiatric patients. The other was a surgeon whose work had been a focus of attention; the exact number of his cases was not known, but, on the basis of information from other sources, the number is more than compensated for in the above statistics.

With respect to the extent of psychosurgery by a number of nonrespondents, a series of inquiries was made of other physicians, principally psychiatrists practicing in the same geographical area. These inquiries, mainly in metropolitan areas of the United States, revealed that psychosurgery had been carried out by a few of the nonreporting neurosurgeons but in no case was there a significant amount. This included two neurosurgeons not listed in the membership of either the American Association of Neurological Surgeons or the Congress of Neurological Surgeons. It was concluded that the inclusion of all doubtful and unspecified operations under the category of psychosurgery (table 1) plus the statistical correction for nonrespondents more than allowed for procedures not reported.

DISCUSSION

Careful study was made of the published articles and statements of those neurosurgeons who had been the focus of charges of abuse of psychosurgery, while information was sought from other sources, mainly psychiatrists, who were likely to have some knowledge about patients who had been operated on by them. Information about the demographic aspects of patients was also sought from a sample of psychiatrists associated with published reports on psychosurgery. No information was developed which supported the accusations that were the impetus for the task force study.

The National Commission for the Protection of Human Subjects of Biomedical and Behav[illegible] issued its final report and recommend[illegible] March 1977. The APA Board of Truste[illegible] task force report in October 1976. The [illegible] tions and findings of the National Co[illegible] remarkably close to those of the task fo[illegible] difference between the recommendati[illegible] and those of the Commission was w[illegible] forming psychosurgery on minors. The [illegible] ommended that "with the present sta[illegible] psychosurgery should not be perfor[illegible] nors." The Commission, on the other h[illegible] wish categorically to deny children the p[illegible] tages of a new therapy that might be [illegible] effective than long-term use of other t[illegible] phasizing at the same time the neces[illegible] including court review.

Both studies concluded, among oth[illegible] psychosurgery may be an effective tre[illegible] resort in selected psychiatric disorders [illegible] desirable effects have been exaggerated [illegible] tion, often by erroneously attributing t[illegible] niques those adverse consequences w[illegible] the earlier prefrontal procedure. Both [illegible] cluded that there is no reliable evidence [illegible] surgery has been used for political p[illegible] control, or as an instrument for racist [illegible]

In the allotted space, it is not possi[illegible] analyses of the kinds and frequency of [illegible] surgical approaches now used or of the [illegible] conditions so treated. Neither has the in[illegible] garding the neurosurgical treatment of [illegible] been presented.

As mentioned earlier, there have bee[illegible] in the past to ascertain—but much gu[illegible] the number of psychosurgical procedu[illegible] in the United States. The high numbe[illegible] ents—78%—together with extensive in[illegible] ing the practices of those not returnin[illegible] naire should set at rest the fears th[illegible] aroused in the minds of so many.

Let it be hoped that the unfounded cl[illegible] those by psychiatrists, that thousands o[illegible] are performed annually for nefarious p[illegible] lated to the relief of otherwise intractal[illegible] disorders have ended and will no longe[illegible] anxieties of the public.

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APPENDIX 1
American Psychiatric Association Task Force on Psychosurgery Survey Questionnaire

1. Have you performed any neurosurgical procedures for treatment of psychiatric patients in the period January 1, 1971, to September 30, 1973? Yes _____ No _____
(If no, please indicate and return the questionnaire.)

2. If yes, how many in 1973 _____, 1972 _____, 1971 _____?

3. Number of procedures performed?

	1973	1972	1971
1) Orbital undercutting			
2) Cingulotomy			
3) Bimedial leucotomy			
4) Amygdalotomy			
5) Thalamotomy			
6) Posterior hypothalamotomy			
7) Standard lobotomy			
8) Other			

4. What techniques have you used?

	1973	1972	1971
1) Surgical (scalpel)			
2) Stereotaxis			
3) Isotopes			
4) Ratio frequency current			
5) Other			

5. For which conditions have the procedures been used?
   1) Intractable pain _____
   2) Tension and anxiety states _____
   3) Phobias _____
   4) Depression _____
   5) Obsessive-compulsive states _____
   6) Paranoid states _____
   7) Schizophrenic states _____
   8) Behavior problems (e.g., repeated violent behavior) _____
   9) Sexual psychopathology _____
   10) Hysterical syndromes _____
   11) Other _____

Signature _____
(optional)

Special Issue of *American Journal of Psychiatry* Planned

A special issue of the *American Journal of Psychiatry* will be published in mid-April 1979. It will consist of a collection of articles on the subjects of alcohol, depression, or both.

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First-Rank Symptoms in the Postacute Schizophrenic: A Follow-Up Study

BY MARSHALL L. SILVERSTEIN, PH.D., AND MARTIN HARROW, PH.D.

Schneider's diagnostic system of first-rank symptoms (FRS) is acknowledged by psychiatrists throughout the world as a decisive basis for the diagnosis of schizophrenia. Recently, the authors' and others' works have challenged this view. This report examines the relationship between FRS, psychotic symptoms, prognosis, and outcome at the postacute stage. The assessment of FRS was made from an interview schedule (a modification of the Wing Present State Examination) developed for the systematic assessment of psychotic symptoms. The results question the utility of FRS as a primary diagnostic approach to schizophrenia, suggesting that Schneider's system may not be superior to other diagnostic approaches.

KURT SCHNEIDER'S diagnostic system of first-rank symptoms (1) is acknowledged by a large number of psychiatrists throughout many parts of the world as the decisive basis for a diagnosis of schizophrenia. Although widely used by psychiatrists throughout Europe, first-rank symptoms (FRS) as a diagnostic system are not as well known in the United States. Since important theoretical formulations and treatment strategies in schizophrenia require reliable diagnostic criteria, the investigation of the utility of a diagnostic system that is used as widely throughout the world merits careful study.

Despite Schneider's claim that, in the absence of an organic brain syndrome, FRS occur only in schizophrenic patients (1), recent reports by Carpenter and associates (2, 3) and Newmark and associates (4) have challenged the view that FRS are pathognomonic of schizophrenia. Our own research (5) has
FRS are less prominent than other
schizophrenic patients who show psychot
after the acute stage of the disorder. Th
port extends these findings by examin
lence of Schneider's FRS in schizophren
schizophrenics during the postacute sta
was to assess whether FRS are more cha
schizophrenia than are a broader range
symptoms.

Schneider's diagnostic system is de
cal experience and is often associated w
nologic method of study of psychot
though his views on the course and ev
in schizophrenia were closest to those
Schneider maintained that psychiat
should be based on the presenting symp
the course of the illness. Recent resea
Taylor (6) and Carpenter and associate
vestigated relationships between Schnei
tic system and prognosis and outcome
which assessed FRS at the acute stage
generated conflicting results concern
fectiveness of FRS as a diagnostic syste
sphrenia. The present study extends the
tions by examining key characteristics of
ing relationships with prognosis and ou
the postacute stage, an important period
are less disturbed by flagrant psychot
approach was designed to enhance ou
of the theoretical relevance of FRS to
schizophrenic disorders.

The relationship between FRS and
psychotic symptoms over the long-te
schizophrenia has not been studied ext
lor (7) studied FRS in chronic schizophr
been hospitalized for many years. We rec
ed (5) that a large percentage of schizo
played psychotic symptoms several ye
acute illness, and further that FRS were
lent in these postacute schizophrenic pati
postacute nonschizophrenics. In this resea
focuses on psychotic symptoms in general.
inary indications suggested that FRS we
tremely common. This suggested to us th
der's criteria may not be stable or longsta
tures across different phases of the sch
disorder. FRS exclude a broad range of
symptoms, present in many schizophrenics

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The authors are indebted to Roy R. Grinker, Sr., M.D., and Philip Holzman, Ph.D., for providing information on the current patient sample via interviews and other studies of the patients at an earlier phase of their disorder. They also wish to thank Roberta Laffey, Joanne Buckley, Francine Brennan, and Kathleen Checker, who aided in collection and analysis of data.

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described in *DSM-II*. Although Schneider did claim that in the individual case schizophrenia could be diagnosed in the absence of any FRS, his emphasis on their primacy in the diagnostic process has influenced recent applications of FRS as a complete system. Thus, our investigation also attempts to examine the relationship between FRS and psychotic symptoms, with a particular emphasis on the relative discriminating power of these indices in distinguishing important characteristics of relatively young schizophrenic and nonschizophrenic patients.

Our research examines the relationship between FRS, psychotic symptoms, prognosis, and outcome, using a larger sample than in our previous follow-up study. Specifically, the following questions are addressed: 1) How prevalent are FRS in schizophrenics and nonschizophrenics at the postacute stage? 2) Are FRS more sensitive than a broader range of psychotic symptoms in identifying schizophrenic patients at the postacute stage and in distinguishing between schizophrenics and nonschizophrenics? 3) Do patients with FRS differ from patients with a broader range of psychotic features in areas other than symptomatology? 4) Do major prognostic indices associated with premorbid history and the onset of illness predict whether FRS persist beyond the acute stage in a sample of young early schizophrenics?

METHOD

Patient Sample

The patient sample consisted of 126 patients hospitalized at Michael Reese Medical Center during relatively early phases of their disorder. These patients, who were between the ages of 17 and 35 years, were studied at the time of hospitalization and then later assessed during the posthospital period (an average of 2.7 years after discharge from acute psychiatric hospitalization). These patients were part of the ongoing multidisciplinary schizophrenia project being conducted at Michael Reese Hospital and the University of Chicago (8, 9).

The total sample consisted of 2 major subgroups: 72 schizophrenic and 54 nonschizophrenic patients. The schizophrenic population included a subsample of 10 acute schizophrenics, 18 paranoid schizophrenics, 6 schizo-affective schizophrenics, 27 relatively young chronic schizophrenics, and 11 patients in other subdiagnostic groups. The mean age of the schizophrenics was 22.7 years at the time of the index hospitalization. The nonschizophrenic sample was composed of 31 patients with depressive disorders, 16 patients with severe personality disorders, and 7 patients with other varied diagnoses. The mean age of the nonschizophrenic group was 22.1 years at the time of the index hospitalization. At the time of the follow-up assessment 47% of the schizophrenics were on phenothiazines. During the postacute period, the schizophrenics on phenothiazines had more psychotic features ($p<.01$) than those who were phenothiazine-free. In many cases, however, their poorer clinical condition may have been the reason for phenothiazine therapy. Thus, caution in interpreting the results comparing different medication groups is warranted, since the patients were not assigned to the medication groups on a random basis. The schizophrenics receiving psychotherapy at follow-up did not differ in the prevalence of FRS from those not receiving psychotherapy.

Data Collection on First-Rank Symptoms and Other Psychotic Symptoms

The subjects were contacted to appear for an interview that was part of a series of investigations studying psychosis and other major symptoms in schizophrenia (10, 11). The assessment of psychotic symptoms (including FRS) was obtained from a structured interview, the Psychotic Symptoms Inventory (PSI), which we developed using items from the ninth edition of the Present State Examination (12) and Strauss and Carpenter's modification of the eighth edition of the Present State Examination (13).

The interview covered 17 categories of psychotic symptoms, which were then combined into composite ratings on 3-point scales (1=symptom absent, 2=weak or infrequent features, or probable but not absolutely certain symptoms, and 3=symptom present). These composite ratings were in four areas: 1) delusions, 2) hallucinations, 3) other sensory aberrations and/or strange experiences, and 4) an overall psychotic symptoms summary rating. The Psychotic Symptoms Inventory (PSI) interview schedule and rating system was described in fuller detail in a previous report (5).

Each of Schneider's FRS was investigated systematically, and ratings (also on 3-point scales) were obtained for the individual FRS. In order to maximize precision in the assessment of these symptoms, careful and detailed inquiry was conducted with the special care required in defining FRS emphasized by Schneider and more recently by Wing and Nixon (14). A summary rating (based on the presence or absence of at least 1 FRS) was also obtained.

The specific interview questions for the assessment of FRS included those described by Carpenter and associates (2), who studied the following 8 FRS: audible thoughts, somatic passivity, thought insertion, thought withdrawal, thought broadcasting, made feelings, made impulses, and made volitions. We also obtained information on the following additional FRS identified by Schneider: voices arguing, voices commenting on one's actions, and delusional perceptions. Each of the 17 categories of psychotic symptoms included an item such as "What do you make of these experiences? Do they have a special meaning or significance for you?" Interviewers were trained to make careful distinctions between delusional perceptions and delusional notions; this is generally regarded as a particularly difficult discrimination. Thus, in order for a delusional perception to be judged present, positive responses were required to satisfy the following four criteria: 1) the

phenomenon is a *normal* perception (hallucinations and strange sensory experiences are excluded); 2) the perception is interpreted in a delusional manner with abnormal unrealistic significance ascribed to the perception (e.g., a salt shaker is perceived normally but is believed to be an omen); 3) special personal meaning or self-reference is present; and 4) special meaning or a quality of urgency is present.

Data Collection on Outcome and Prognostic Indicators

A second structured interview, administered as part of the follow-up evaluation, provided a systematic detailed assessment covering functioning in the areas of social and work adjustment and rehospitalization, as well as a global composite (15), during the 12-month period prior to the follow-up evaluation. Other ratings were systematically obtained at the time of index hospitalization, including the prognostic indicators used successfully by Vaillant (16, 17) and Stephens and Astrup (18) and the Phillips Scale of Premorbid Adjustment (19, 20).

RESULTS

Prevalence of First-Rank Symptoms

Using a larger sample than in our previous report (5), we determined that FRS occurred significantly more frequently among schizophrenic than nonschizophrenic patients during the postacute phase of their disorder ($\chi^2=8.05$, $p<.01$). However, only 24% of the schizophrenic patients showed definite evidence of Schneider's signs: thus, FRS occurred relatively infrequently among schizophrenics as a group. Of the 72 schizophrenics 76% were free of FRS at the postacute phase. The fact that only 4% of the 54 nonschizophrenics had FRS after the acute phase of their disorder could be considered as one type of evidence in support of diagnostic systems based on FRS.

These data are in accord with our previously reported findings that while FRS do not occur in most schizophrenics (diagnosed according to *DSM-II*) after the acute stage, they do occur more frequently in these patients than in nonschizophrenics. An important question centers around whether the schizophrenics with FRS differ from those with any psychotic symptoms when they are compared in areas other than symptomatology. This issue is explored later in this report.

Efficiency of First-Rank Symptoms Compared with a Broader Range of Psychotic Symptoms

In evaluating the utility of any diagnostic system for schizophrenia based on FRS, a question of central importance arises that has not been explored fully in the past. This question is whether the use of FRS as a key index for the diagnosis of schizophrenia is superior in any way to the use of other combinations of psychotic symptoms. Specifically, one value of a diagnostic system should center around its efficiency in identifying important characteristics of psychopathology associated with the syndrome. This raises the question of whether patients diagnosed schizophrenic on the basis of FRS show a different premorbid background, course, or outcome than the group of schizophrenic patients with any psychotic symptoms. Psychotic symptoms defined in other ways than FRS, for instance, are FRS associated with schizophrenic deterioration more consistently than other combinations of psychotic symptoms, or do they predict chronic schizophrenic outcome more accurately? The question is whether FRS are more useful as prognostic indices of schizophrenia than different combinations of psychotic symptoms. Unless Schneider's first-rank system can be demonstrated to be uniquely related to central characteristics of the syndrome (such as distinctive clinical course) in a uniform manner, it will be difficult to justify contentions that FRS have greater diagnostic validity than any other psychotic symptoms.

Major combinations of psychotic symptoms. The analysis of whether FRS identify an important subgroup of schizophrenics was suggested to us by our previous findings (5), which indicated that while many schizophrenics showed psychotic symptoms at follow-up, a moderate percentage of these patients did not. Therefore, analyses were conducted to examine combinations of psychotic symptoms to determine the relative efficiency of FRS compared with other psychotic symptoms in discriminating between schizophrenic and nonschizophrenic patients during the acute stage.

In order to examine some of these issues, we divided the schizophrenic sample into patients with and without psychotic symptoms, as assessed by the overall psychotic symptoms index and the various combinations of psychotic symptoms. The findings presented in Table 1 indicate that 1) in the group of schizophrenics with definite evidence of first-rank symptoms, 53% also displayed FRS, whereas the remainder 47% did not have any of Schneider's signs; 2) of the 28 schizophrenics with delusions of reference, 3) all of the 11 schizophrenic patients with auditory hallucinations also showed definite evidence of at least one FRS; and 4) 44% of the schizophrenics who reported strange experiences or sensory aberrations showed evidence of FRS.

Thus, of the *DSM-II*-diagnosed patients with psychotic symptoms during the postacute stage, 47% would not have satisfied criteria requiring the presence of FRS for a diagnosis of schizophrenia. This finding is similar to a recent report by Strauss and Gift (21), who observed that in a sample of first-admission patients less than half of those considered schizophrenic by *DSM-II* criteria also had FRS. Although there are substantial areas of overlap between these diagnostic systems, there nonetheless remain major areas of

TABLE 1
Prevalence of First-Rank Symptoms and Other Psychotic Symptoms Among 72 Schizophrenic Patients

Other Symptom Groupings	FRS Present N	FRS Present %	FRS Absent N	FRS Absent %	χ^2	p
Overall psychotic symptoms						
Present (N=32)	17	53.1	15	46.9	24.95	<.001
Absent (N=40)	0	0.0	40	100.0		
Delusions						
Present (N=28)	15	53.6	13	46.4	20.16	<.001
Absent (N=44)	2	4.5	42	95.5		
Hallucinations						
Present (N=11)	11	100.0	0	0.0	7.37	<.01
Absent (N=61)	6	9.8	55	90.2		
Other strange experiences and/or sensory aberrations						
Present (N=9)	4	44.4	5	55.6	1.33	n.s.
Absent (N=63)	13	20.6	50	79.4		

TABLE 2
Correlations Among Various Combinations of Psychotic Symptoms and Other Outcome Indices in Schizophrenia

Symptom Grouping	Social Functioning r	Social Functioning p	Instrumental Work Role Functioning r	Instrumental Work Role Functioning p	Rehospitalization r	Rehospitalization p	Klein Global Outcome Scale r	Klein Global Outcome Scale p
First-rank symptoms	.41	<.01	.30	<.01	.43	<.01	.53	<.01
Overall psychotic symptoms	.50	<.01	.32	<.01	.32	<.01	.54	<.01
All delusions	.42	<.01	.18	n.s.	.23	<.05	.53	<.01

ancy between Schneider's approach and other current approaches such as *DSM-II*—Carpenter and associates' "flexible system" (22), the New Haven Schizophrenia Index (23), Taylor and associates' criteria (24), and, to a lesser extent, Feighner and associates' criteria (25).

Comparisons among different psychotic symptom indices. An important question centers around the relative discriminating power of different psychotic symptom combinations in contrast to FRS. Specifically, we were interested in which combinations of psychotic symptoms at the posthospital phase yield the best discrimination between patients previously diagnosed schizophrenic and nonschizophrenic at the time of the index hospitalization.

Three major indices were thus compared: 1) FRS, 2) all delusions, and 3) a subset of delusions comprised of delusions of reference, control, and/or persecution. The differences between schizophrenics and nonschizophrenics were statistically significant on all three indices ($p<.01$, chi-square analysis). The index of all delusions identified 29 (40.3%) of the schizophrenics; 24 (33.3%) of the schizophrenic patients had delusions of reference, persecution, and/or control. The index of FRS identified fewer of the schizophrenics (N=17, 23.6%) than either of the two other psychotic symptom indices. Thus, for this purpose the FRS was the least effective of these three indices of psychotic symptoms at the posthospital phase. This result is of particular interest, since Carpenter and associates (26) reported FRS to be less effective than other combinations of signs and symptoms in identifying diagnosed schizophrenic patients at the time of index hospitalization. Very few of the nonschizophrenics (3.7%–7.4%) showed any psychotic symptoms (including FRS) when these three indices at the postacute stage were used.

It appears that although FRS after the acute stage had discriminating power in differentiating schizophrenics from nonschizophrenics, this approach did not offer the best differentiation between these patient groups. There appears to be no compelling evidence to suggest that FRS after the acute stage are better or worse than other indices of psychotic features in discriminating schizophrenic from nonschizophrenic patients when current standard *DSM-II* criteria are used for establishing the original diagnosis.

First-Rank Symptoms and Outcome

The data on several aspects of outcome functioning at the postacute stage indicated that both FRS and a broader range of psychotic symptoms were related to other aspects of functioning. In table 2, it can be seen that schizophrenic patients with FRS and those with definite evidence of any psychotic symptoms were functioning poorly in other areas, although these correlations were far from perfect.

These data on other aspects of functioning were used to examine whether FRS at follow-up were more closely related to schizophrenic outcome than other combinations of psychotic symptoms. The results for schizophrenic patients indicated that 1) 53% with FRS and 47% with a broader range of psychotic symptoms showed poor social functioning; 2) 59% with FRS and 53% with a wider range of psychotic symptoms had poor work or instrumental role functioning; and 3) 65% with FRS and 75% with any psychotic symptoms had been rehospitalized since the index hospitalization.

In each specific outcome area a comparable proportion of schizophrenic patients with poor outcome was regularly identified by both the index of overall psychotic symptoms and of FRS. In regard to these specific outcome assessments, it appears that the presence or absence of Schneider's FRS during the postacute phase was not an automatic correlate of outcome functioning. Although in select areas there was a limited, nonsignificant tendency for the schizophrenic patients with FRS to have a somewhat poorer outcome compared to those patients with any psychotic features at follow-up, the evidence was not compelling. Our data on relatively young schizophrenic patients do not suggest that FRS as the basis for a diagnostic system in schizophrenia offer unique advantages in characterizing outcome in schizophrenia as compared to other psychotic symptoms.

First-Rank Symptoms and Prognosis

We explored whether seven major prognostic indices obtained at the time of the index hospitalization predict FRS during the posthospital period. These seven prognostic indices included 1) the Phillips Scale of Premorbid Social-Sexual Adjustment (19, 20), 2) acute onset, 3) precipitating factors, 4) emotional blunting on admission, 5) premorbid social adjustment, 6) premorbid work adjustment, and 7) premorbid schizoid personality (16–18). A series of correlations was obtained between each of these prognostic variables and both FRS and the overall psychotic symptoms index.

The results indicated that FRS at the postacute stage were not predicted by any of these prognostic variables. All of these correlations were very low and statistically nonsignificant. The absence of a relationship was also observed between these prognostic variables and the overall index of psychotic symptoms. Therefore, these data indicated that major prognostic indices used by Vaillant, Stephens, and others did not appear to be predictive of the occurrence of FRS after the acute stage, nor, for that matter, was there a strong relationship between these prognostic indicators and any type of psychotic symptomatology at the postacute stage.

DISCUSSION

After combining the results from several substantive areas of investigation, we question whether FRS are more important than any other psychotic features in schizophrenia.

With regard to prevalence, our data indicate that Schneider's FRS occur more frequently in schizophrenic than in nonschizophrenic patients at the postacute stage. This suggests that these symptoms are likely to persist over time for select patients and thus show a moderate degree of continuity over the long-term course of the disorder. The fact that some of our preliminary indications suggest that FRS may tend to be more prevalent among chronic and paranoid schizophrenic patients could offer some limited evidence supporting the utility of FRS, providing that this trend continues to hold up. However, FRS were not very common in the present sample of young adult schizophrenics, and, in addition, when psychotic symptoms of any type were present at the postacute stage, these psychotic features frequently were not FRS.

The comparison of the relative discriminating power of FRS and other psychotic symptoms indicated that FRS after the acute phase offer no more powerful distinctions between patients diagnosed schizophrenic and nonschizophrenic at the time of the index hospitalization than a variety of other combinations of psychotic symptoms. Therefore, there does not seem to be an apparent advantage in Schneider's system compared with other systems based on alternate combinations of psychotic symptoms in discriminating schizophrenic from nonschizophrenic patients at the postacute stage. This finding coincides w
observation by Carpenter and associates
data collected at the time of index hosp

In regard to major prognostic indicate
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These data further challenge assumpti
about the theoretical and practical ut
der's diagnostic system as a decisive bas
nosis of schizophrenia. It is well know
diagnostic procedures currently prev
country are broader in scope than mo
nostic systems in wide use elsewhere, w
clude Schneider's system. Given the ce
lem of varying criteria for the diagnosis
nia, a crucial question is which s
effective in delineating important char
as prognosis, clinical course, and outc
FRS may offer potential advantages in
bility of symptom assessment and re
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whether a broad or a narrow definitio
nia is preferable. We have attempted
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DSM-II-diagnosed schizophrenics wit
up and comparing them with a samp
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low-up. We have used key indices tha
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Overall, the results indicate a few
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are traces of potentially positive findin
considered in total this system fails to
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nations of psychotic symptoms.

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Hyperkinetic/Aggressive Boys in Treatment: Predictors of Clinical Response to Methylphenidate

BY JAN LONEY, PH.D., ROBERT J. PRINZ, ED.S., JOEL MISHALOW, M.A., AND JESSE JOAD

Data on 84 nonretarded boys aged 6–12 with the hyperkinetic/MBD syndrome were drawn from a comprehensive, longitudinal investigation in the interest of identifying factors that contribute significantly to variation in clinically rated improvement during treatment with methylphenidate. The size of the multiple correlation (R=.50) indicates that 25% of the variation in the children's response to methylphenidate is jointly predictable from age at referral, degree of perinatal complications, and score on the hyperactivity factor. The authors discuss stepwise multiple regression analysis as the method of choice in drug response prediction studies and the possible effects on the results of such studies of differing definitions of improvement.

EXPERIENCE, CLINICAL LORE, and reports of controlled studies have led many physicians to prescribe CNS stimulant medication as the initial and primary treatment for the child diagnosed as having hyperkinetic reaction or minimal brain dysfunction (HK/MBD). There is little doubt that stimulant medication has at least a short-term positive effect on the behavior of a certain percentage of hyperactive children. At times the behavioral effect is dramatic, with a marked reduction in activity level and fidgetiness, a lengthened attention span, etc. However, HK/MBD children do not form a homogeneous group, and it is well documented that stimulant medications are not equally effective in the management of all cases. Recently cited examples of the overuse and misuse of these drugs (1) and studies describing their potentially dangerous side effects (2) remind us that scrupulous attention must be focused on the question of who is to receive such medications.

Predicting the response of hyperkinetic children to stimulant medication has been a small but significant issue in the literature (3–5). It is apparent that this body of research is in considerable disarray, with some predictive factors operating inconsistently at different times in opposite directions from one study to another. This is probably due at least in part to the use of various definitions used to select treatment samples; heterogeneity of drugs, dosages, and lengths of treatment time employed; variations in design and control procedures; and differing criteria considered to measure improvement (3).

Although certain possible predictors are often advanced (e.g., "organicity" or attention deficits), there are few cross-validation data. Furthermore, the relationships found between predictor and outcome variables, even when statistically significant, rarely are more than "modest" in size. Thus, as Sprague (6) noted, "to date no basis exists for predicting whether a specific hyperactive child will benefit from drug intervention, or for determining which medication will be most effective and what the minimum effective dosage is for him."

The present study was stimulated by an interest in identifying factors that contribute significantly to variation in clinically rated improvement among HK/MBD boys during a treatment trial of methylphenidate.

METHOD

The Iowa HABIT project (Hyperkinetic Aggressive Boys in Treatment) is a comprehensive, longitudinal investigation of the hyperkinetic/minimal brain dysfunction syndrome. The data to be considered here were drawn from a part of the project that focuses on information contained in outpatient medical charts routinely maintained by the University of Iowa Psychiatry Service.

Subjects

Of all 4- to 12-year-old boys seen for outpatient diagnostic evaluation by the University of Iowa Child Psychiatry Service between January 1, 1967, and September 1, 1972, 482 had not been treated with drugs within the previous 6 months. Sixty-two percent (300) were recommended by senior psychiatrists for trials of cerebral stimulants because their history and symptom picture suggested minimal brain dysfunction. Of these 300 boys, 59 were excluded from the HABIT project sample because either their tested IQ's were less than 70 or they were suffering from peripheral

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sory loss, major psychosis, epilepsy, cerebral palsy, or unequivocal brain damage.

The HABIT sample was further limited to boys whose drug treatment had been initiated and followed by staff psychiatrists and/or psychiatric residents of the University of Iowa Child Psychiatry Service. Subjects were included only if drug treatment had started within 6 weeks of the day of the outpatient evaluation and at least one treatment progress note had been placed in the medical record before termination of drug treatment, addition of other drugs or therapies, or referral to a local physician for drug follow-up.

Application of these exclusionary criteria resulted in a sample of 135 boys. This HABIT group includes boys from both rural and more urban areas of Iowa and several surrounding states, and 98% of the sample is white, a figure similar to that for the Iowa general population. The families' Hollingshead and Redlich (8) socioeconomic classes were as follows: I and II, 21%; III, 33%; IV, 26%; and V, 20%. Of the 135 boys 95 (70%) were given an initial diagnosis of hyperkinetic reaction of childhood (*DSM-II*, 308.0).

Before we performed the analyses that will be described, a more homogeneous subsample with minimal amounts of missing data was formed by selecting boys who were 1) between 6 and 12 years of age at the time of the outpatient evaluation, 2) living in intact families (i.e., both a mother and father figure present in the home) at the time of referral, 3) placed on methylphenidate within 6 weeks of the outpatient evaluation, and 4) between 12 and 18 years of age at the time of subsequent 5-year follow-up (9). The resulting subsample consisted of 84 boys whose average age at referral was 9.2 years. The mean full scale IQ of the 61 who were given the Weschsler Intelligence Scale for Children at referral was 101.08 (SD=15.18, range=72–139).

Diagnostic and Treatment Procedures

The initial outpatient diagnostic evaluations were scheduled one per morning and supervised by whichever of two senior psychiatrists was assigned to the day in question. The supervising psychiatrist presided over an afternoon meeting at which the psychiatric resident and the outpatient staff members (social worker, psychologist, and educational consultant), who had assessed the child and his family that morning, presented verbal accounts of the psychiatric interview and physical-neurologic findings, social-developmental history, psychological and educational test results, the school report, etc.

The senior psychiatrist was responsible for making the psychiatric diagnosis, prescribing a course of treatment, and dictating a lengthy staff summary. The resident and each of the outpatient staff members dictated a detailed narrative report of their interactions with and impressions of the child and/or parents; these were also typed and placed in the child's chart as the medical-psychiatric summary, the social summary, the psychological report, and the educational report.

After the staff evaluation, most cases were assigned to the psychiatric resident for drug regulation and follow-up. Medication levels were determined clinically. The maximum daily dosage of methylphenidate during the treatment period ranged from 5 to 85 mg (mean=37.5).

Predictor Variables

Symptom severity at referral. Among the variables that have been assessed (9) are ratings of each child by two judges on the severity of primary (central) and secondary (resultant) HK/MBD symptoms (10–12). A pair of advanced graduate students in school psychology independently reviewed the staff, medical, and social summaries and psychological and educational test reports for each child and then rated on a 5- or 6-point scale the severity and/or frequency of 6 primary symptoms (hyperactivity, fidgetiness, inattention, judgment deficits, negative affect, and uncoordination) and 4 secondary symptoms (aggressive interpersonal behavior, compulsivity, self-esteem deficits, and control deficits). The effective rater reliabilities (13) for the symptom ratings ranged from .73 to .92. For analysis, the two judges' ratings were averaged to produce a composite severity score for each child on each symptom.

In another study, Loney and associates (14) performed a principal axis factor analysis and identified two relatively independent factors in these judges' chart ratings of primary and secondary symptoms at referral. The first factor, which accounted for 44.6% of the factor variance, consisted of negative affect (e.g., excitability, irritability, low frustration tolerance), aggressive interpersonal behavior (e.g., bullying, fighting, refusing to mind), and control deficits (e.g., delinquent acts, evasion of rules). The second factor, which accounted for 23.4% of the factor variance, consisted of hyperactivity (e.g., always on the go, overactivity, restlessness), inattention (e.g., inability to concentrate, distractibility, forgetfulness), and judgment deficits (e.g., immaturity, impulsivity). These two factors, labeled aggression and hyperactivity, respectively, are used in the present investigation as the two measures of symptom severity at referral.

Parenting styles. Among the family variables that have been assessed (9) are ratings of each parent by two independent judges on 9 parenting variables. The same set of chart summaries and reports was used to make both the parent ratings and the symptom severity ratings, but the judges in each instance were a different pair of advanced graduate students. The parent rating variables included separate 7-point ratings along both the love-to-hostility and autonomy-to-control parenting style dimensions (15). The effective rater reliabilities (13) for the parenting ratings ranged from .51 to .83. Again, for each dimension the two ratings for each parent were averaged to produce a composite score.

Neurologic and perinatal status. The child's age and a synopsis of the psychiatric resident's neurologic and

physical findings as detailed in the medical summary were retyped. The content of these synopses varied, but they included such presumably salient material as observations of fine and gross motor movement and tests of synkinesia, graphesthesia, tandem walking, cranial nerves, alternating motion rate, postural reflexes, etc. (16). Based on these synopses, two faculty members (a pediatric neurologist and a pediatrician/child psychiatrist) independently rated soft signs of neurologic impairment on a 5-point scale.

Similar independent ratings were made by the pediatrician/child psychiatrist and an obstetrician/child psychiatry resident based on a separate synopsis of the narrative summary of perinatal events as reported by the mother (e.g., mother's reproductive history, details of pregnancy and delivery, child's condition at birth).

The effective rater reliabilities (13) for these two sets of synopses were .83 and .89, respectively, and composite ratings were again used for each.

Socioeconomic status. In devising a measure of socioeconomic status (SES), a preliminary factor analysis was used to reduce by empirical means a relatively large set of conceptually similar variables to a smaller group of more or less independent dimensions that would account for most of the variance in the data set. The variables that loaded on the single resultant SES factor and that were therefore used to generate an SES factor score for each child were father's education (8, 17), mother's education, father's occupation (8, 17), family income, and psychiatric fee. Three categories constituted the psychiatric fee variable: private or full fee, clinic or partial fee, and state (i.e., indigent) or no fee.

Selection of predictors. Based on considerations of completeness, reliability, saliency, scope, and parsimony (18), the final list of 11 variables selected as potential predictors of drug response was age at onset of referral problem, age at referral, perinatal complications rating, neurologic signs rating, aggression and hyperactivity factors, SES factor, and ratings of the mothers and fathers on the love-to-hostility and autonomy-to-control parenting dimensions.

Criterion Variable

All of the children's study charts contained at least one medication progress note. Many of these were written summaries by a physician or social worker of a verbal account of positive, negative, or no change obtained from a parent, teacher, or local professional person. Many other progress notes were written summaries of behavioral observations of and interviews with the child during a return visit to the clinic. Other progress notes took the form of letters from the child's parents or teachers reporting on his condition, letters from a staff person to the child's school or local physician, repeated educational and/or psychological test reports, etc.

The project secretary removed from each progress note any reference to the patient's name, age, or grade in school; the names of all staff persons involved in the case; and any information indicative of socioeconomic class, parent-child relationship, or other variables hypothesized to be related to improvement. The entire set of over 800 amended progress notes was then retyped, 1 per page, and placed in random order. Each note was independently rated on a 5-point response to medication scale (19) by two experienced psychiatricians. The progress notes were then replaced in temporal sequence for each child, and an overall improvement rating was independently assigned to each case by the two raters. Progress notes from an average of 6.8 months of treatment with methylphenidate were rated for each child. Effective rater reliabilities for these drug response measures ranged from .8[illegible] to 9[illegible] and composite ratings of each measure were derived for each child.

A factor analysis was used to reduce empirically the set of several measures of medication response, each of treatment, dosage, and side effects. Six response measures loaded on the single resulting factor and were therefore used to generate for each child a response to medication factor score that served as the criterion variable. These 6 measures were the first response to medication, the first response to the highest dose, the highest rated response, the last response to medication, the mean response over the first 3 months, and the overall response.

RESULTS AND DISCUSSION

Three of the 11 predictors correlated significantly with the response to medication factor score: age at referral ($r=.36$, $p<.001$), perinatal complications ($r=.25$, $p<.05$), and the hyperactivity factor ($r=[illegible]$, $p<.10$). The stepwise multiple regression analysis to predict drug response took these 3 predictors, in order, to produce a multiple correlation of .50, indicating that 25% of the variation in children's response to methylphenidate is jointly predictable from age at referral, degree of perinatal complications reported by mothers, and scores on the hyperactivity factor.

The fact that age is an important predictive variable is puzzling. The child who is referred at an older age apparently differs from the younger child in a way that affects his response to methylphenidate. The appearance of the hyperactivity factor as an additional independent predictor in the analysis indicates that the way in which the older child differs is not simply that he is more hyperactive. There is some evidence that important aspects of HK/MBD children's problem behavior tend to diminish naturally with age (20), thus raising the possibility that the association found here between age and improvement is artifactual.

Some previous investigators (21, 22) have reported a relationship between drug response and one or more variables from a subset of "organic" predictors. The support lent to the hypothesized connection between organicity and drug response by the appearance of

perinatal complications in the present prediction equation must be balanced against the nonappearance of the more direct assessment of neurologic signs.

The least important of the 3 significant predictors of drug response in the present study is the hyperactivity factor, an unweighted sum of the child's standard scores on the symptom ratings of hyperactivity, inattention, and judgment deficits (impulsivity). Its appearance as a predictor is compatible with a connection between inattention and drug response recently hypothesized by Barkley (3).

Although a well-known study by Conrad and Insel (21) linked parent characteristics and drug response, we found no parenting or SES predictors of response to methylphenidate. Further, the child's degree of aggression did not influence drug response. This study thus does not provide a basis to advocate withholding drug treatment in the belief that environmental and symptomatic complications preclude good response. On the other hand, the fact that older children in this study responded better to medication than younger ones means that those practitioners who would opt for the use of CNS stimulants as a "last resort" should find nothing in these data to dissuade them.

Few investigators seeking to identify possible predictors of HK/MBD children's responses to stimulant drugs have used the stepwise multiple regression method, although it has several appealing features that probably make it the method of choice. Like ordinary two-variable linear correlation (Pearson r), this technique enables one to identify independent variables that are correlated with (i.e., that predict or contribute to variation in) the dependent variable. Unlike ordinary correlation, however, stepwise multiple regression enables one to assess the relative importance of each of the independent variables in terms of the specific amount of variance in the criterion variable for which it independently accounts. In the present analysis, for example, one can conclude not only that scores on the hyperactivity factor predict scores on the response to medication factor but also that 1) the hyperactivity factor is a relatively less important predictor than age at referral or perinatal complications, and 2) it makes a predictive contribution independent of both age at referral and the perinatal complications ratings. Because the addition of the hyperactivity factor increases the multiple correlation from .44 to .50, it is also possible to conclude that the hyperactivity factor accounts independently for only .36% of the variation in scores on the response to medication factor. Since response to stimulant drugs is probably multiply determined, the continued use of stepwise multiple regression or other multivariate methods (23, 24) holds considerable promise.

Although correlational data do not allow the identification of cause and effect, the three independent variables in this study (perinatal complications, age at referral, and severity of hyperactivity) could plausibly be considered actual precursors of drug response. One could in fact use a multiple regression equation to predict in advance of prescribing medication a precise rating for a given child on a medication response scale, and one could base the decision to initiate or to withhold medication on the value of that predicted rating. Of course, careful derivation and cross-validation of any such predictive equation would be necessary. Those who believe that there are no significant risks in using medication might argue that such a predictive approach is not even needed; all that is required is to initiate a drug trial and to observe response. Given the modest size of the multiple correlation in the current study and the likelihood of its shrinkage on cross-validation, the clinical utility of these three identified predictors is probably limited. An identical point could be made concerning other predictor variables proposed to date (21, 22); their present interest is more theoretical than practical.

The current study used a global response to medication score derived retrospectively from contemporary narrative progress notes. This approach has disadvantages that operate to reduce the reliability and, consequently, to compromise the validity of the findings. The data are less objective, focused, and systematic than one would wish, and they are filtered through the multiple perspectives of parents, teachers, treating physicians, and raters. For these reasons, one might consider the present results as generating hypotheses about predictors of response to methylphenidate rather than as identifying predictors. However, these data are derived from exactly the sort of global response information that clinicians routinely collect and use in assessing effects and titrating dosages of medication. To this extent, the findings here should reflect predictions that could actually be made in similar settings.

Much of the difficulty in arriving at a settled opinion on the predictors of response to CNS drug treatment arises from difficulties in defining response. Some investigators, for example, have used an initial response or improvement measure, while others have employed a longer-term response or outcome measure. Initial response or improvement tends to be framed in self-relative terms ("He is more attentive than he used to be") or derived from change scores. Long-term response or outcome tends to be expressed in terms of severity ("He is quite attentive") or in terms of the hypothetical normal child ("He is more attentive than most"). It is one thing if an improved child is one who merely has less of a problem than he used to have and quite another if he is one who has less of a problem than others do. As the work of Weiss and colleagues (25) has recently suggested, the connection between early response to treatment and condition at follow-up is far from simple or strong. In addition, improvement measures based on symptom severity may yield different results from improvement measures based on behavioral frequency.

There appears to be a tendency for the more severely hyperactive child to be rated as having a better response to medication. This might mean 1) that the

more severely hyperactive child actually responds better to medication, 2) that what improvement he does show is merely more discernibly different from his previous high rate of hyperactive behavior than is an equal improvement in a child who was less severely hyperactive or more variable before treatment, or 3) that high hyperactivity scores are simply more likely to display a statistically artifactual regression to the mean which would be mistaken for improvement. Few studies to date have attempted to disentangle these possibilities.

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The Group Psychotherapy Dropout Phenomenon Revisited

BY LESLIE M. LOTHSTEIN, PH.D.

In this two-year study, patients referred for group therapy were extensively screened and carefully prepared for the therapy. In spite of this effort there was a 29% dropout rate after group therapy began. All of the therapists reported that they disliked the patients who dropped out, and many of these patients were seen as hostile toward the therapists. The author suggests that for many groups the dropout phenomenon may be basic to the establishment of group cohesiveness and that instead of trying to prevent or reduce the dropout phenomenon, efforts should be directed toward reconceptualizing the more positive aspects of it.

THE DROPOUT phenomenon, which is reported to occur during the early phases of group therapy, has been recognized as an important obstacle to group formation (1-3). In a classic study of group therapy dropouts, Yalom (4) reported a 36% overall dropout rate during the initial phase of group therapy. He attributed the dropouts to nine factors, one of which, the patient's lack of orientation to group therapy, played a key role. Regardless of the therapeutic approach to group therapy, the dropout rate seems to average about 30%, with a range of 25% to 50% (5, 6). A patient's premature termination of group therapy can pose a significant problem for group formation; Yalom (4) has suggested that adequate patient preparation for group therapy be employed to prevent or reduce this phenomenon.

Although preparing patients (and therapists) for group therapy is well recognized as an important factor in preventing or reducing early dropouts, it is a difficult task even for the experienced therapist (7-9 and Lothstein[1]). The techniques of pregroup patient preparation (PGPP) vary considerably and include having patients listen to tapes of group therapy sessions (10), providing written literature about groups (11), and conducting pregroup individual sessions (8) and pretherapy group sessions (12). What these PGPP techniques have in common is the underlying philosophy that dropouts should be discouraged and that one can prevent or reduce the dropout phenomenon by introducing clarity, structure, goals, and certainty and by reducing ambiguity in the early phases of group therapy.

While group therapy researchers have looked for ways to prevent or reduce the dropout phenomenon, no one has seriously considered some of the positive aspects of this phenomenon. It is my aim in this paper to show that the dropout phenomenon has not been fully understood and that future research to prevent or reduce dropouts should be reconsidered. The findings suggest that the dropout phenomenon may serve a positive clinical function for increasing the integrity of the group while consolidating group cohesiveness.

When I first began my investigation of the dropout phenomenon, I hoped to empirically demonstrate that a structured group therapy training program could prevent or reduce early dropouts by employing a combination of intensive trainee supervision and course work around the problems raised in the initial phases of forming a group. Because of the large effort to prevent or reduce dropouts, it was surprising to learn that approximately 30% of the patients still dropped out of the groups. The findings presented here should be of interest to group therapists who have taken the inevitable 30% dropout rate too personally.

THE STUDY

The results reported here stem from a two-year study of group therapy dropouts at Case Western Reserve. The patient population was drawn from Cleveland's diverse urban community and was a heterogeneous group with respect to social, economic, ethnic, racial, and educational backgrounds. The study was designed to determine whether the dropout phenomenon could be controlled through a combination of factors introduced into the training program. Advanced trainees with diverse educational, vocational, and theoretical backgrounds were enrolled in the program. The student therapists reflected a spectrum of trainees found in most training centers and included three psychiatric residents, nine psychology interns, two social workers, and two staff psychologists. The theoretical orientation of the group reflected a combination of approaches, but the primary focus was on expressive-dynamic group psychotherapy.

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[1]Lothstein LM: Preparing patients for group psychotherapy (unpublished paper, 1975).

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All of the trainees were required to participate in a 2½-month seminar that focused intensively on the PGPP interview. A standardized PGPP interview was introduced.[2] Guidelines for PGPP interviewing were established, and the trainees observed the director of the training program interview prospective group patients using the PGPP method. In addition, all trainees were provided a minimum of 1 year of supervised group therapy experience (using cotherapy teams) and an option to participate in a continuing awareness group for group therapists.

Pregroup Patient Preparation Interview

In the PGPP the patient is systematically oriented to group therapy by means of a standardized interview technique in which three main areas of inquiry are pursued: educative, sensitizing, and evaluative. These areas describe predetermined functions, rules, and procedures that the therapist subscribes to during the PGPP interview. Within each of the three areas of inquiry there is an inventory of questions to be asked of the patient (a total of 27 questions). Although each of the three areas of the PGPP interview may be logically distinguishable from the others, in reality they are intermixed and during an interview may be indistinguishable.

In the educative part of the interview the group therapy contract is spelled out, and the patient is provided with important information about the pragmatics of the group therapy offered. These procedures provide the patient with a reality base with which to assess his or her therapeutic expectations and demystify group therapy.

In the sensitizing part of the interview the therapist helps the patient to confront and work through his or her expectations, fears, concerns, anxieties, hopes, and ambivalence about entering group therapy. In addition, the patient's interpersonal skills and styles are explored along with his or her ability to empathize, be supportive, give and take, be assertive, and benefit from group therapy.

In the evaluative part of the PGPP interview the therapist identifies the patient's level of psychopathology, isolates the patient's primary defensive strategies, and assesses his or her strengths and weaknesses vis-à-vis the other group members. Although a formal diagnosis is helpful, it is also important that the therapist assess the patient's interpersonal functioning and try to determine the effect he or she will have on other members of the group.

The value of employing a standardized PGPP interview technique is that since all patients are similarly prepared for group therapy, it is easy to research the effects of patient preparation on other group measures.

[2]Lothstein LM: Preparing patients for group psychotherapy; two models for the pregroup psychotherapy evaluation; techniques and interview problems in preparing patients for group psychotherapy (unpublished papers, 1975).

Procedure and Findings

Ninety patients initially assigned for g[illegible] were interviewed by the director of the g[illegible] training program and were then assigned [illegible] therapy groups for further evaluation an[illegible] for group therapy. During this period [illegible] were intensively supervised around th[illegible] the initial phase of group therapy and rec[illegible] training on the problems raised durin[illegible]

Of the 90 patients assigned to group [illegible] eventually entered group treatment (an [illegible] rate of 47%). Of these 47 patients, 31 wer[illegible] 16 were men (a ratio of approximately [illegible] tion, there was almost a 4:1 white to bla[illegible] tio, with 37 white patients and 10 black[illegible] white patients consisted of an almost equ[illegible] men and women, while all of the black [illegible] women. The average age was 22.7 years [illegible] women and 24.1 years for men).

About 11 patients were referred to e[illegible] groups. About 2 patients per group dropp[illegible] entering therapy (during their PGPP inte[illegible] another 2 per group eventually dropped [illegible] tering group therapy (all but 1 of the drop[illegible] before the 12th session). It was found tha[illegible] stabilize the patient groups at an average o[illegible] per group, a patient pool of twice that size [illegible]

In addition to the 47% dropout rate fr[illegible] group therapy referrals, there was a 29% [illegible] (N=13) once therapy began. None of [illegible] contributed significantly to a large part [illegible] However, over 70% of the patients (N=34, [illegible] group treatment remained in it. This figu[illegible] than Koegler and Brill's figure of 58% for [illegible] ric outpatients who completed their treatm[illegible] it is consistent with figures from other gr[illegible] research.

The initial pregroup dropout rate may ha[illegible] lated to initial selection criteria that we[illegible] ciently stringent, but it is within the expec[illegible] for group therapy. In the present sample, [illegible] to have a particularly difficult time stay[illegible] therapy. All of the black male patients [illegible] during the initial evaluation phase. In add[illegible] male patients (N=8) accounted for 65% o[illegible] dropped out after entering group therapy. [illegible] ings will be discussed below.

All of the patients who dropped out e[illegible] entering the group or during the course o[illegible] were interviewed by the director of the p[illegible] ther on the phone or in person (100% res[illegible] each patient was asked to complete an [illegible] questionnaire focusing on his or her react[illegible] group therapy experience (40% response. [illegible] addition, all of the patients who completed g[illegible] apy filled out the same questionnaire, and a[illegible] the therapists were interviewed about the re[illegible] patients either not entering group therapy or [illegible] out of a continuing group.

The questionnaire included 18 direct questions (to be completed by the patient) relating to one's overall group therapy experience. The pregroup dropouts who were interviewed on the phone were asked those questions which related to pregroup patient preparation. The patients who dropped out before entering group therapy (i.e., during the evaluation interview) fell into two categories: those who requested individual therapy (20%, N=8) and those who decided not to pursue treatment (27%, N=11). All of the patients who dropped out after entering group therapy reported that they had been adequately prepared for the group, and none of them requested additional treatment. Based on the patients' questionnaire responses, there were no overt characteristics that distinguished the dropouts from the patients who remained in group therapy. However, based on the therapists' reports, there were definite differences between these two groups. These findings will be elaborated below.

Of all the patients surveyed who remained in group therapy, only 1 felt that she was not adequately prepared for the group. All of the others felt that they had received more than adequate pregroup preparation, and all of them reported that they felt positive about their group experience and would recommend group therapy to other patients.

DISCUSSION

In spite of a strong emphasis on pregroup patient preparation, there was a 29% dropout rate for the patients who entered group therapy. Efforts to reduce or prevent group dropouts were initially deemed a failure. The dropout rate seemed high given the fact that the outpatient clinic initially referred suitably motivated patients to the program, the program director evaluated the patients' motivation and suitability, and the cotherapists further evaluated and then prepared the patients for group therapy. At first I wondered whether the extensive screening process might have accounted for the dropouts, especially the male dropouts. However, the overall stability of the therapy groups compared with those several years earlier ruled this out. The group stability was attributed primarily to the effectiveness of the PGPP program. In this sense, preparing a patient for group therapy had an important effect on controlling the early dropout phenomenon, which may lead to group dissolution.

Six of the 8 groups had a male and a female cotherapist, and 2 of the groups had same-sex cotherapist teams (1 male and 1 female team). The type of therapy team did not affect the dropout rate.

All therapists received extensive supervision on the PGPP interviewing, and only those patients deemed suitable for group therapy were assigned. In this way initial questions of suitability for group therapy, degree of psychopathology, and interpersonal styles and abilities had already been assessed by senior clinicians, and some idea of a patient's likelihood to benefit from group therapy was assessed. In addition, all of the patients expressed a positive interest in obtaining group therapy during their initial outpatient evaluation. Thus some of the more formidable work had already been carried out even before the cotherapists interviewed the patient.

The overall composition of the patients who dropped out before the first session differed from that of the group entering therapy in only one aspect: all of the black men in the sample (N=6) dropped out before entering therapy. One factor that may have accounted for so many black men dropping out was the lack of any black therapists. It is also theoretically and practically possible that the therapists and group members were racially biased and transmitted their values to these patients. However, the fact that black women remained in the group seems to preclude racial bias as a sole explanation for the dropout rate among black men. Although the therapists may have perceived the black men as more threatening and dangerous, none of them were dangerously impulsive individuals. It was puzzling that none of the black men requested alternative treatment and that they were unanimous in the view that racial factors were not involved in their reasons for dropping out of group therapy. The phenomenon has not yet been adequately explained.

The fact that white men accounted for 65% of the dropouts (N=8) once group therapy began was also an intriguing finding. It is not known if this phenomenon is peculiar to our clinic or whether men are more stressed by the early issues of group therapy and need to be addressed in a special way.

In reviewing the therapists' explanations for why a patient dropped out of group therapy, the ideas of stress and fit occurred regularly. Therapists saw the initial period as quite stressful for some patients, who either became scapegoated by the group or withdrew because of a fear of involvement. Although the patients' reasons for dropping out focused on such factors as no longer needing treatment and scheduling problems, the therapists focused on dynamic reasons for the dropouts. All of the therapists reported that they disliked the patients who dropped out, and many of those patients were seen as hostile toward the therapist. This finding suggested that there were some very clear characteristics that differentiated the dropouts from the patients who remained in therapy.

It is well known that during the initial period of therapy, it is sometimes difficult to arouse patients' hostile feelings toward the therapist. For some groups it appeared that the group's therapist-directed aggression was being displaced onto the potential dropout, often in the form of not listening to the patient, being unsupportive, interrupting the patient, or being generally unconcerned about the material the patient brought up. In some cases it appeared that the therapists may have been supporting displaced aggression in order to enhance their attractiveness as leaders. By confronting a patient's resistance and expelling him, the leader may have been attempting to consolidate control over the

group by having other group members fearful of the therapist. Some leaders may unconsciously engage in this ritual and may be acting out the group's hostility toward the patient, that is, doing the group's work while also acting out their own sadism. In a dependency group in which there is a patient need for a strong leader, the group members may actively support such a strategy.

Some of the patients who dropped out were able to express their disappointment that the therapists and other patients were disinterested and unsympathetic with them. When these issues were brought back to the group members, the patients' disinclination to discuss the matter supported the dropouts' views. Furthermore, most of the remaining patients stated that the dropouts really did not fit into *their* group.

In no group did the issue of a patient's dropping out arouse the feeling among patients of being on a sinking ship. Rather, the effects of the group's choice not to accept one or two patients seemed to consolidate a feeling of belonging and a sense of "we-ness" among group members that resulted in good group attendance and which, as Yalom (3) pointed out, is a "precondition of effective therapy." Because our training program focused on some of the more thorny issues of group solidarity, we did not experience the phenomenon of the high patient-therapist turnover that usually leads to a noncohesive group.

In analyzing the results of the early group therapy dropouts, it was considered how the diversity of the therapists' backgrounds and level of training, the use of cotherapists, and the inadequate handling of immediate countertransferance issues may have contributed to the 29% dropout rate. In addition to these factors, it was considered how the patients may have been inadequately assessed or misdiagnosed or how some of the 9 factors related to group therapy dropouts reported by Yalom (4) may have been operating against us.

Whatever explanations arrived at to account for the dropout phenomenon, one thing was clear: after the 12th session the group membership stabilized and the dropout phenomenon was no longer in evidence. At that point the most outstanding feature was the cohesiveness of the groups.

During the initial 12 sessions considerable stress was placed on the patients to deal with such issues as belonging, acceptance, and inclusion. Every group went through an initial period of turmoil in which the hostility among group members and the therapists led to the expulsion or sacrifice of a few group members. This process of expulsion or sacrifice helped each [redacted] become a more cohesive unit in which a sense [redacted]ess or "we-ness" was acknowledged. The [redacted]for group cohesiveness helped each group [redacted]ndaries and identity as a group. As each [redacted]ned its potential membership and decided [redacted] be included, the group established its spe[redacted]s a separate, independent entity that was not [redacted]and dependent. Once a sense of integrity was [redacted]ed by the members who had some control over group membership, the boundaries [redacted] were firmly established and therapy could [redacted]

Although some groups may develop a [redacted] cohesiveness without the need to sacrifice [redacted] is suggested that for many groups the [redacted] may be basic to the establishment of [redacted] ness. Indeed, patients previously labeled [redacted] may actually serve an important function [redacted] expression of group hostility.

Instead of trying to systematically prev[redacted] the dropout phenomenon, we ought to be [redacted] ceptualize the more positive aspects of it [redacted] pist who confronts a patient's initial res[redacted] tending the early meetings may, in addition [redacted] his or her role as therapist, in some case[redacted] pating in a ritual of group expulsion and [redacted] acting out an important group function [redacted] therapist's reported fear of the group's d[redacted] ing the early dropout period may, in fact [redacted] flect the therapist's anxiety and guilt o[redacted] hostility toward the so-called dropout p[redacted]

These findings suggest that the 30% [redacted] student therapist-led group therapy m[redacted] ly preventable. The so-called dropouts m[redacted] the victims of group forces of hostility a[redacted] ness that we are only now beginning to [redacted] order to establish group boundaries, [redacted] ness," loyalties, and a sense of belong[redacted] araderie, some groups may have to ritu[redacted] one or two members. Unless this pa[redacted] complished, there is probably little hope [redacted] sense of cohesiveness will be establish[redacted] group members.

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Families and Hospitals: Collusion or Cooperation?

BY HENRY T. HARBIN, M.D.

The author describes the therapeutic problems that can arise on a family-oriented psychiatric inpatient service. The stress emerging from the therapeutic demands on and changes in the internal structure of the family may cause the family to seek to reestablish its internal equilibrium by changing the hospital staff and structure at the ward level, department level, and the hospital administration level. These transactions between the hospital and families, which are complex, are extremely important to recognize because they can undermine the therapeutic process.

AS WARD ADMINISTRATOR of a psychiatric inpatient unit, one of several in a psychiatric department that is part of a large university teaching hospital, I have developed a family-oriented inpatient service. This ward has managed to involve over 90% of the families of inpatients in the hospital treatment program using a variety of methods. This intensive involvement of families represents a new dimension for psychiatric hospitals and has consequently produced unique therapeutic problems. The details of how the inpatient staff attempts to change the family have been presented elsewhere.[1] In this paper the focus will be on how the family attempts to change the hospital staff and structure at the ward level, the department level, and the hospital administration level.

A review of the pertinent literature reveals a great deal of interesting work that unravels the intricate structure and function of psychiatric wards and mental hospitals (1-4). Most of these studies concluded that the hospital has a powerful influence on the behavior of inpatients. However, they did not focus to any significant degree on the interactions between the families of inpatients and the psychiatric staff. A few papers have discussed some of the transactions between families and psychiatric staff (5, 6). However, I am unaware of any literature that primarily focuses on the different mechanisms that families use to change the structure of hospital organizations.

In the hospital being discussed the clinical team with direct therapeutic responsibility for the family consists of the psychiatric resident (the team leader), members of the nursing staff, and the social worker. These people operate within a subsystem, i.e., the psychiatric ward of the hospital organization. The ward subsystem is constantly interacting with the other components of the hospital, and they are naturally influenced by any changes in the larger hospital complex. In the ward described here, the therapist (the psychiatric resident) of the identified patient is also the family therapist.

The family of a psychiatric patient entering the hospital is often in a crisis emerging from a chronic conflictive situation. Their usual expectation is that the hospital will take charge of and cure their sick member. They expect that this will be done with minimal effort on their part. When the hospital staff responds by placing certain therapeutic demands on the family, a unique and often stressful situation can arise. In addition, when the patient begins to change, it necessarily upsets the status quo of the family. The family must change to deal with these new influences.

As the family goes through these necessary changes, it may use a variety of mechanisms aimed at reestablishing a stable equilibrium. Some family members wish to prevent any modification of their internal structure, while others may hope to decrease the speed of the change process since they may fear consequent fragmentation. A mechanism frequently used is a maneuver by one or more family members aimed at influencing the behavior or attitudes of the therapist. As the therapist attempts to alter the patient's behavior by changing his or her most important interpersonal context (the family), the family will attempt to alter the context in which the therapist operates. This can mean the psychiatric ward, the department within which the ward exists, the overall hospital administration, or the sociopolitical agencies that control the hospital. One of the family's underlying h[illegible] that the therapist will withdraw his or her pre[illegible] change or modify the therapy for either t[illegible] patient or the family in such a way that t[illegible] continue to operate as usual (thereby m[illegible] stable equilibrium).

If the family is successful in significantly i[illegible] or gaining control of the treatment team, the [illegible] will find that he is operating in a system that l[illegible]

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[1]Harbin HT: A family-oriented psychiatric inpatient unit (unpublished paper, 1977).

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flexibility and freedom of action. This tends to lead to therapeutic failure because the family is then able to resist making necessary internal modifications of its individual members or of its own structure. Attempts by family members of psychiatric inpatients or by designated agents of inpatients to take control of the treatment process occur in many hospitals, but they seem to have been intensified and hence illuminated in the special ward discussed here. The struggles for control between hospitals and families usually take place covertly and are not often articulated by either group; yet the way in which a hospital staff manages these actions by the family is crucial to the therapeutic outcome.

Many different types of families use these mechanisms in order to avoid or minimize change. However, there are some common characteristics of the families that use these maneuvers most often. They can be categorized as follows: middle and upper socioeconomic class families, parents of adolescents and young adults, and families in which the patient has a fairly dramatic or disturbing problem, e.g., violent outbursts, psychotic and bizarre behavior, and suicidal behavior.

TRANSACTIONS AT THE WARD LEVEL

Families of psychiatric patients have the most contact with the ward staff, most frequently with psychiatric aides and least often with physicians and ward administrators. The most common type of maneuvering for control of the therapeutic process occurs at this level of the hospital organization. Some of the methods that families use include involving nontherapist staff members (e.g., the activity therapist or housekeeping personnel) in family disagreements, attempting to split the nursing staff from the physicians, forming coalitions with the lower administrative echelons (the aides) against the higher administrative echelons (the physicians or nurse chairman), forming coalitions with the higher administrative echelons against the lower echelons, and attempting to undermine the treating physician's authority by going over him to his supervisor, the ward administrator.

If successful, these actions can lead to fragmentation of the treatment team, thereby decreasing its efficiency as a therapeutic agent. The last maneuver is especially common; family members (usually the oldest or most authoritative) often appeal to the ward administrator for special treatment. Resistance to therapy can take many forms, one of which is to attack the credentials and status of the psychiatric resident who is the primary therapist.

> The father of one 19-year-old patient telephoned the ward administrator, apparently to obtain some data about a ward policy. Once his question was answered, he began to complain that he did not really understand the therapist's recommendations to the family. The father commented on the therapist's "foreign accent" and began to suggest that he was not a very competent p[illegible]. some discussion the crucial issue surfaced: [illegible] agreed with the therapist about the need f[illegible] to receive medication, and the father (who [illegible] fession related to medicine) wished to [illegible] over the prescription of medication to his [illegible] hoped to influence the supervisor of the [illegible] to intervene on his behalf in his daughter's [illegible]

Another common tactic is the formation of a negative coalition with the nurse and physician administrator of the unit, the highest echelon of the hospital bureaucracy, in order to undermine the authority of the psychiatric aide, the lowest echelon of the ward [illegible]

> A male attendant had set firm limits [illegible] the therapist) on a male teenage patient [illegible] ing out on the ward. The parents had [illegible] firm limits on their son at home and were [illegible] his complaints that the male attendant [illegible] abusive to him. The patient retracted [illegible] times and at one point said that he [illegible] theless, the parents, who were being [illegible] family therapist (who was also the boy's [illegible] more firm with their son, believed him [illegible] ward administrators fire the aide. The nurse [illegible] physician administrator were in an [illegible] cause they had two roles: that of supervisor [illegible] peutic team and head of the ward administration. [illegible] Their response was twofold: they would [illegible] charges, and if they found no evidence to [illegible] ent's view, they would attempt to limit [illegible] out behavior so that they could continue [illegible] therapy. They did this by trying to uncover [illegible] of the parents' anxiety, which was the [illegible] change the way they handled their son [illegible] necessitate a change in the internal structure [illegible]

Certain organizational characteristics of the administrative structure make it vulnerable to manipulation by the family. One of these is the hierarchical arrangements frequently found in the hospital. Henry (7) has described the hospital as having a particular kind of structure labeled multiple subordination: a type of structure in which personnel lower in the hierarchy have multiple supervisors setting clear-cut lines of authority. Henry points out that this type of structure often leads to chaos because of the contradictory splits in the directives given to subordinates. When [illegible] faced with conflicting messages from [illegible] sors, he or she may relinquish autonomy, [illegible] overly dependent or may withdraw and [illegible] her own structure autonomous from the [illegible] leadership. Indeed, this type of structure [illegible] on the ward level and can sometimes [illegible] deficits in communicational efficiency. [illegible] that this is similar to the kind of structure [illegible] that one observes in disturbed families. [illegible] treatment team usually attempts to establish [illegible] cut generational hierarchy in dysfunctional [illegible] and to prevent the kinds of conflicting [illegible]

two parents may be giving their offspring who is a psychiatric patient. Naturally, the ward staff's ability to change a family will be compromised if its own structure is fragmented and disjointed. A vicious cycle can be set up if the ward staff communicate contradictory messages to a family; it can encourage the family to respond with the kinds of self-protective maneuvers described in this paper, e.g., attempts to further split the ward staff.

TRANSACTIONS AT THE DEPARTMENT LEVEL

The next most frequent step that a family takes in order to decrease the pressure of changing its internal functioning is to go outside the ward level to someone higher in the department bureaucracy in order to have the ward staff alter its policies or therapeutic actions. This usually occurs when the ward staff has been successful in maintaining a cohesive unit that the family was unable to split. The most common object for influence is the next person in the administrative hierarchy, the director of all psychiatric inpatient units. Usually the family will call or see the director because of a seemingly legitimate complaint about the ward's policy, such as the need for the patient to stay longer in the hospital or some financial concern. There is often a covert message reflecting the family's fear of the types of changes that are occurring in their own structure. This can be due to either the direct influence of the family therapy or indirectly to the changes that the patient has undergone that necessitate a change in the family system. Sometimes the inpatient director refers the family back to the ward; at other times he may decide to allow the family to discuss the situation with him. If these moves are successful the family may calm down and continue to deal with their problems in the context of the ward's therapeutic approaches.

Another common tactic is for the family to contact a private psychiatrist affiliated with the department or a faculty member who knows the ward staff and ask him or her to intervene. Weintraub (8) has reported several cases of VIP patients whose private physicians were able to successfully intrude into the therapeutic freedom of the ward staff. The family described in the following vignette proved particularly resourceful in their attempts to mold the hospital bureaucracy.

> The patient was a 22-year-old schizophrenic woman in a symbiotic relationship with her parents. The upper middle class parents were accustomed to manipulating bureaucratic structures. They were initially reluctant to enter family therapy because they felt they were not a part of their daughter's problems; however, they finally agreed to participate. After a few weeks the mother announced that she needed more therapy than she was getting and said that she was engaging a private psychiatrist, who happened to be a senior psychiatrist in the department. This threw everyone including the ward administrator into confusion, since this person was a dominant figure in the administrative hierarchy. As everyone knew, this meant that the mother could potentially have a powerful ally outside the influences of the treatment team. The situation worked moderately well until a struggle ensued at discharge, with the family therapist making certain recommendations and the mother refusing to go along with them, saying that she had the agreement of her private therapist. Naturally, the family therapist (the psychiatric resident) was hesitant to challenge the mother given the chance that she was correct in interpreting her special alliance with the senior psychiatrist. Even though some clarification was eventually reached after the ward administrator contacted this psychiatrist, there was a certain amount of harm done to the family therapy work.

This example illustrates a weakness in the ability of a psychiatry department to remain unified vis-à-vis the family. This vulnerability is partly due to the presence of divisive ideologies among professional staff. In previous studies Strauss and associates (9) have identified three types of ideologies in the psychiatric profession: the psychoanalytic, the sociotherapeutic, and the somatic. They felt that these differences in professional ideologies can lead to considerable conflict and it is out of this arena that a specific treatment strategy emerges. In the above example there was a conflict of ideologies between the psychiatric resident (the professional subordinate in the administrative hierarchy) and the senior psychiatrist (the superior in the administrative hierarchy). The resident was operating from a sociotherapeutic ideology with a family emphasis and the senior psychiatrist from a psychoanalytic ideology, with the result that neither strategy could be carried out in a consistent and thorough manner.

HOSPITAL-LEVEL AND EXTERNAL SOCIOPOLITICAL AGENCIES

Families occasionally attempt to go beyond the ward or department level to the administrator of the general hospital. Others go even further—to such outside systems as the state or city government, the press, or the courts—in order to influence the therapeutic process. The organizational structure of the hospital is complex, and there are certain aspects that make it vulnerable to the actions of a small system such as the family. Smith and Kaluzny (10) have described several competing organizational models that are represented within health care institutions. Most important is the bureaucratic/industrial model and its conflict with the professional model. The conflict between these different types of organizational models leaves the hospital vulnerable to outside influences.

Family members may come to the hospital administrator complaining of the unavailability of a single room, impolite nurses, and so forth. The hospital administrators, who are usually operating under the corporate or industrial model, will probably react to the consumer complaint at face value and may see their responses necessary only in terms of maintenance of the organization. They may ignore what Smith and

Levinson (11) have called the instrumental functions of the hospital, which in this case would be the family psychotherapy. The physician/therapist, operating under the professional model, will stress the need for not allowing the family to act out and avoid changing itself. However, if the family is successful in forming a coalition with the hospital administrator, the professional and therapeutic goals may be minimized and lost.

Some families, after failing in their attempts to change the hospital structure, will go to social agencies that govern the hospital. The family in the following vignette was particularly successful in gaining control of the treatment team's attitudes in a very positive but powerful way.

> The wife was a psychiatric inpatient, and the husband was involved in marital therapy in the inpatient unit. At that time the air conditioning system was broken, and the husband (with some covert urging by the staff) decided to help the ward by trying to get it fixed. He had connections in the state governor's office and indeed managed to get the air conditioning system corrected (something that the ward administrator had been unable to do). The ward staff was extremely grateful for the husband's help. However, they had also become dependent on him because of his intervention, and this had the consequence of subtly limiting their ability to treat him as a patient.

COMMENT

From this example and the others, one can see that the hospital physician at the ward level must be aware of the many ways in which family member... his freedom of action by altering the or... structure of the hospital. These transacti... the hospital and families are complex, t... tremely important to recognize because t... dermine the family and individual psycho...

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Hysteria Split Asunder

BY STEVEN E. HYLER, M.D., AND ROBERT L. SPITZER, M.D.

The authors present the proposed DSM-III *classification of the traditional hysterical disorders, i.e., disorders that suggest physical illness but in which psychological factors are judged to be of importance. The use of the* DSM-III *inclusion and exclusion criteria—physical mechanism explains the symptom, symptoms are linked to psychological factors, symptom initiation is under voluntary control, and there is an obvious recognizable environmental goal—are discussed in the differential diagnosis of somatoform disorder, factitious disorder, malingering, psychological factors affecting physical condition, and undiagnosed physical illness.*

THE PROPOSED draft of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)* presents a new classification for the diagnosis of disorders that suggest physical illness but in which psychological factors are judged to be of importance in the initiation, exacerbation, or maintenance of the disturbance. In *DSM-II* (1) many of these disorders were classified among the neuroses (e.g., hysterical, hypochondriacal, neurasthenic) or psychophysiologic disorders. In *DSM-III* conditions in which psychological factors are judged to be important, whether they are physical disorders, syndromes, or symptoms, are dissected and redefined according to certain fundamental distinctions. It is hoped that this will result in more reliable and valid diagnostic categories, not merely new theoretical explanations.

TRADITIONAL CONCEPT OF HYSTERIA

Traditionally the concept of hysteria included the following four criteria: the disturbance suggested physical illness, there were no demonstrable organic findings that explained the disturbance, there was positive evidence that the disturbance was linked to psychological factors, and the disturbance was not symptomatic of any other mental or physical disorder.

In the classical hysterical disorders an emotional conflict is converted into a physical symptom (hysterical conversion) that is considered a direct expression of the underlying conflict. Such disorders typically involve the voluntary nervous system and the sense organs. Among the most common classical hysterical conversions are paralysis, paresthesia, deafness, and aphonia.

In *DSM-II* the hysterical disorders were differentiated from the psychophysiologic disorders, which involved either a recognized physical disorder with demonstrable organic findings (e.g., bronchial asthma) or a physical symptom (e.g., sighing). In both cases the physical disturbance is caused by emotional factors.

The dichotomy between hysterical and psychophysiologic disorder seems to work well when the presentation is classical; it seems unlikely that anyone would make a mistake in the diagnosis of hysterical blindness or peptic ulcer. However, it is increasingly common to see atypical presentations in which these two categories are not adequate for describing the disturbance. Take the example of the person who seeks hospitalization by producing symptoms of physical illness, e.g., hematuria, by the deliberate ingestion of anticoagulants. It is clear that such a person has a diagnosable physical illness, yet the illness is initiated voluntarily. In the previous classification such a disorder would be forced into the classification of hysterical conversion, yet the nature of the disturbance has little to do with the strict definition of a conversion. Another example is the person who develops back pain as a consequence of an occupational injury and is thus unable to work. Even after the compensation issue is settled the patient continues to complain of pain, yet there is no known disease entity or mechanism that explains the bizarre pain the patient describes. What if it becomes obvious during therapy that the patient's husband is now attending to her "hand and foot"? What is this patient's diagnosis? What about the person who from an early age reports recurrent and multiple somatic complaints for which medical attention is sought but which are apparently not due to any physical disorder?

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TABLE 1
DSM-III Inclusion and Exclusion Criteria Used in the Differential Diagnosis of Symptom(s) Suggesting Physical Illness

Classification	Physical Mechanism Explains the Symptom	Symptoms Are Linked to Psychological Factors	Symptom Initiation Is Under Voluntary Control	Only [illegible]
Somatoform disorders	No	Yes	No	
Factitious disorders	Variable	Yes	Yes	
Malingering	Variable	Variable	Yes	
Psychological factors affecting physical condition	Yes	Yes	No	
Undiagnosed physical illness	Variable	Variable	No	

DSM-III CLASSIFICATION OF "HYSTERICAL" DISORDERS

Table 1 presents the *DSM-III* inclusion and exclusion criteria used in the differential diagnosis of mental disorders when the presenting symptom(s) suggest physical illness.

"Physical mechanism explains the symptom" means that either the symptoms represent an accepted and diagnosable physical disorder or syndrome (e.g., angina pectoris, duodenal ulcer, epilepsy) or that there is a specific pathophysiological mechanism that explains the symptom (e.g., vasovagal reflex for fainting, muscle tension for tension headache, muscle spasm for backache).

"Symptoms are linked to psychological factors" indicates that the clinician believes that there is a recognizable temporal relationship between the presence of specific psychological factors or an emotional conflict and the onset or regular occurrence of the physical symptom. Often the individual may not be aware of the role of psychological factors. An example is the individual who becomes nauseated and vomits regularly prior to sexual intercourse.

"Symptom initiation is under voluntary control" means that either the individual simulates the symptom, e.g., feigns the typical pain of a myocardial infarction, or initiates the process that results in the physical symptom, e.g., takes crystalline sodium warfarin (Coumadin), which leads to hematuria. The fact that the individual may claim that he or she cannot refrain from engaging in the behavior that initiates the physical symptom does not rule out its voluntary production. Although the individual may deny the voluntary production when confronted with evidence of such behavior, the clinician is justified in assuming voluntary production because of the nature of the presentation. The accidental production of symptoms is usually ruled out by the repeated occurrence of the symptoms.

"Obvious recognizable environmental goal" indicates that the examiner must make the determination as to whether the symptom production is in pursuit of a goal that is obviously recognizable, using a knowledge of the environmental circumstances rather than the individual's psychology. Examples of such goals include monetary compensation, evasion of justice, and avoidance of conscription. These are obvious and recognizable goals to any layman. In di[illegible] that only become apparent with a [illegible] individual's psychology include expr[illegible] rental substitutes, satisfying unmet d[illegible] and playing the role of a patient to s[illegible] impulses. These goals are understa[illegible] pretable only by the trained clinician.

SOMATOFORM DISORDERS

The somatoform disorders include [illegible] orders that have been classified as hy[illegible] in this category is somatization (Br[illegible] which is a chronic but fluctuating dis[illegible] early in life and is characterized by rec[illegible] tiple somatic complaints for which m[illegible] sought but that apparently are not du[illegible] disorder. The complaints refer to m[illegible] tems, e.g., abdominal pain with vom[illegible] aches, joint pain, and menstrual abn[illegible] tization disorder has been removed f[illegible] of hysterical personality because it ha[illegible] individuals with a lifelong pattern of s[illegible] for multiple somatic complaints d[illegible] have a hysterical, i.e., histrionic, per[illegible] versa (2, 3). When these two disorder[illegible] ommended that both diagnoses be m[illegible]

Conversion disorder has also been [illegible] the category of hysterical personality [illegible] been noted that persons with other p[illegible] orders, or with psychoses, may unde[illegible] tions manifest conversion symptoms [illegible] version disorder the predominant fe[illegible] alteration in functioning that suggest[illegible] but which is actually a direct expres[illegible] logical conflict that is not under volu[illegible] not explainable on the basis of a kno[illegible] mechanism. The most common conver[illegible] are those that suggest disorder of the v[illegible] vous system or the sense organs, e.g. pa[illegible] nia, and seizures. This disorder shoul[illegible] nosed when the clinical picture is part o[illegible] vasive somatization disorder or when t[illegible] are limited to sexual dysfunction, which is [illegible] rately. This disorder replaces the *DSM-I* [illegible] hysterical neurosis, conversion type.

The third disorder in this group is p[illegible]

predominant feature of which is the complaint of pain not associated with any physical condition or explained by any pathophysiologic mechanism. Psychological factors are judged to be important in the initiation, exacerbation, or maintenance of the pain. The pain follows no neurological distribution and may present a bizarre pattern. This disorder has been removed from the category of hysterical conversions, in which pain is the presenting symptom. Because this is a new diagnostic category, its validity has not yet been established. It is recommended that extreme care be used in making this diagnosis. Many persons who have been described as having compensation neurosis may now be classified in this category. An example of this is the individual who, even after a monetary compensation issue has been settled, continues to experience and complain of pain when there is no evidence of a physical disorder, syndrome, or mechanism to account for the pain. Persons whose complaints of pain disappear soon after settlement of a compensation issue may best be classified as malingerers.

The fourth disorder in this group is hypochondriasis (6). Its predominant feature is the individual's fear that he or she may have a disease. This fear is refractory to simple reassurance, and the person often goes "doctor shopping" until he finds a physician who can make the appropriate diagnosis. This disorder is differentiated from somatization disorder in that the individual's fear that he may have a disease, rather than multiple somatic complaints, is predominant. In hypochondriasis the complaints are usually more circumscribed. This category may be used for patients who were previously described as having cardiac neurosis, and who have a preoccupation with cardiac symptoms, or cancer phobia, and who may interpret minor aches and pains as evidence of neoplasm.

FACTITIOUS DISORDERS

An entirely new diagnostic category in *DSM-III* is factitious disorder, which was included to fill the large middle ground between the hysterical disorders and malingering. "Factitious" means not real, genuine, or natural and describes that which is produced by art or design. Factitious disorders are therefore characterized by physical or psychological symptoms that are voluntarily initiated by the patient. This differentiates the factitious from the somatoform disorders. The factitious disorders are also differentiated from malingering. In cases of malingering the patient also produces symptoms voluntarily but they are for a goal that is obviously recognizable with a knowledge of the environmental circumstances. In factitious disorders there is often no apparent goal other than to assume the role of a patient. In time it may become apparent that such a patient is attempting to gain mastery over a previously experienced psychic trauma or is acting out a sadomasochistic relationship with physicians who are regarded as parental figures, but these are considered individual goals that are related to the patient's psychology. In addition, whereas under certain circumstances an act of malingering may be considered adaptive, as in the hands of the enemy during a war, a diagnosis of factitious disorder always implies severe pathology that is usually indicative of an underlying personality disturbance. The factitious disorders are subdivided depending upon whether the symptoms presented are psychological or physical.

In the subcategory of factitious illness with psychological symptoms the essential feature is the voluntary production of symptoms suggestive of a mental disorder, in the absence of an obvious recognizable goal. The mental symptoms are usually a representation of the person's conception of what a mental disorder should be rather than being indicative of any specific diagnostic category. This disorder subsumes former diagnoses such as hysterical Ganser syndrome (7), pseudopsychosis, and some cases of pseudodementia (8). The individual with this disorder is often recognized by the pansymptomatic complex of mental symptoms that is presented, e.g., memory loss, visual and auditory hallucinations, approximate answers, and not being able to recognize the color red. This disorder is very difficult to differentiate from malingering. Often a combination of recognizable environmental goals coexists with recognizable psychological goals. An example of this is the patient with a schizophrenic disorder who upon learning that he is to be discharged presents a bewildering array of new and bizarre symptoms. If it is thought that psychological factors (such as a dependency on the hospital) rather than environmental factors (such as an absence of a place to live) are instrumental in the production of the new symptoms, then the symptoms should be considered factitious and superimposed on the original schizophrenic disorder. The validity of this disorder when not associated with any other mental disorder remains to be established.

The next subtype is chronic factitious illness with physical symptoms (9), often referred to as the Munchausen syndrome (10). The essential feature is the patient's plausible presentation of factitious physical illness of such a degree that he is able to obtain and sustain multiple successive hospitalizations. The symptoms described are limited only by the patient's medical knowledge, sophistication, and imagination. Other synonyms have included hospital addiction syndrome (11), hospital hoboes (12), and peregrinating problem patients (13).

Associated features of this disorder include pseudologia fantastica, an extensive knowledge of hospital routines (which improves with each new hospitalization), analgesic abuse, and "gridiron abdomen" from multiple surgical procedures. This disorder is thought to be extremely incapacitating and is apparently refractory to any type of therapy. This category was created to facilitate the recognition of such patients, who clearly have serious psychopathology not classifiable in *DSM-II*. In the past such patients have

been diagnosed as having schizophrenia, antisocial personality, hysterical personality, and hysterical neurosis, conversion type. Should these patients meet the criteria for any other mental disorder, both diagnoses should be made.

Other factitious illness with physical symptoms is a residual category for individuals with factitious physical symptoms who do not meet the criteria of the better-studied, more pervasive disorder mentioned above. The patient may not seek hospitalization but may appear at an outpatient department with a dermatitis artefacta or a voluntary dislocation of the shoulder.

It should be emphasized that the presence of a factitious disorder with physical symptoms does not necessarily imply the absence of organic pathology. Many times the individual is very ill indeed, and almost any of the self-inflicted traumas results in true physical pathology. It is the initiation of symptom production that is factitious.

CATEGORY 316

Category 316, psychological factors affecting physical condition, is also a new category in *DSM-III*. It subsumes the *DSM-II* category of psychophysiologic disorders but is broader in scope. Here the clinician notes those physical conditions for which he believes psychological factors are important in the initiation, exacerbation, or maintenance of the condition. The clinician is further requested to specify the certainty that he believes the psychological factors are contributory as either probable or definite. The physical condition is coded separately on axis III. An example of the use of this diagnostic category would be to indicate the suspected importance of psychological factors in such diverse disorders as bronchial asthma, angina pectoris, tension headache, syncope, or nausea and vomiting. A more detailed discussion of this category is available elsewhere (14).

UNDIAGNOSED PHYSICAL ILLNESS

Given the present state of the art of medical diagnosis and limitations in objective diagnostic laboratory tests, it is a common occurrence that a person may manifest certain physical symptoms for which no diagnosis of either physical or mental disorder can be made. The physical complaints should not be classified in any of the aforementioned disorders unless the newly established criteria are met. For example, a person may have weakness, tremors, and paresthesias. If all diagnostic tests are negative or equivocal, if it is thought that the symptoms are not under voluntary control, if there are no obvious recognizable environmental goals, and the examiner does not have positive evidence that the symptoms are linked to psychological factors, the possibility of an undiagnosed physical illness should be considered. A number of [illegible] orders, e.g., cortical blindness or extrapy[illegible] ease, may be mistaken for hysteria (15). [illegible] been shown that when a number of patie[illegible] been given diagnoses of hysteria were fo[illegible] time, they developed full-blown medical c[illegible] ulcerative colitis or multiple sclerosis (16).

Mention should be made of two othe ca[illegible] have in the past been considered as hyster[illegible] physical symptoms may not have been [illegible]

BRIEF REACTIVE PSYCHOSIS

In brief reactive psychosis the essenti[illegible] a florid psychosis of at least a few hou[illegible] lasting not more than one week, with [illegible] immediately after severe emotional s[illegible] tually terminating in complete recov[illegible] psychotic state. This disorder is des[illegible] cause it has been informally referred to [illegible] psychosis, and some patients given the [illegible] nosis of acute schizophrenic episode w[illegible] classified here. Despite its occasional [illegible] similarity to a schizophrenic disorder (e[illegible] hallucinations), the distinction is impo[illegible] the course and prognosis are different

HISTRIONIC PERSONALITY DISORDER (HYSTERICAL PERSONALITY)

Here the essential feature is a patter[illegible] behavior that is overly reactive, intense[illegible] and perceived by others as shallow, sup[illegible] sincere, with characteristic disturbed [illegible] relationships. This personality disorde[illegible] trionic" rather than "hysterical" to em[illegible] trionic mode of presentation and to remov[illegible] tive connotation to women (4, 5) (and any[illegible] to the uterus). The occurrence of this diso[illegible] is well recognized (17).

DISCUSSION

The differentiation between "real" [illegible] terical" symptoms has been among the [illegible] diagnostic tasks for physicians. Not on[illegible] been the problem of establishing what s[illegible] toms are without physical basis, but th[illegible] been the problem of how to classify the [illegible] order, its causes, the course, the prog[illegible] appropriate therapy. The purpose of recla[illegible] terical disorders was to maximize the [illegible] the diagnosis for the outcome and select [illegible] priate therapy. It is expected that the [illegible] individual with a somatoform disorder wi[illegible] at the psychological component, which is [illegible] etiologic, rather than at the overt physi[illegible]

that are presented. In factitious disorders it is expected that the liaison psychiatrist will be able to recognize the etiologic psychological factors that are often hidden by the dramatic presentation of acute physical illness. Psychiatrists should attempt to educate physicians of other disciplines in the recognition of such patients so as to avoid needless, costly, and dangerous somatic procedures and operations. Perhaps in the future an effective type of psychiatric therapy will be developed for this condition. A diagnosis of category 316 will usually require the cooperation of the psychiatrist and the physician who is treating the physical condition and appropriate coordination of therapy directed at the physical and psychological components.

In creating new diagnostic categories for hysteria in *DSM-III* it was decided that all that is not genuine is not the same. It is hoped that the distinctions will prove useful.

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TOPICAL PAPERS: Psychiatric Residency and Clerkship

Group Therapy Training in Psychiatric Residency Programs
A National Survey

BY EDWARD L. PINNEY, JR., M.D., STEPHEN H. WELLS, PH.D., AND BERNARD FISHER, PH.D.

The authors used a questionnaire sent to the directors of the 256 approved American psychiatric residency programs to examine the availability, content, and orientations of group therapy training. They found that 78% of the responding programs offered group therapy training, a substantial increase from earlier estimates; that an approach incorporating didactic courses, observation and leadership of groups, supervision, and participation in an experiential group was gaining increased acceptance; that most of the programs provided a variety of arrangements for supervision of group training; that the theoretical orientations mirrored the field of group therapy itself; and, finally, that associated disciplines were gaining increasing acceptance.

INCREASING RELIANCE on group treatment approaches has been characteristic of the psychiatric scene over the past three decades, closely paralleling the development of the small group movement in the country at large. Although considerable interest has been generated by the profusion of what many regard as new, effective, and economical techniques, the number of warnings about leadership competency has been growing. In a 1970 task force report (1), the American Psychiatric Association decried the "extraordinary heterogeneity" among group leaders, therapists as well as others, in levels of competence, skills, discipline, goals, and motivations. If this statement has substantial merit, examination of the training experiences of professionals who practice as group therapists is a matter of some urgency.

Although psychiatry has not generally been in the leading force among the mental health disciplines in the advancement of group therapy, psychiatric residency programs patently represent the single most significant training source of practitioners in this area. Relatively meager data are available, however, on the nature and extent of group therapy training offered in residency programs. Based on a 1950 questionnaire survey of residents at 14 centers, Ward (2) reported that only one-third of trainees in his sample had the opportunity to participate in or practice group therapy. When Ward and Kaplan (3) repeated the survey in 1961 for purposes of historical comparison, exposure to group therapy training was found to have increased to 72% of the residents at the psychiatric centers. Both survey reports strikingly underscored residents' enthusiasm for the modality: group therapy emerged in each study as "most valued" by those who had experienced it and as "most desired" by those who had not been afforded the opportunity.

In 1971 Sadock and Kaplan (4) estimated that group therapy training was provided to residents in 60% of the existing programs. Although this figure may have been somewhat on the low side, the factors they cite to account for the relative paucity of opportunities observed in 1971 are quite accurate: a dearth of qualified training faculty, time constraints of the residency schedule, resistance to a new and unfamiliar

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The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

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and prejudice against a "secondary" or "inferior" technique.

The present study sought to provide a comprehensive and contemporary picture of group therapy training in approved residency programs. To that end, a national survey was performed in which the availability, content, and orientations of group therapy training in these programs were examined.

METHOD

A survey questionnaire was mailed to the program directors (or chiefs of service) of the 256 approved psychiatry residencies listed in the 1975–1976 *Directory of Approved Residencies* of the Liaison Committee on Graduate Medical Education of the American Medical Association (5). These programs included 196 residencies affiliated with university teaching hospitals and 60 unaffiliated residencies.

The questionnaire was a brief survey form that requested the following information[1]: 1) availability of training in group psychotherapy for psychiatric residents, 2) postgraduate year(s) during which group psychotherapy training was available, 3) training for members of other professional groups, 4) the types and relative importance of training techniques used, 5) whether residents worked alone with groups or with a cotherapist, assistant therapist, or observer-recorder and the profession of the cotherapist, assistant therapist, or observer-recorder, 6) the theoretical orientation of group psychotherapy training, and 7) the type and focus of supervision of trainees.

A total of 172 of the 256 mailed questionnaires were returned, giving a response rate of 59.3%. Responses were received from 138 of the affiliated and 34 of the unaffiliated residency programs.

RESULTS AND DISCUSSION

Resident Access to Group Therapy Training

Residents in 135 of the 172 programs reporting (78.5%) had access to group therapy training during at least one of their postgraduate years (see table 1). Only 37 of the programs (21.5%) offered no such experience to their trainees. Although the results of the earlier surveys discussed above (2, 3) are not directly comparable to the results of this study, there would appear to be little doubt that the acceptance of group therapy in residency training programs has increased.

Even more striking than our finding that 4 of the 5 programs we surveyed included group therapy training are the findings with regard to duration and extensiveness of the experience offered. The most prevalent arrangement, representing more than one-third of the cases, was one in which the resident participated during postgraduate years 2 to 4 inclusive. Approximately 62.2% of the programs (N=107) offered group therapy training over 3 or 4 postgraduate years, and in only 16 (9.3%) of the programs was the resident's introduction to the modality delayed until postgraduate year 3.

This development is of great importance for group therapy training. It has long been recognized that starting group therapy training as early as postgraduate year 1 would enhance the trainee's overall level of interpersonal awareness in such a way as synergistically to augment functioning in other therapeutic modalities. Early exposure, as Sadock and Kaplan (4) noted, also serves to forestall the development of prejudices toward group therapy on the part of the resident, since he or she comes to regard it as an accepted component of residency training rather than as a secondary and auxiliary mode of treatment.

An efficacious personal impact on the resident may also be observed as a consequence of experience in group therapy and in related trainee groups and supervisory triads or groups. All these can assist the resident in gaining greater confidence and effectiveness in

TABLE 1
Postgraduate Years During Which 172 Psychiatric Residency Programs Offered Group Psychotherapy Training

Item	Programs	
	Number	Percent
Postgraduate year		
1 and 2 inclusive	2	1.2
1 to 3 inclusive	2	1.2
1 to 4 inclusive	46	26.7
2 and 3 inclusive	10	5.8
2 to 4 inclusive	59	34.3
3	6	3.5
3 and 4 inclusive	10	5.8
Total with group therapy training	135	78.5
Total with no group therapy training	37	21.5

TABLE 2
Teaching Modalities Used in 135 Group Psychotherapy Training Programs for Psychiatric Residents*

Modality	Programs	
	Number	Percent
1. Didactic seminars or lectures	130	96.3
2. Live observation of groups	98	70.4
3. Videotape observations	76	56.3
4. Group psychotherapy simulations	46	34.1
5. Experiential groups	105	77.7
6. Combinations	126	93.3
1, 2, and 5	58	43.0
1 and 5	30	22.2
1 and 2	19	14.1
1, 2, 3, and 5	8	5.9
2 and 5	5	3.7
Other combinations	6	4.4

*All of the 135 residency programs offering group therapy training included supervision as a program component. Data on the nature of the supervision offered are presented separately in table 3.

[1]A copy of the questionnaire is available on request from Dr. Wells.

the group settings and interpersonal situations that are inevitably part of professional life.

Group Training Modalities Employed

Table 2 presents the survey findings on modalities used for the teaching of group therapy in the residency programs. It is clear that the use of multiple training modalities has gained ascendency; 126 of the programs (93.3%) used more than one training technique. The primary training pattern, used by nearly one-half of the programs, included didactic seminars or lectures, live observation of groups, and participation in an experiential group (in addition to some form of supervision, which every program incorporated). It is not surprising that supervision was a feature of each of the programs and that all but 5 included a didactic seminar or lecture component. What is striking is the extent to which program directors recognized the need for participation in experiential groups. More than three-quarters of the programs provided groups in which the resident participated as a member rather than a leader.

A comparison of the survey findings with the training model proposed by the American Group Psychotherapy Association (AGPA) in 1970 (6) is instructive. The AGPA guidelines call for didactic courses and case seminars, observation and leadership of at least three therapy groups, supervision, and participation as a member in a group psychotherapy or group training experience. This model is finding increasing acceptance in residency training programs.

Patterns of Supervision

As previously reported, every residency program offering training in group therapy provided supervision in some form to its trainees. The possible supervision arrangements examined in this survey included individual or dyadic (one supervisor with one group therapist), triadic (one supervisor with two trainees who worked as cotherapists), and group (one supervisor with several group therapists) supervision. As table 3 indicates, more than 80% or 110 of the programs had flexible arrangements in which two or more of these variants could be employed. Within this group, the predominant model was to permit the use of all three supervisory patterns.

The most conventional arrangement, derived from earlier experience in the supervision of individual psychotherapy, is the dyadic. In the view of some (7), however, the triadic pattern has greater efficacy for group therapy. The triadic supervisory process permits the examination not only of events within the group but also of the relationship between the cotherapists, both as it manifests itself within the group and as it emerges in the context of supervision. We found that the triadic was the most often employed of the three models, as shown in table 3.

Group supervision provides still further possibilities for training. The several trainees themselves constitute a group, the dynamics of which can be explored to elucidate aspects of the residents' group and interpersonal experiences, particularly with regard to countertransference. There is a likelihood, in the view of Marohn (8), that under such conditions "the themes of the formal therapy with patients" will be "similar to the themes of subsequent supervisory meetings." We found that group supervision was employed by fewer centers than either of the other patterns, a phenomenon probably due, at least in part, to the highly challenging nature of such work for the supervisor.

TABLE 3
Patterns of Supervision in 135 Group Psychotherapy Programs for Psychiatric Residents

Type of Supervision	Number
1. Individual	82
2. Triadic (cotherapists as a unit)	96
3. Group	75
4. Combinations	110
1, 2, and 3	43
1 and 2	29
2 and 3	21
Other combinations	17

TABLE 4
Theoretical Orientations of 135 Group Psychotherapy Programs for Psychiatric Residents

Orientation	Number
1. Psychoanalytic	86
2. Interpersonal	89
3. Group process	118
4. Group dynamics	108
5. Gestalt	25
6. Transactional Analysis	22
7. Behavioral	22
8. Other	8
9. Combinations	122
1, 2, 3, and 4	44
2, 3, and 4	32
1, 3, and 4	26
3, 4, and 5	11
Other combinations	9

Theoretical Orientations of Group Training

Table 4 presents data on the theoretical orientations of the 135 residency group therapy training programs. The compiled findings are striking in the extent of pluralism portrayed. Fully 90% of the programs (N=122) described their orientations as a combination of two or more theoretical viewpoints. A group process orientation was the single most frequently cited theoretical stance, cited in 118 (87.4%) of the programs. The most frequent combination of orientations, presented by more than one-third of the responding programs, incorporated psychoanalytic, interpersonal, group process, and group dynamics theories.

It is not inconceivable that the prevalent

bined orientations indicates that many of the programs used a well conceived and carefully elaborated combination of approaches. A more likely conclusion is that the survey findings precisely mirror the situation in the group therapy field itself. Throughout its history, as Peck (9) observed, there has been a "plaintive and continuing call for a more definite conceptual framework for group psychotherapy." That the call has not yet been answered may reflect deficiencies in the work done. On the other hand, it may express certain special difficulties inherent in the task of bringing psychodynamic, group-focused, and social change perspectives to bear in the developing role of the group therapist.

Training of Associated Disciplines

Given the significant role of such associated disciplines as psychology and social work in the development of group therapy, it is interesting to examine the extent to which residents have contact with other professionals in the course of their group training. Based on examination of the survey data presented in table 5, it is clear that such contact is more extensive than might have been anticipated. Fully 82% or 111 of the programs offering group therapy training to their residents permitted the participation of trainees from at least one other discipline. Clinical psychologists and social workers were, not unexpectedly, the most frequently included professional groups, each being represented in more than 3 of 5 reporting programs. Although no other group achieved such "penetration," a broad range of disciplines found representation.

Findings on the arrangements under which psychiatric trainees function as cotherapists in the group therapy programs show that the predominant pattern, reported for 69.6% or 94 of the centers, was one in which residents worked with each other; however, in more than half of the programs they also trained as cotherapists with psychologists, social workers, and psychiatric nurses.

Ward and Rickels (3) reported a striking decrease in residents' contacts with associated disciplines between their 1950 survey and the one undertaken in 1961. Two-thirds of the residents in 1961 had little or no such experience. Quite clearly a very different situation exists for residents who participated in group therapy training in 1977.

TABLE 5
Inclusion of Other Professionals in 135 Group Psychotherapy Training Programs for Psychiatric Residents

Item	Programs	
	Number	Percent
Professional group		
Clinical psychologists	89	65.9
Social workers	83	61.5
Psychiatric nurses	69	51.1
Occupational and related therapists	37	27.4
Paraprofessionals	29	21.5
Others	8	5.9
Total programs including other professionals	111	82.2
Total programs with no others included	24	17.8

There is considerable sentiment within psychiatry favoring the inclusion of other mental health professionals as coequal faculty members and trainees in medical centers. Group therapy training programs appear to embody this sentiment, operating as distinctively open environments within the residency context. The very diversity, complexity, and richness of group therapy itself apparently serves to promote recognition of the need for varied perspectives in its teaching.

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Consultation-Liaison Versus Other Psychiatry Clerkships: A Comparison of Learning Outcomes and Student Reactions

BY WILLIAM W. WEDDINGTON, JR., M.D., FREDERICK R. HINE, M.D., JEFFREY L. HOUPT, M.D., AND CAROLE S. ORLEANS, PH.D.

The authors present the results of a one-year study showing equivalent mastery of basic psychiatric knowledge and skills and equally favorable student reactions after psychiatry clerkships on a consultation-liaison service and on other more traditional psychiatry services. They recommend further research highlighting the unique contributions of consultation-liaison clerkship training to knowledge, skills, and attitudes for holistic primary care.

RECENT DEVELOPMENTS have formalized the demand that all physicians be capable of providing holistic care for their patients—care that includes competence in the behavioral sciences and clinical psychiatry (1). Developments at the national and local levels have combined to give force to that demand. They include the *Report of the Citizens' Commission on Graduate Medical Education* (2) detailing the required areas of competence for primary care physicians, the priorities of the National Institute of Mental Health Psychiatry Education Branch favoring support for educational programs that demonstrate collaboration between psychiatry and general medicine (3), and local medical school policies fostering productive relationships between psychiatry and other departments.

As one outgrowth of these developments, increasing numbers of medical students are receiving training in consultation-liaison psychiatry (4). In addition, consultation-liaison services have been recommended as favored settings for psychiatry clerkship training (5).

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Because they ensure direct contact with patients whose needs and symptoms closely approximate those which most students will encounter in their own patients, it has been argued that clerkships on a consultation-liaison service will facilitate the learning of psychiatric knowledge and skills and thereby contribute to more holistic future patient care. Moreover, since core experiences on consultation-liaison services involve primarily surgical, medical, and gynecological patients, whereas psychiatric clerkship training has traditionally occurred in psychiatric settings where patients are identified as psychiatric patients under psychiatric supervision, psychiatric educators might appropriately wonder whether consultation-liaison clerkships really provide adequate basic knowledge and skills.

To address this issue the present study compared students clerking on a consultation-liaison service with students clerking on more traditional inpatient and outpatient psychiatry services. Two questions were asked: Do students on a consultation-liaison clerkship acquire sufficient psychiatric knowledge and skills, and do they rate their clinical training experiences as favorably as students on traditional psychiatry clerkships?

METHOD

The subjects consisted of 106 second-year Duke University Medical School students who had previously completed a first-year psychiatry-behavioral science course and were enrolled in the required psychiatry clerkship during the 1975–1976 academic year. (Under the Duke curriculum, students complete required clinical clerkships during their third year.) Fourteen students completing their clinical clerkships on the psychiatry consultation-liaison service were compared with 92 students who clerked on traditional inpatient and outpatient psychiatry services.

Postclerkship knowledge of traditional psychiatric concepts and techniques was determined from special administrations of the National Board of Medical Examiners' part II psychiatry examination. Findings on signed course-evaluation questionnaires provided four measures of student reaction to clerkship training and instruction. Students rated their "overall learning experience on the service" and "adequacy of supervision and teaching" on

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and senior staff" and the contributions of a Multidimensional Psychiatric Diagnosis and Treatment (MPDT) program (described below) to improving their 3) "interactional skills and interviewing techniques," and 4) "understanding of psychiatric concepts and techniques as they apply to nonpsychiatric patients." Student ratings, which remained sealed until after grades were determined, were obtained using 5-point rating scales with the following anchor statements: 1="poor, not worth the time"; 2="unsatisfactory, mediocre"; 3="fair, useful"; 4="good"; and 5="excellent, outstanding."

Because the clerkship comparison groups were constituted through self-selection rather than random assignment, preclerkship measures were used to control for selection factors that might affect training outcomes (6). Students' numerical grades on a written take-home examination administered after the behavioral science course in the previous year constituted a single measure of preclerkship psychiatric knowledge.[1] Ratings of the first-year course, which also remained sealed until after first-year grades had been submitted, were obtained using the same 5-point rating scales employed for clerkship ratings. A measure of student reaction to preclerkship psychiatry training was obtained by averaging student ratings of the adequacy of the course to "introduce concepts and techniques of interviewing," "communicate behavioral science concepts and their importance to medicine," and "demonstrate the approach of the behavioral scientist." Ratings of the "general teaching effectiveness of small group instructors" provided a measure of student reaction to preclerkship psychiatry instruction.

Description of Psychiatry Clerkship and Human Behavior Courses

The second-year clinical psychiatry clerkship is one of five 8-week, full-time required clerkships. Students request assignment to one of the following six basic services: Duke Hospital Consultation-Liaison Service, Duke Hospital Inpatient Service, Durham Veterans Administration Hospital Inpatient Service, John Umstead (State) Hospital Inpatient Service, Duke Hospital Psychiatric Outpatient Clinic, and Durham Child Guidance Clinic. Assignments are based on students' requests except when a particular service is oversubscribed; in those cases students are usually assigned second- or third-choice services.

Students assume clinical responsibilities under the primary supervision of a resident and under more general supervision from senior faculty. They work up and follow patients according to the program used on their service and attend work rounds and case conferences with residents. Students on the consultation-liaison service work primarily with surgical, medical, and obstetric-gynecological inpatients, while students on other psychiatry services work primarily with identified psychiatric inpatients or outpatients. All students on noninpatient units (consultation-liaison, adult and child psychiatric outpatient) evaluate inpatients one half-day per week at John Umstead Hospital to gain experience with more severe forms of psychopathology.

In addition to rotations through basic clinical services, all clerkship students participate in two common core activities: didactic lecture-seminars on such topics as psychiatric diagnosis and treatment modalities, and the MPDT program, designed to increase students' understanding of the value and importance of psychiatric concepts and techniques in medicine generally. The MPDT program, described in detail elsewhere (7), begins with lecture-seminars in which psychiatric diagnoses are presented as dimensions that may be present in severe degrees, as in psychiatric illness, or in mild or moderate degrees, as may be found in the population generally and medical-surgical patients in particular. After this introduction students interview patients randomly selected from nonpsychiatric hospital services, tape these interviews, and receive detailed one-to-one supervision from members of the psychiatry staff.

Students also share a common preparation for clerkship training. All students take a first-year psychiatry behavioral science course that combines 60 hours of lectures on behavioral neurobiology, medical sociology, human development, psychodynamics, and communication processes with small group sessions directed at extending lecture material and providing experiential training in interview techniques.

TABLE 1
Preclerkship Measures of Students' Psychiatric Knowledge and Ratings of First-Year Psychiatric Training and Instruction

Group	Mean Scores		
	Course Exam	Training Rating	Instruction Rating
Consultation-liaison (N=14)	86.57	3.45	4.07
Traditional psychiatry (combined) (N=92)	88.00	3.36	4.01
Duke inpatient (N=34)	88.44	3.29	3.88
VA inpatient (N=32)	88.84	3.44	4.13
State inpatient (N=7)	89.86	3.43	4.00
Child guidance (N=9)	85.89	3.33	4.11
Duke outpatient (N=10)	84.40	3.30	3.95
Intergroup comparison			
F*	.95	.18	.52
t**	.68	.41	.31

*Resulting from one-way analyses of variance on scores for six single clerkship groups, df=5, 100.
**Resulting from comparisons of consultation-liaison with traditional psychiatry group means, df=104.

RESULTS

Means were obtained for all preclerkship and postclerkship measures for the consultation-liaison group,

[1]A copy of the examination with scoring instructions is available from Dr. Hine on request.

TABLE 2
Postclerkship Measures of Students' Psychiatric Knowledge and Ratings of First-Year Psychiatric Training and Instruction

Group	Mean Scores				
	National Board Exam	Training Rating	Instruction Rating	MPDT 1	MI DT
Consultation-liaison(N=14)	557.86	3.86	4.04	3.00	[illegible]
Traditional psychiatry(combined) (N=92)	556.90	4.11	3.99	3.67	[illegible]
Duke inpatient (N=34)	571.32	3.97	3.76	3.74	[illegible]
VA inpatient (N=32)	547.50	4.28	4.09	3.75	[illegible]
State inpatient (N=7)	511.43	4.29	4.14	3.72	[illegible]
Child guidance (N=9)	573.89	4.00	3.94	3.67	[illegible]
Duke outpatient (N=10)	554.50	4.00	4.40	3.20	[illegible]
Intergroup comparison					
F*	.66	1.07	1.63	1.50	[illegible]
t**	.04	1.18	.24	2.25*	[illegible]

* Resulting from one-way analyses of variance on scores for six clerkship groups, df=5, 100.
** Resulting from comparisons of consultation-liaison with traditional psychiatry group means, df=104.
* p<.05, two-tailed t test, df=104.

for each of the five single traditional clerkship groups, and for the larger traditional psychiatry group consisting of all students clerking on the five combined traditional psychiatry services. To determine whether students could be significantly differentiated on the basis of clerkship services, one-way analyses of variance were performed on the scores of the consultation-liaison group and of the five single traditional clerkship groups, yielding a single F value for each measure. To compare students in the consultation-liaison group with students in the larger traditional psychiatry group, t tests were performed on the mean scores of both groups, yielding a single t value for each measure.

Table 1 presents the means and F and t values for preclerkship measures of psychiatric knowledge and student ratings of first-year psychiatry training and instruction. As can be seen from the table, none of the scores of the preclerkship measures were significantly differentiated on the basis of clerkship service (F values<1, df=5, 100, n.s.). Further, separate t tests revealed that students clerking on the consultation-liaison service did not differ significantly from those clerking on the combined traditional psychiatry services (t values<1, df=104, n.s.) with respect to any of the measures obtained before clerkship training.

As can be seen from table 2, the analysis of variance on National Board psychiatry scores, with means of 511.43–573.89, revealed that the type of clerkship service had no significant overall effect on students' scores. Further, the t test revealed that the National Board scores of students clerking on the consultation-liaison service did not differ significantly from those of students clerking on the combined traditional psychiatry services.

As can also be seen from table 2, analyses of variance on ratings of service training, with means of 3.86–4.29, and ratings of service instruction, with means of 3.76–4.40, revealed that the type of clerkship service had no significant overall effect on students' reactions to their clerkship experiences. Further, separate t tests revealed that the ratings of students clerking on the consultation-liaison service did not differ [illegible] from those of students clerking on the combined traditional psychiatry services.

Finally, table 2 shows that the analyses on ratings of MPDT contributions to interviewing skill (MPDT 1) and to understanding the role of psychiatry in medicine (MPDT 2), with means of 3.[illegible]–3.29–4.03, respectively, revealed that [illegible] clerkship service had no significant overall effect on students' reactions to the MPDT program. However, separate t tests revealed that students clerking on the consultation-liaison service rated the MPDT program as contributing significantly less to their interviewing skill (t=2.25, df=104, p<.05) and to their understanding of the role of psychiatry in medicine (t=[illegible], df=104, p<.05) than did students clerking on the combined traditional psychiatry services.

COMMENT

The results of our investigation suggest that students who clerk on a consultation-liaison service receive more traditional psychiatry services and participate in a common core curriculum acquire [illegible] knowledge of basic psychiatric concepts and techniques. In addition, our findings suggest that consultation-liaison clerkship students value psychiatry experiences as highly as do students clerking on other services.

The finding of equivalent learning outcomes extends previous findings (5) showing consultation-liaison clerkship students to perform as well on a comprehensive psychopharmacology exam as students on traditional psychiatry services, by using the nationally standardized National Board of Medical Examiners' psychiatry exam, and by demonstrating no significant preclerkship differences that might affect interpretation of uniform postclerkship knowledge. The present findings are also consistent with those of a recent study (6) showing no significant differences on [illegible]

Board part II exam and objective exams among students clerking on five different psychiatry inpatient services. In this and the previous study (8), it is likely that standardized didactic instruction offered before and during the clerkship contributed to uniform outcomes in formal knowledge.

The present study, unlike a previous study (4), does not show that consultation-liaison clerkship experiences are valued more highly than more traditional clerkships. The unexpected finding that MPDT training was rated as less valuable by consultation-liaison students, who saw primarily nonpsychiatric patients, than by clerkship students, who saw primarily identified psychiatric patients, is probably due to real and perceived overlap in consultation-liaison and MPDT experiences. While the random selection of patients in the MPDT program was designed to demonstrate the relevance of psychiatry to all medical-surgical patients, many patients reviewed on the consultation-liaison service were similar in that they did not have obvious psychiatric illness and would probably not have been referred for psychiatric consultation. In addition, MPDT and consultation-liaison instruction and supervision were similar in focusing on the role of psychiatry in medicine. Against this background it is possible that the unique contributions of the MPDT program, such as detailed process supervision, were less salient for consultation-liaison than for other clerkship students. Moreover, questionnaire items were not specifically designed to bring into sharp focus unique benefits of MPDT over consultation-liaison experiences.

The present study examined only whether consultation-liaison clerkship experiences were as adequate to impart basic psychiatric concepts and techniques as more traditional psychiatry clerkship experiences. Using both National Board exam scores and student ratings of the quality of training, it was demonstrated that consultation-liaison students are not placed at a disadvantage relative to students clerking on inpatient and outpatient psychiatry services. Given continued federal support for the development of consultation-liaison training programs and the likelihood that consultation-liaison services will be increasingly used for clerkship training (4, 5), the time is now ripe for research that replicates and extends these and previous (5) findings. Specifically, we recommend further research exploring the unique contributions of consultation-liaison clerkship training to the knowledge, skills, and attitudes fundamental to holistic care.

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The Transition to Practice Seminar

BY JONATHAN F. BORUS, M.D.

The author discusses the lack of organized faculty attempts to facilitate psychiatric residents' transition to professional practice. Interviewed residents suggested that faculty discontinue their "benign neglect" of the transition and provide information and discussion regarding the realities of different types of practice. He describes the six-session seminar he developed at Massachusetts General Hospital, which focuses on the process of transition from residency to practice, emphasizes the role of faculty as decision facilitators, and explores the costs and benefits of different types of psychiatric practice.

THE TRANSITION from residency to practice is an important and stressful period of professional development that annually affects one-third of all psychiatric residents. In the past, obligatory military service at the end of residency provided most psychiatristis with a two-year moratorium during which they could consolidate their professional identities—while practicing semi-autonomously in a sheltered institutional environment—and leisurely explore postmilitary practice opportunities. The discontinuation of the physician's draft in the early 1970s ended this sanctioned hiatus between training and practice and has pushed decision making about practice into the final year of residency training, making it a legitimate concern of senior residents and training program faculty.

Despite the fact that this predictable transition period comes at the culmination of the prolonged formal medical educational process and helps determine the ways in which residents will use their extensive training, there has been surprisingly little attention paid to it in the professional literature. I conducted a five-year interview study of 61 senior residents graduating from the residency training program of the Massachusetts General Hospital Department of Psychiatry and Harvard Medical School and describe elsewhere[1] the issues facing residents in this stressful a[illegible] tressing transition period and some o[illegible] and affective ways in which residents c[illegible] transition.

THE NEED FOR FACULTY INTERVEN[illegible]

During the transition to practice resid[illegible] fine, weigh, and assign relative priorities [illegible] flicting professional aspirations, pers[illegible] ily expectations, and practice opportu[illegible] a satisfactory practice plan. Recent [illegible] atric Association meeting panels [illegible] cussions at training directors' mee[illegible] anaugh's article (2) on postresidency c[illegible] focused on the need for faculty to fi[illegible] senior residents in this decision-ma[illegible] though specific faculty interventio[illegible] groups, regular meetings with trainin[illegible] courses in autognosis) to facilitate t[illegible] into psychiatric residency are freque[illegible] the literature revealed only a single d[illegible] formal faculty program to facilitate [illegible] tion from training to psychiatric prac[illegible] per, Allen (3) described a seminar at [illegible] which he discussed with a group of se[illegible] practical and ethical issues of enterin[illegible] therapy practice.

There have been many hypotheses ab[illegible] have not been more organized facult[illegible] cilitate residents' transition to practice [illegible] that the annual loss of highly cathected [illegible] progeny is such a painful event for a[illegible] deny or minimize the importance of this [illegible] the residents. Others suggest that sinc[illegible] residents are clearly competent adult [illegible] any attempt by faculty to intervene in [illegible] making would be inappropriately intrusi[illegible] tal. At the opposite end of the spectrum [illegible] tion that because residents become pract[illegible] end of training, they pose a competitive t[illegible] ty members with whom they will be co[illegible] variety of limited practice resources (e.[illegible] ferrals, research monies, and academic a[illegible] al positions) and that in areas that a[illegible] with psychiatrists faculty are not engag[illegible] more recently trained competition. Wha[illegible] sons, I believe it is clearly within the r[illegible] make formal efforts to facilitate (curious [illegible] and "facilitate" have the common Lati[illegible]

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The author would like to thank the resident and faculty psychiatrists who have joined in this effort at gaining a better understanding of our personal and professional development.

[1]Borus JF: The transition to practice: I. Decision issues (unpublished manuscript); and The transition to practice: II. The decision-making process (unpublished manuscript).

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easy) the growthful transition of their residents from trainees to psychiatric practitioners.

In this paper I will delineate some of the faculty interventions suggested by Massachusetts General senior residents in the interview study I conducted to facilitate their transition to practice and discuss some of the attempts of our faculty to meet transition needs. I will specifically describe a major faculty intervention, the "Transition to Practice Seminar," which has been part of the training program's core curriculum for all third-year residents over the last four years.

FACULTY FACILITATION OF THE TRANSITION TO PRACTICE

The residents I interviewed described three types of faculty interventions that they felt could be helpful to their transition decision making. These were that faculty should 1) legitimate and openly discuss the transition as a stressful part of residency training and an important part of professional development; 2) encourage residents to consult specific faculty members as decision facilitators to help each resident define his or her professional strengths, skills, and identity, and to discuss the rewards and costs of different types of psychiatric practice; and 3) provide information to aid the exploration of specific practice opportunities.

The most basic intervention suggested was for faculty to discontinue the attitude of "benign neglect" toward the transition to practice and legitimate open discussion of this transition as a stressful, important part of professional development that occurs expectedly in the final year of residency training. The residents felt that faculty should focus on the end of residency as an important part of the total training experience in a manner similar to the emphasis psychiatrists put on the termination of other relationships. Faculty should familiarize residents with the process of this termination and the decision-making tasks of the transition to practice, the normality of stress during this period, and methods used by previous residents to actively cope with this transition. They should also state formally their receptivity to and availability for discussion of transition decisions in order to decrease residents' apprehension that approaching staff for help with transition issues will stigmatize them or be interpreted as individual immaturity or psychopathology.

The second suggested intervention was for faculty to encourage residents to use individual staff members as decision facilitators during the transition. This could begin by the training director reviewing with each resident early in the final year of training the faculty's evaluation of his or her strengths and weaknesses and the practice areas that interest him or her. The training director could then suggest faculty members with specific expertise and contacts in the resident's areas of interest with whom he or she could explore strategies for practice-seeking early in the year and discuss the pros and cons of different types of practice opportunities or offers as they arise.

Because psychiatric residents often do not get the opportunity to observe closely how their supervisors and other role models actually practice, they could also benefit from candid discussion with practitioners in their area of interest of the day-to-day costs and benefits of that type of practice. At a later point in the transition period such decision facilitators could also be helpful in discussing some of the practical but often taboo issues concerned with entering practice, for example, prevailing fee schedules, office and billing procedures, debt collection, and interaction with third-party payers for those interested in private practice; and the intricacies of academic politics, requirements for academic rank, and grantsmanship hints for those interested in academic-research practice. These important practical realities are frequently left up to each new practitioner to rediscover, often through repetition of avoidable mistakes.

The final type of helpful faculty intervention suggested was the provision of information about and linkages to specific practice opportunities. A file of job opportunities could be maintained and a listing of faculty members with contacts in various subspecialty practice areas throughout the country could be compiled to provide residents with information on whom to check with concerning opportunities in a specific area or institution. This is especially important for those residents interested in academic-research careers because the academic buddy system often influences both the dissemination of information about junior faculty positions and who eventually gets hired.

THE TRANSITION TO PRACTICE SEMINAR

From these suggestions I developed a six-session weekly seminar at Massachusetts General Hospital for all third-year residents, which has been part of the core curriculum since 1974. Although officially titled "The Transition to Practice Seminar," it has become known among the residents as "Reality Rounds" because of its focus on many of the reality issues and problems of choosing and entering a psychiatric practice. The goals of this brief seminar, which is given each September, are to focus the attention of senior residents on the decision-making tasks that lie ahead of them and provide them with opportunities to learn about the realities of a variety of types of psychiatric practice.

The seminar is divided into three segments. The first session is devoted to a presentation from my research data about some of the issues, stresses, and options commonly involved in the transition to practice. The types of personal, professional, family, and practice opportunity variables that must be considered and assigned priorities are described as well as the process of exploring the costs and benefits of practice opportunities and negotiating for the practice rewards necessary to make the cost-benefit ratio a satisfactory one. In ad-

dition, some of the common affective reactions (increased anxiety, depression) experienced by residents in response to the stresses of the transition to practice are delineated to help the residents anticipate them as normal and ubiquitous. Lastly, I emphasize the availability and potential usefulness of the faculty as decision facilitators in the coming months.

The next four sessions are devoted to the careful exploration of different types of psychiatric practice. In successive weeks active practitioners of general psychiatry, academic-research psychiatry, administrative-teaching psychiatry, and private office psychotherapy make themselves available for open-ended dialogues with the residents about the realities of their practices. Two practitioners participate in each session, one senior and one relatively recent to practice, so that the residents can explore both the long-term potential and the current entry opportunities of that practice type.

After brief introductory statements by the two practitioners, the major part of each seminar is devoted to a no-holds-barred discussion in which the residents are encouraged to ask the practitioners any questions that will give them greater insight into the professional and personal sides of the life associated with that type of practice. The residents frequently probe the practitioners' personal reasons for choosing that particular type of practice; how they got started and what their advice is for starting such a practice today; what their perceptions of the most important rewards of their type of practice are (i.e., what level of income, academic status, professional stimulation, time for family and personal pursuits, and other rewards their practice provides) and what its costs, complications, and frustrations are; and over the years what their major likes and dislikes in their practices have been and what trade-offs or compromises their type of practice has required for success and satisfaction.

These sessions have often given the residents their first feel for the realities of practice, which they will soon face, such as the politics and hierarchy of their own and other academic institutions, the business ends of private practice and assessments of the availability of patient referrals in Boston and other locations, methods for seeking research support, and techniques for dealing with the bureaucracy of public or academic institutions or delivery systems. The faculty practitioners who have participated in these sessions over the years have been exceedingly courageous in sharing important parts of their personal and professional lives with the residents; this openness has demonstrated the true availability of faculty to discuss such issues with residents as they grapple in the coming months with decisions about practice.

The final session has been devoted in recent years to a "New Practitioners' Panel" in which residents who have graduated from the training program and entered their own practices in the last few months discuss their very recent transitions to practice. These neophyte practitioners, only one year ahead of the residents in the seminar, have been able to bring [illegible] sions and immediacy of the decision-ma[illegible] ing the senior residents and to share [illegible] recently planned and experienced the [illegible] their new practices. Resident questio[illegible] are more focused on "What's it like?" [illegible] go about it?" "How does it feel?" [illegible] Massachusetts General's training [illegible] market?" issues that the current res[illegible] fortable asking their colleagues from t[illegible] who have gone through the transit[illegible] training under similar market circum[illegible]

RESPONSE TO THE SEMINAR

In general, the Transition to Prac[illegible] been very well received by the resi[illegible] resident reaction during the seminar i[illegible] creased anxiety as the seminar bre[illegible] dents' denial and confronts them wi[illegible] the coming months they will have to [illegible] decisions that will have many ramif[illegible] lives. The seminar couples raising o[illegible] concurrent provision of information [illegible] help the residents begin an active exp[illegible] internal professional self-definition a[illegible] tiations for practice opportunities.

When interviewed individually nea[illegible] residencies six to nine months later (a[illegible] tinuing research project), the resident [illegible] positive about the seminar, and man[illegible] been an important stimulus to their a[illegible] in the decision-making process. Man[illegible] the seminar's description of the tra[illegible] helpful in their mapping a strategy fo[illegible] tice opportunities, anticipating the st[illegible] sion-making period, and understandi[illegible] tions to it. Several said that the sem[illegible] useful insights into the professional a[illegible] ties of the various types of psychiatric[illegible] some found these insights useful in [illegible] practice area in or out for intensive [illegible] exploration. Several residents cited the [illegible] seminar faculty as a convincing dem[illegible] faculty's desire to help in their trans[illegible] and some said that the practitioners [illegible] tions of their continued developmen[illegible] skills after residency and the continu[illegible] nities for shifts in practice decrease[illegible] these residents felt to make "final" [illegible] helped them put the transition to prac[illegible] spective of an important one of many [illegible] course of their professional develop[illegible]

CONCLUSIONS

In many residency programs, the t[illegible] training to practice is ignored, and resi[illegible]

information about or help with the important issues raised by this stressful stage in professional development must attempt individually to find help on their own. This benign neglect increases the isolation and apprehension that residents feel in making decisions about practice, reinforces normal denial that significant life changes will occur at the end of training, and may delay active coping efforts in this predictably stressful professional transition. In this report I have described some of the ways in which training program faculty can facilitate the transition of their residents to practice and a specific core curriculum seminar used over the last four years by the Massachusetts General Hospital Department of Psychiatry to meet some of these resident needs. We have found this type of intervention relatively easy and inexpensive to provide, and our senior residents have found it to be of great benefit. Although specific aspects of the transition to practice may be different in other settings, many of the issues raised are faced by most psychiatric graduates, and the faculty interventions described above can be replicated by other training programs to achieve similar beneficial effects.

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Supervision and the Professional Development of the Psychiatric Resident

BY JOHN J. DRUCKER, M.D., DAVID B. KLASS, M.D, AND MICHAEL STRIZICH

The authors used a questionnaire survey to study the way psychiatric residents experience their psychotherapy supervisors at two points in their training. Results show that second-year residents were more critical in their evaluations of supervisors than first- or third-year residents and that there appeared to be a characteristic progression in the experience of residents from year to year. The authors believe that the resident's experience of supervision reflects a developmental process, which others have described and viewed as important in the formation of identity as a psychiatrist.

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The authors wish to thank William Offenkrantz, M.D., for his rating of the supervisors.

A NUMBER OF REPORTS have focused on psychotherapy supervision as an important part of the psychiatric resident's training. Our own observations of residents' difficulties and satisfactions with supervisors suggested a relationship between the residents' experience in supervision and the process of their professional development. Our initial approach was influenced by the classic work of Holt and Luborsky (1), who emphasized the need for clinical research in psychiatric education to combine with the strength of statistical techniques in achieving more powerful findings. The existing literature discusses problems residents experience in supervision (2–6) and their professional identity development (7–8), but we could find no studies that used quantitative methods to measure changes in residents' attitudes toward supervisors in an attempt to clarify the relationship between the supervisory experience and professional development. Sharaf and Levinson (9, 10) developed a scale based on attitudes of senior faculty and used it to reflect changes in residents' therapeutic ideology over the course of the residency. However, they did not quantitatively evaluate the process of supervision as we report here. Most of the supervision studies reported are based on observations that are unsystematic. We felt

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that an attempt to quantify changes in attitudes toward the supervisory experience would be a useful contribution to research in the area as well as an aid in formulating a theory of professional development.

This study takes for granted that residents' responses to questionnaires concerning supervision not only relate to their outside world (i.e., their supervisors) but also inform us about the internal structure of the perceiver (the resident).

METHOD

During the second year of his residency, one of us (J.J.D.) actively participated in open criticism of the psychotherapy supervision program. In an attempt to collect systematic observations and complaints about all supervisors, this author developed a questionnaire for resident use in evaluating psychotherapy supervisors. The original purpose of the evaluation was to document for the residency director what residents felt were supervisors' deficiencies. Items on the questionnaire were generated by discussions with residents and faculty and from texts of psychotherapy and resident supervision (5, 6, 11). Faculty were also invited to offer suggestions and criticisms of the questionnaire. The final form was constructed by one of us (J.J.D.) with the general consensus of his resident colleagues.[1]

The questionnaire consisted of a section of 16 items in which residents rated specific aspects of their psychotherapy supervisors. Items covered the supervisor's attendance, interest in supervision, empathy for patients, ability to elucidate psychodynamics, ability to use alternate conceptual models of therapy, encouragement of the resident's capacity for self-observation, and other areas.

Questionnaires were given to all residents. A separate questionnaire was completed for each of the resident's 2 or 3 psychotherapy supervisors assigned for a 12-month period. Each item was rated on a scale of 1–5 (1=poor, 5=excellent). In addition, residents gave a "global rating" indicating the overall helpfulness of the supervisor. Written comments about each supervisor were solicited at the end of the questionnaire.

The results of the initial survey surprised us in demonstrating how inept all of the second-year supervisors seemed to be. At this point, we began to suspect that the low ratings of supervisors assigned to the second-year residents pointed to an attribute of second-year residents. Of course, it was possible that the low ratings simply meant that all of the poor supervisors were in fact assigned to the second year, or that this particular group of second-year residents rated things unfavorably even before entering psychiatric training. We hypothesized, however, that the resident's developing and somewhat conflicted professional identity was a factor in how he or she rated the supervisors. If so, we could use the supervision questionnaire [illegible] titude changes in residents.

Without revealing to the residents the [illegible] initial study, we repeated the survey [illegible] This gave us an opportunity to replicate [illegible] results as part of a longitudinal study and [illegible] portunities to test whether residency [illegible] ing. Two classes of residents were followed [illegible] nally: one group consisted of those in their [illegible] ond year and the other consisted of those [illegible] to third year. We wondered whether [illegible] pervisors would again be rated poorly by [illegible] of second-year residents.

We then compared residents' ratings [illegible] while correcting for the possibility of [illegible] tion of supervisors across years. We [illegible] faculty psychiatrist (the former resident [illegible] who knew each supervisor's professional [illegible] experience well, to independently rate [illegible] according to his own global assessment [illegible] sor's skills. Using this independent rating [illegible] ed residents' global ratings in each year [illegible] squares regression. We used the resulting [illegible] equation to represent each resident's [illegible] the analysis of variance. The above procedure [illegible] control for the fact that supervisors were [illegible] assigned on a random basis to residents [illegible] three years, leading to a possible bias [illegible] good and bad supervisors. The former resident [illegible] tor gave only a global rating because it [illegible] to have him rate each supervisor on each [illegible] vidual items.

Data were analyzed by analysis of [illegible] cover possible differences in ratings [illegible] years. A multiple regression analysis [illegible] sample of residents studied longitudinally [illegible] with the residents' global ratings as the [illegible] iable and the 16 individual items as the [illegible] variables. This regression analysis, designed [illegible] the relative contribution of each of the 16 [illegible] variables to the global rating, showed that [illegible] were highly correlated with the global rating [illegible] no particular variable stood out in importance [illegible] therefore added all of the individual items [illegible] obtain a global rating with a larger base [illegible] nal global rating, which was only a single [illegible] on a 5-point scale. This total score was [illegible] analysis of covariance as explained above [illegible] al score, i.e., with the residual scores [illegible]

RESULTS

The response rate was 92% on the 1975 [illegible] 26 residents) and 100% in 1976 (23 of [illegible] Thirty-three different supervisors were [illegible] year, and about two-thirds of these were [illegible] than 1 resident each year. In the data [illegible] questionnaire was considered separately [illegible] each of the two surveys were 56 and 53. [illegible]

[1]Copies of the questionnaire are available from Dr. Drucker on request.

FIGURE 1
Global Ratings of Supervisors by Residents in 1975 and 1976

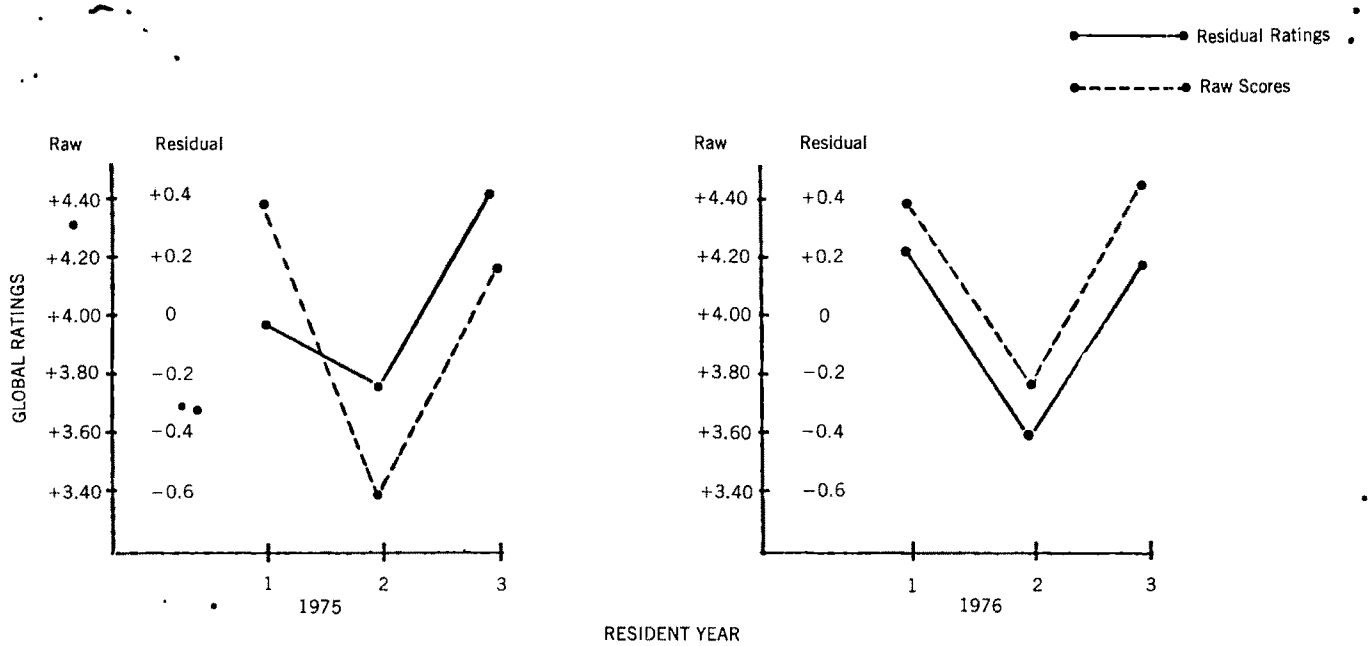


TABLE 1
Global Ratings of Supervisors by Residents in 1975 and 1976

Resident Year	1975 Residual Global Score	1975 Total Score	1976 Residual Global Score	1976 Total Score
1	−0.0483	+0.1045	+0.2548	+0.1226
2	−0.2240*	−0.2082**	−0.4032***	−0.3710†
3	+0.4106	+0.2936	+0.1826	+0.2264

*Significantly different from first- and third-year groups ($p<.004$, $F=9.30$, $df=1,53$).
**Significantly different from first- and third-year groups ($p<.03$, $F=5.36$, $df=1,53$).
***Significantly different from first- and third-year groups ($p<.04$, $F=4.46$, $df=1,50$).
†Significantly different from first- and third-year groups ($p<.03$, $F=5.33$, $df=1,50$).

corresponding to the number of questionnaires completed each year.

On each of the two sampling occasions, the relationships of the residual global ratings across resident years were identical. Residual global rating refers to the global rating after correction for possible biased distribution of supervisors according to the senior psychiatrist's rating. The second-year residents had significantly lower residual global ratings compared with the first- and third-year groups ($p<.004$ in 1975 and $p<.03$ in 1976) (see table 1). Negative numbers indicate that second-year residents' ratings clustered in a direction opposite those of their first- and third-year colleagues in the analysis of variance. Results were the same for the total scores (see table 1).

A striking pattern followed by the raw scores on all 16 items as well as the global ratings was a V-shaped one of high ratings in years one and three and low ratings in year two (see figure 1). However, it was only the residual global ratings that showed any significant differences across resident years. None of the 16 individual items showed significant differences.

The two groups that were followed longitudinally were compared against themselves for the two time periods covering a 15-month interval. The first-year residents in 1975 who became second-year residents in 1976 moved in the direction of lower ratings in the second year; this change just missed statistical significance ($p<.06$). The second-year residents in 1975 who became third-year residents in 1976 gave significantly higher ratings ($p<.0003$) in their third year compared with their second year. These changes show that the original low ratings by second-year residents were not due to their being a group of consistently low raters.

COMMENT

Results of the multiple regression analysis demonstrated the high internal consistency of the rating scale we developed. The 16 items provided a framework on the 1–5 scale indicating to residents what factors should be taken into account in making their global ratings. The global ratings were thus not haphazard guesses but were based on the preceding variables, as the high correlation between all items and the global ratings shows. When we saw the differences across years in the residual global ratings, we expected to see differences in at least some of the 16 independent variables that might explain these differences. However, there were none. No item was independent of the others. Perhaps if the independent variables were cor-

rected by having the senior psychiatrist rate them and then testing them in an analysis similar to the global ratings, differences might have emerged.

Although the profiles of the independent variables did not vary, an interesting finding was the depression of the whole scale in the second year compared with the first and third years. This paralleled the low residual global ratings in the second year. Our data clearly document that residents' experiences of psychotherapy supervision follow the "second year slump" discussed by Sadock and Kaplan (12). They described this period as characterized by "vague dissatisfaction leading to persistent complaints and formal protests against the institutional structure. Whereas the first year of the residency is characterized by eagerness, enthusiasm, and a glowing desire to learn, the beginning of the second year is often followed by discontent, dissatisfaction, and negativism." This phenomenon was described in 1959 by Holt (13) in a paper reflecting on the less obvious effects of psychiatric training, unsystematically observed in the process of carrying out a long-term research project at the Menninger School of Psychiatry.

The only study we found quantifying this "slump" is that of Pasnau and Bayley (14), who found MMPI depression scales elevated in residents at the end of their first year compared with their entering scores. The literature indicates that residents in almost every program complain about not getting enough good psychotherapy supervision (15); our study points out that these complaints, at least in some degree, are part of the slump. Further, although our measurements are different from the standardized MMPI profiles of Pasnau and Bayley's report, our findings go beyond their work in showing that residents seem to snap out of the slump by the third year. We have no data on whether or not our residents were in personal psychotherapy before, during, or after our surveys. An increasing number of residents enter treatment as the residency progresses. It would be interesting to know whether residents come out of the slump with or without therapy.

How do we explain the existence of the second-year slump? We agree with others (7, 8, 13, 16) that it may represent part of the process of identity development as a psychiatrist. Psychiatric training imposes major stresses on the resident's developing identity (17), and a "regression in the service of residency education" occurs as an adaptive step in the process. Shershow and Savodnik (18) used this phrase to describe certain behaviors of first-year residents on an inpatient unit. Although we believe this process extends [illegible] second year of training, we agree with [illegible] Savodnik that with appropriate super[illegible] vention these regressive behaviors can[illegible] dents' further development as psychiatr[illegible] suggest that this development continues [illegible] year. Our belief that the resident's g[illegible] tion with the supervisor is an impor[illegible] process is supported by Sharaf and Le[illegible] (10) that residents' therapeutic ideolog[illegible] similar to those of their experienced [illegible] the third year of training.

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Training Psychiatrists for Working with Blacks in Basic Residency Programs

BY WALTER H. BRADSHAW, JR., M.D.

The author summarizes recommendations on how specific training for working with black and other minority patients can be integrated into the existing relatively traditional residency programs. He also discusses psychotherapeutic issues and problems encountered in biracial and/or transcultural therapy that must be resolved if the training is to succeed.

RESIDENCY TRAINING PROGRAMS today are faced with a variety of competing pressures. There is strong public and government demand to train psychiatrists who will provide services in diverse locations to poor and minority populations (1). At the same time, training programs wish to maintain quality education and avoid role diffusion. Despite the fact that these pressures have been with us for some years, there is a notable lack of specific structure and content regarding the treatment of blacks and other minorities within most residency programs. Thus, psychiatry is called on to treat greater numbers of black people, but psychiatrists are lacking in critical knowledge of black people, the systems that impinge on them, and pertinent growth and development data. Even more serious is the lack of an organized approach to the understanding of the psychodynamics of racial relationships in the United States, factors that are bound to have a profound effect on treatment of blacks within the framework of existing general psychiatry training programs.

PROGRAM STRUCTURE

Orientation to Blacks

The American Psychiatric Association Task Force on the Delivery of Psychiatric Services to Poverty Areas[1] noted that community psychiatric training is generally started late in the residency, after primary attitudes and values regarding psychiatry have been formed. In addition, there is poor integration of knowledge of the dynamic perspective and knowledge of the broader systems relating to primary and secondary prevention that have impact on patients.

In programs that provide services for blacks, from the beginning there should be continuing contact with the agencies and social structures of the community. This contact should involve on-spot visitation. Didactic offerings should include history and ethnic culture, growth and development data, family structure and dynamics, group and community characteristics, sociological literature, and selected readings on "growing up black in America."

Furthermore, literature to dispel myths about blacks should be presented. Briefly stated, here are the main myths: 1) Africans arriving in the United States as slaves were only "primitive stock"; they had no culture or civilization of their own (2). 2) The black family is a "tangle of pathology" (3), as opposed to the white nuclear family. 3) A matriarchal structure is universal in black families. (Hill [4] indicated that the most common authority patterns in lower-class intact black families are more egalitarian than matriarchal.) 4) The black family is in total disarray because of slavery. (Guttman's study of the black family [5] casts serious doubt on this widespread assumption and suggests that family stability has broken down more recently under the impact of urbanization and racism since the 1920s.) 5) Psychopathology is the inevitable consequence of one-parent families; strengths cannot emanate from such families. 6) The black man is totally inferior sexually or, conversely, he is a "super-stud." 7) Blacks rarely develop severe depressions or commit suicide. (Depression is a widespread phenomenon among blacks, and there is an extremely high rate of suicide among young urban blacks [6, 7].)

Also included in this orientation should be group experiences providing the opportunity for residents to role-play minority patients and nonpatients.

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[1]Rae-Grant Q, Lightfoot O, Becker A, et al: Report from the Task Force on Delivery of Psychiatric Services to Poverty Areas (unpublished report, American Psychiatric Association, Washington, DC, 1973).

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Clinical Services

Inpatient and outpatient rotations. Part of the resident's inpatient experience should be conducted in a hospital setting separate from the main training site (i.e., separate from the university hospital). The resident should have the opportunity to diagnose and treat black patients, including those suffering from drug abuse, alcoholism, and severe personality disorders. Part of the outpatient experience should be conducted in a community mental health setting serving a black lower-class population. Diagnostic assessments and work in all forms of psychotherapy should be done on an ongoing basis. It is essential that these experiences be closely supervised by faculty who are sensitive to the minority experience (be they minority members or not). Academically their capacity must be of the highest level because they must be able to dissect the issues most relevant to successful diagnostic assessment and the psychotherapeutic process. Their skills have to include the capacity to evaluate cultural adaptation and identification versus pathology, a finely tuned knowledge of ego functions, the capacity to differentiate the role of reality as reality and as defense, techniques for the fostering of the therapeutic alliance, and sensitivity in the detection of and assistance with working through special countertransferences to minority individuals. It should be obvious that minority faculty would be invaluable for this teaching.

The chronic patient. A disproportionate number of chronic patients are minorities or poor. Their care must be elevated from neglect and being regarded as an unpleasant chore to the status of a legitimate field of training and research. This could be done by providing residents a controlled and supervised experience in constructing, running, assessing, and modifying a small system of mental health care delivery. This should begin early in the residency, preferably as a research project. For example, several sets of two or three residents might construct alternative systems of aftercare delivery using matched groups of patients.

Community consultation. Although it has already been suggested as an important component of approved residency programs, community consultation should be mandated. Assuming adequate supervision, didactic exposure, and seminar discussion, I would suggest a part-time consultation rotation in community agencies serving a lower-class black population.

The residency training director. Implementing such recommendations represents a major extra burden on the training director (8). This person must find and negotiate arrangements with new institutions and field placements, fleshing out the curriculum in the process. Furthermore, strenuous efforts will have to be made to recruit supervisors, many of whom may be uncomfortable with the broader frames of reference and confrontation with their own latent racism. Finally, the director will have to contend with the increase of resident anxiety and identity diffusion inherent in the expansion of their roles, settings, and perspectives.

THE ESSENTIAL EDUCATIONAL EXP

Seminars in psychotherapy conducte the training program should include, a vals (e.g., every three weeks), a con supervisors from both the university a nity facilities join with the trainees to chotherapeutic experience in the diff tings. Among the many possible top ferences and similarities in the patien in the psychotherapeutic technique self-perceptions of trainees in each broader issues of the structure of the and their efficacy in each setting.

One sine qua non of training psych is the development of a complete dia pist. The psychiatrist must have the an accurate diagnosis and to prescrib kind of therapy in any setting. In w patients there are many extra dimen to make this education satisfactory. above to ignorance regarding histo culture, living conditions, and so on portant are the psychodynamics that intertwined with race in every Amer white.

The dynamics of racism and racist psychotherapeutic effectiveness can be all the developmental layers of the Many have contributed to our unde phenomenon (9–14). Pinderhughes (15 have provided the most recent summa a person strives to protect himself ag anxieties precipitated by unacceptab each of his developmental stages by a tal mechanisms. The greater the dif the therapist and the patient, the mor pist would be to ward off his or her o variety of unconscious distancing an neuvers. It is not farfetched to say th goats has been protective of the a white American throughout our co e.g., the initial American attitudes ward the immigrant Chinese, Jews, a outrageous treatment of Native Americ tient's side, Pinderhughes (15) poin group-related paranoia shared by a n it is reinforced by the majority soci intensify the difficulty of psychothera signed to free the person from the crippling of the internalized conflict.

Davis and Coleman (17) stated th whelming problems of lower-class ghetto to the internalization of a hostile objec culty in establishing a stable ego ideal. uncertainty of continuity ("average ex ronment") can lead to adaptations tha the here and now, precocious self-suf constriction of intellectual freedom and c multiple disruptions of secondary auto

the same line, Spurlock (18) mentioned the constriction of hope, fantasy, and cognition that leads to intellectual underachievement and chronic depression. On the other hand, these authors have strongly pointed out the tendency of white psychiatrists to overgeneralize middle-class standards of development as the norm, thus overlooking the adaptive capacities of the ghetto-raised child. Notwithstanding multiple mothering via extended families, many of our ghetto residents manage to grow up without mental disorder. These authors also pointed out that psychiatrists neglect the synthetic ego function of the ghetto resident that allows for maximum adaptation to the reality of existing in a world in which expectations are frequently frustrated. Further, these authors stated that in looking at ghetto families, psychiatrists have paid no attention to the strengths within such families. With these biases, there is an inevitable tendency to prematurely label or mislabel as psychopathological some of the phenomena manifested by black patients.

The extremity of distortion that can result from such tunnel vision regarding the black patient has been pointed out by Thomas and Sillen (19) in their comment on Kardiner and Ovesey's *The Mark of Oppression* (20). In the latter work a major hypothesis is that blacks have practically no possible basis for healthy self-esteem and almost every possible incentive for self-hatred, a dogmatic overgeneralization of an entire people. We see a similar attitude in the tendency to glibly overextend psychoanalytic concepts to explain an entire people, e.g., the idea that slavery reduced all blacks to an infantile-oral-dependent level. Likewise, it is seen in the tendency to perceive differences from the white culture as representative of the "deficit model," according to which variations in the use of language, syntax, attitudes toward time, and other aspects of the dominant culture are assumed to be psychopathological, even among children. This latter idea has been effectively refuted by many, including Coles (21), who noted the resilience and ingenuity of the black schoolchildren who first entered desegregated schools in both the South and the North.

More subtle preconceptions can creep into the use of shorthand terminology, such as "culturally disadvantaged," "slow learner," and other phrases, however well-intentioned these were in their original descriptive origins (22). This is illustrated by a 1968 study (23) in which one group of students was represented to their teachers as late spurters while another group was not. Both groups were in fact matched. However, the children who had been identified as late spurters did so academically, while those who had not did not. Like the teachers in this study, mental health professionals can easily have entire stereotyped pictures called to mind with the use of a single phrase. Furthermore, to many black children the phrases "culturally disadvantaged" and "slow learner" have become the equivalents of the epithet "nigger" and represent just another instance of the micro-aggressions beating daily on their self-esteem.

With this background in mind, there are specific ways in which countertransference and technique errors impinge on the evaluation and treatment of black patients. One such error is that premature closure is instituted on patient evaluation or treatment with the rationalization that the patient is unsuitable for psychotherapy because the patient has difficulties with impulse control, verbalization, ego deficits, and so on. A variant of the same theme is occasionally heard from some black mental health professionals, who argue that dynamic psychotherapy is not suitable for black patients because it is too cold and intellectualized, that the average black patient's needs are more immediate, and that it is not relevant to the patient's struggles in the world. This strikes me as being partially tinged by the unconscious adoption of some of the racist myths about black people that are prevalent in our society. These remarks have certain implications, e.g., that black patients do not have the ego strength to tolerate some forms of therapy, that they do not have the ego capacity to form a therapeutic alliance even when they receive an appropriate preparatory education by therapists about the way some forms of therapy are conducted, that they do not have the capacity to use insight in relation to their own personal dynamics, that they must be invariably and strenuously infantilized, and that they do not have the capacity to use therapeutic methods that are specifically designed to offer the maximum opportunity for ego autonomy and freedom from neurotic conflicts.

Ticho (24) described a case in which she was psychoanalyzing a patient in a foreign country when she experienced a temporary breakdown of empathy and a reaction of distrust toward the patient. She recognized this reaction as projection. The impairment of her skills was reinforced by the fact that not only was she deprived of the internal identification with the patient (a foreigner) but also by the fact that she was deprived of the support of her own familiar culture.

Another error made in therapy with black patients is that everything the patient says is interpreted in glib reductionist constructs involving racial stereotypes or psychoanalytic jargon. The opposite problem can also occur: nothing the patient says is considered in relation to race—a kind of global denial or avoidance by the therapist—or all references to race are immediately defined as defensive, without any regard for the internal and external realities of the patient's life as a black person in the United States.

A potpourri of overidentifications, overcompensations, condescension, and flight, all linked together by countertransference involving warded-off aggression and/or compensatory mechanisms against feelings of inferiority and badness, can also occur. One of the more typical patterns seen, particularly in a beginning therapist confronted with feelings of increasing impotence or narcissistic injury, is the tendency to abandon psychotherapy completely and fling himself or herself into a group situation that is going to restore all mental health by changing the community. The resident can

thus become part of a hopeful situation involving an infallible force that would change the world. This reaction is also linked to the therapist's having difficulty with aggression, which is warded off by joining with the patient in an assault on the projected evil outside.

Other therapists, although they do not flee headlong from psychotherapy, may keep the patient in treatment in a condescending oversolicitous relationship in which the patient is never confronted on critical issues either within the patient's external world or the transference. In a situation in which a black patient is involved in significant amounts of destructive acting out, the white therapist may receive the added bonus via identification of satisfying his or her own aggressive impulses vicariously from a relatively protected and self-righteous position.

Schacter and Butts (25) and Grier and Cobbs (26) have described how references to race, the use of subcultural language differences, and the gross acting out of racial stereotypes can be used primarily in the service of resistance in therapy—with both therapist and patient engaged in mutual avoidance. Often, because of the projected fear of the blacks' retaliatory aggression, the therapist rationalizes his or her overemphasis on culture by saying that to do otherwise would be "trying to make the patient white."

Poussaint and Ladner (27-29) have gone into some detail regarding a particular type of masochistic overcompensation that has relevance to psychotherapeutic relationships. In this variant a white person compensating for feelings of inferiority and badness approaches the relationship with rescue fantasies involving unconscious feelings of superiority. When confronted by a black person's perception of the racist attitudes inherent in that position, the white person then either flees or places himself in the position of being abused masochistically.

It is clear that the task of the supervisor is made more complex by the issues raised here. Jones and associates (30) indicated that in their experience in residency training they found similar patterns in their supervisors' treatment of them and in the supervisors' advice regarding their black patients. Basically, if supervisors are not sensitive to these issues and have not confronted their own unconscious racism, it is unlikely they can help residents to do so. Once again, one can see the importance of recruiting minority faculty.

GENERAL PRINCIPLES

There are several fundamental and deceptively obvious guides for the therapist. The first is that therapists must engage in continuous introspection and self-analysis. Even more rigorous attention must be paid when there is a transcultural or biracial situation because the therapist is in greater danger of regressing and projecting when the patient or external environment seems significantly unfamiliar.

Therapists must make a serious effort to learn as much as possible about the culture of [illegible] that they have some comprehension [illegible] sents "normality" and ego-syntonic [illegible] patient. This will help to avoid the assumption that [illegible] understand the meaning of everything [illegible] Therapists must be more active in asking [illegible] what the patient means by a particular [illegible] or anecdote.

There must be some acknowledgment [illegible] directly or indirectly, of the therapist's [illegible] the realities of the external racist environment [illegible] pressures to which the patient is subjected [illegible] patient does not experience the reality [illegible] therapeutic alliance may be impossible [illegible] although the therapist must remain aware [illegible] tial dangers of infantilizing the patient [illegible] times when it is necessary to help the patient [illegible] the realities of external existence before [illegible] ous with the internal conflicts.

Therapists must recognize and become [illegible] with their own and their patients' ages [illegible] varying and different perspectives. For [illegible] black power movement had a significant [illegible] as a phase of self-assertiveness that enhanced [illegible] tal health of black people in this country [illegible] with black patients, therapists must [illegible] adaptive value of autonomous constructive [illegible] tive action, thought, and feeling and not [illegible] by it. However, on the other side of the [illegible] must be prepared to deal with the noradaptive [illegible] of aggression and neuroses of black patients [illegible] obstacles these place in the path of psychotherapy [illegible] of these have been outlined by Pinderhughes [illegible] nial, paranoid feelings and distrust, silence [illegible] passivity, precipitation of crises, and [illegible] payment of bills, and missed appointments [illegible] phenomena frequently represent indirect [illegible] due to fear of assertiveness in the psychotherapy [illegible] though they may have been adaptive defenses [illegible] and historically (and, as mentioned above, [illegible] by the dominant society in the present) they [illegible] sent maladaptations in the present. Pinderhughes [illegible] vocates quick identification and interpretation [illegible] resistance in order to engage the patient's [illegible] ful attention following any therapeutic [illegible] determine whether distortion has occurred [illegible] jection or misunderstanding via subcultural [illegible]

Within the context of a therapeutic relationship [illegible] which the alliance is secure, not only can [illegible] gin but it can flourish with black patients [illegible] dismissed as "unsuitable." It requires, however, that [illegible] therapists have attained sufficient resolution [illegible] control of their own fears and aggressions [illegible] can mitigate their own racism.

There are many challenges and joys [illegible] transcultural work. Many of us have noted [illegible] ic effect of racial differences on the therapeutic [illegible] ess. The differences often bring certain [illegible] sharp and dramatic focus, facilitating and [illegible] therapy. This occurs most often when the [illegible]

and stereotypes are related to the same conflicts and affects as the transference. Furthermore, the intensified necessity for self-observation enhances the professional skills of therapists as well as their own personal growth.

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Psychiatric Education and Training of Asian and Asian-American Psychiatrists

BY NORMUND WONG, M.D.

The Asian and Asian-American physician in a standard psychiatric residency is faced with discrimination at several levels, as well as special problems of professional role confusion and personal identity, all of which can create barriers to professional development and productivity. Further, most standard psychiatric residencies do not include teaching or supervision specific to treating Asian patients. The author proposes the establishment of a specialized program, focusing on ethnic minority groups, within a general psychiatric residency; he discusses specific features of such a program. This approach may help to alleviate the problem of inadequate resources for handling the mental health problems of Asian-Americans and underutilization of mental health resources by this group.

WHAT HAPPENS when an American citizen with an Asian background becomes mentally ill, especially when that individual has lived for many years in a racial ghetto? Historically engulfed by hostile and culturally foreign environs, Asian-Americans tend to cluster together for mutual assistance and comfort—to have ready access to customary foodstuffs and to communicate in and perpetuate the native language and cherished customs. All too often Asian-Americans tend to keep mental health problems a family affair. By the time they seek outside help at a mental health facility, personal resources have been depleted, the family unit is exhausted and angry, and intrafamilial strife has become overwhelming. As a result, an appeal for outside help often signifies a rejection of the sick member by the family and community.

At the mental health facility the Asian-American patient is seldom seen by a psychiatrist who understands his or her traditions and mores, even if the psychiatrist also happens to be an Asian-American. Although the psychiatrist may have graduated from [illegible] garded residency he or she probably [illegible] speak the Asian patient's language. Ce[illegible] will be difficult to diagnose and treat [illegible].

A recent survey (1) revealed that of t[illegible] Asian-American psychiatric residents [illegible] Menninger School of Psychiatry, onl[illegible] treated an Asian patient while in trai[illegible] there are several hundred Asians in the [illegible] The same situation exists in large citie[illegible] and West coasts, where the Asian pop[illegible] mense by comparison. It is small [illegible] Asian-American citizens find much [illegible] with our psychiatric service delivery sy[illegible]

Asian-Americans represent a signifi[illegible] of the U.S. population and are a heter[illegible] complex group with diverse religions, [illegible] guages, and histories. For example, acc[illegible] census data, there were 1,439,412 pers[illegible] origin living in the United States: of [illegible] were Chinese, 249,145 were Filipinos, [illegible] were Japanese. Certain racial myths exis[illegible] Americans. They have been stereotyped [illegible] zens who are quiet, self-sufficient, ind[illegible] well adjusted to the American society [illegible] ever, this image is being dispelled by [illegible] studies. Within Asian-American popul[illegible] an increasing incidence of suicide, alco[illegible] nile delinquency, and drug addiction. A [illegible] vestigators feel that psychopathology [illegible] Americans has been underestimated and [illegible] resources for handling their mental he[illegible] are inadequate (6, 7, 12–16).

The necessity to develop meaningful [illegible] mental health care programs for Asian [illegible] obvious and has been recognized by [illegible] 1974, when it made available through [illegible] Psychiatric Association stipends to incr[illegible] ber of minorities in the mental health f[illegible] 1975 the National Advisory Mental H[illegible] Subcommittee on Minority Related Re[illegible] mended that at least 20% of all NIMH f[illegible] for "relevant research areas" be earmar[illegible] ity studies by the end of fiscal year 19[illegible] recognition of this problem, the Presid[illegible] sion on Mental Health in 1977 designate[illegible] study the mental health of Asian-Americ[illegible] ic Islanders.

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2-6, 1977.

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TRAINING AND EDUCATION ISSUES

This paper addresses two specific issues in the training and education of Asian and Asian-American psychiatrists. First, I will discuss the plight of the Asian-American physician in a standard psychiatric residency and second, I will conceptualize a residency program especially aimed at preparing Asian-American and Asian psychiatrists to treat Asian-American populations. The educational principles buttressing such a program might well apply to any psychiatric training program with the explicit goal of treating minority group patients.

Asian-American Trainees in a Standard Psychiatric Residency

Institutional racism is experienced by even the most acculturated Asian-American U.S. medical school graduate. It is beyond the scope of this paper to discuss the plight of the Asian foreign medical graduate; however, suffice it to say that discrimination is present in even more gross and blatant forms.

The American-born Asian trainee is confronted by issues different from those experienced by his white counterpart. Despite his or her credentials, faculty members and patients consciously or unconsciously tend to equate him or her with the foreign medical graduate. He may encounter discrimination even before entering the training program. The Asian-American's name often causes the admissions committee to engage in stereotyped fantasies about his alleged docility, passivity, inscrutability, mysterious Asian upbringing, and lack of adaptability to Western mores. He encounters these same fantasies throughout his residency years from a succession of clinical and teaching staff members. Although he may be a third-generation American he remains highly visible. He does not blend easily into the white population. Consequently, he might become entangled in a self-fulfilling prophecy. The faculty and staff of the program may have assuaged any guilt about discriminatory practices by accepting him for training. However, discrimination may exist on an overt or covert level. As a resident, he might be accepted on the same footing as any of the white trainees; such a move would be very appropriate. However, even then, especially for a second-generation Asian-American, the positive cultural and personal assets that he brings to his training might be lost or devalued in a subtle fashion.

Much has been written about the psychiatric trainee's identity crisis and penchant for role confusion. It is generally acknowledged that a successful resolution often hinges on identification with an admired teacher. The task is more complicated for the American-born Asian trainee, who must not only contend with his professional role confusion but must also resolve the matter of his personal identity. Unlike his white counterpart, he rarely has contact with an American-born Asian psychiatrist after whom he can model himself. In treating patients he may encounter initial relationship and transference problems due to his projected image; these problems usually do not arise among other trainees, and they may not be readily apparent to his supervisors. If a strong tendency exists to stereotype him, the staff may assign him primarily non-dynamically oriented and traditionally less desirable patients, minority patients, or multiproblem patients, and in general offer him a less satisfactory psychotherapy training experience than his peers. He may be steered into research tracks with fewer patient contacts.

Should he seek psychoanalytic training, he is likely to encounter the same, although more intensive, biases that he experienced when applying for residency training. Gross generalizations may abound, excluding individual considerations—for example, "It is not possible for a therapist to analyze an individual of another race because of the divergency of psychological biases." "Asians are so obedient, intellectually defended, and need so much structure that free association and therefore analysis are impossible." Despite these views there are a small number of American-born Asian graduates of psychoanalytic institutes.

Problems of the Graduate

Assuming that the American-born Asian psychiatrist successfully hurdles these difficulties and graduates, he or she may run into a new set of problems. Should he choose to pursue private practice, he must again contend with the image of being a foreign medical graduate whom colleagues and patients may regard as inferior. He must struggle more than most medical graduates if he wishes to establish a private practice in a predominantly white middle-class population. Should he wish to avoid the aggressive and competitive stance required for private practice, he might enter a government system, assume an administrative position, do research, or enter the academic field of psychiatry. In any of these settings he still encounters problems as a consequence of his Asian background.

Sata[1] has pointed out that typically the American-born Asian psychiatrist, as a minority member, is asked to serve on numerous committees that consume inordinate amounts of time and create further barriers to his or her professional development and productivity. Sata also mentioned that the Asian-American may be disliked by white colleagues because his mere presence evokes guilt feelings or defensiveness. In addition, in many institutions there is the unspoken feeling that minority faculty, in order to be found deserving, must work twice as hard. It is probable that even the most successful American-born Asian psychiatrist has experienced some such form of discrimination or difficulty. Aside from the obstacles that he must surmount in order to achieve success, one must take into account the losses both to the Asian-American psychiatrist and the members of his ethnic group as he seeks

[1]Sata LS: Cultural diversity and psychiatric training (unpublished paper, 1973).

to assimilate into a white-oriented psychiatric system. Unless he chooses to practice or teach in a community with a significantly large ethnic population of which he is a member, he is unable to pass on much of the dynamic understanding and successful coping techniques that he may have learned in order to resolve his own struggles and which may be difficult for a therapist of another ethnic background working with Asian-Americans to fully comprehend and empathize with. In addition, American-born Asian psychiatric residents and other professional individuals or students who may be experiencing similar conflicts may not have access to him as a resource.

Finally, if an American-born Asian psychiatrist decides to practice in an Asian-American community, he or she often finds that training is of little practical help. He or she may have retained much knowledge about the religions, mores, and philosophy of his or her ethnic group, but with notable exceptions, nowhere during the standard psychiatric residency was he or she exposed to any teaching or supervision specific to treating Asian patients. Frequently, the American-born Asian psychiatrist has never treated an Asian-American patient during his entire residency. From the few studies available (9, 17), it appears that Asian patients are usually assigned to mental health workers other than psychiatrists and psychologists and are infrequently seen in individual and group therapy. Psychiatric residents are exposed early in their training to notions such as "Asian-American patients generally do not respond well to a psychological approach, are somatically oriented, and are unable to question authority." This induces some American-born Asian psychiatrists to follow an organically oriented approach. If the American-born Asian psychiatrist is trained in a psychodynamically oriented program that places great emphasis and value on the highly verbal patient, he or she may be given little encouragement and time for exploring ways of educating Asian patients to the workings of the psychotherapeutic process. Commonly, such patients are specifically referred to him for a "supportive and drug approach."

Many American-born Asian psychiatrists are poorly informed and ignorant of the culture and language of their own ethnic group. Even if they are motivated to work with Asian-American patients, they may be ill-equipped for the task. For example, of the 84 identified Chinese-speaking psychiatrists in the United States, 9 only speak Cantonese and cannot converse in Mandarin or Toi San. Twenty-one can only speak Mandarin (18). There are only 2 Chinese psychiatrists who reside and work in New York City, where most of New York State's 81,378 Chinese live. One can easily conjure up a Kafkaesque scene in which an emotionally disturbed Chinese patient is finally brought to the office of a Chinese-American psychiatrist, and neither can speak to the other except through a translator. Thus despite the presence of accredited American-trained Asian psychiatrists, Asian-American communities may still lack adequate psychiatric services.

The Need for Specialized Psychiatric Tr... Programs

Just as many trainees select a psychi... that they feel will fulfill their needs to p... in psychoanalysis, research, or comm... try, there should be the opportunity for t... international psychiatric or ethnically s... dency programs. Aside from the progr... versity of Hawaii to train Indonesian ps... establish and practice child psychiatry ... country (19), there are no psychiatric r... grams in the United States that incorpo... ment the cultural, social, and philosophi... that exist among Asian-Americans (20) ... edged among American-born Asian psy... while there is an obvious need for psy... among the Asian-American populatio... psychiatric approach that is taught in ... programs must be modified for most o... (9, 13). Basic research and evaluation ... specific treatment programs with follow... lacking. The following program I prop... suitable for all Asian psychiatrists, not ... can-born Asians.

PROGRAM CURRICULUM

Any program specifically designed to ... psychiatrists must meet the general req... tablished for psychiatric graduate med... However, care must be exercised to a... of courses and clinical experiences in ex... pose hardships on the resident. While ... sible within the standard residency per... Asian-American resident to function a... psychiatrist in his or her ethnic com... probably require additional time and ... the stated goals. Similarly, the trainee in ... try may require extended training. ... should carefully monitor the tra... throughout the program as well as per... studies to justify the value of exten... period beyond the time required for ma... ates.

Formal curriculum must provide ... parative psychopathology (focused on ... ethnic variations), cultural norms and ... social psychology, religions, and soc... ethnic group. There should be semin... writing, and speaking the ethnic langua... there should be seminars dealing with se... and mental health issues of other minorit... search and teaching skills are to be enco... courses should be taught throughout the ... years of training rather than being relega... time.

The specialized training program could ... ent, but in all likelihood it would prob... best as a track system within a general ...

gram. Because all of the residents would have contact with the local ethnic population, some of the special courses should be included in the curriculum for all trainees, with others being elective. For example, a seminar on comparative psychopathology and cultural norms might be required and a course on reading, writing, and speaking the ethnic language be elective. All faculty should be encouraged to participate in these seminars in order to become aware of the difficulties frequently experienced by Asian-Americans. A sensitivity group experience should be mandatory for all residents and faculty to bring into awareness unconscious racial issues that may crop up in supervisory and teaching situations.

While the Asian trainee must be exposed to the clinical experiences required of non-Asian trainees, he or she should participate especially in outreach and aftercare programs. Above all, the trainee's patient load should not be restricted to Asian-American patients; he or she needs to be exposed to a wide variety of individuals with different clinical syndromes and requiring various treatment approaches. The trainee should be encouraged to try innovative therapies for Asian-American patients not fitting into clearly defined Western treatment categories.

TRAINEES AND FACULTY

It is proposed that training programs designated to educate Asian psychiatrists within a general residency have at any one time a minimum of two Asian trainees. Peers learn from each other, apart from the formal instruction they receive, and they need a psychological and culturally similar base support in a new environment. The presence of two or more culturally related Asian trainees, for example, two Japanese-Americans or a Chinese-American and a Japanese-American, would help reinforce their ethnic identification and special training objectives. The trainees should be members of that ethnic minority population served by the training institution or be culturally related to it. If an Asian applicant does not wish to enter this specialized track, he or she should not be counted as one of the two trainees deemed necessary to maintain such a program but should be considered instead as a regular resident.

There must be an adequate number of Asian faculty. I suggest that there be a minimum of three key Asian faculty, consisting of a social scientist and two psychiatrists. One psychiatrist should be a significant figure in the didactic teaching program and the other a clinical supervisor. Part-time Asian faculty and consultants should be involved to further strengthen the didactic and clinical teaching. Being an Asian should not be sufficient in itself to qualify the individual as a key faculty member for the special program. The person must know the cultural, social, and philosophical aspects of his or her ethnic heritage; be able to communicate in the language; and have worked with Asian-American patients and community groups. Outside Asian faculty should be invited to teach in this specialized program. Because it may be difficult to find within a single institution sufficient, qualified Asian faculty, it is essential to engage visiting consultants from other geographic locations in order to provide the program with breadth and depth.

It is important that expert non-Asian faculty also be involved in teaching and supervision. Other faculty not directly involved with the special curriculum or clinical track should be encouraged to attend these seminars and case conferences in order to prevent a sense of fragmentation of the general training program and isolation of the Asian trainees. The resident in the training program for Asian psychiatrists should be regarded in the same light as the fellow in child, forensic, community, or research psychiatry who is expected to possess a sound grounding in basic psychiatry on top of which he or she chooses to superimpose additional, specialized interests.

LOCATION, FISCAL CONSIDERATIONS, AND PROGRAM VIABILITY

The institution should be significantly involved in rendering psychiatric services and readily accessible to a large Asian-American population. An institution located in a large geographically identifiable area, such as a Japantown or Chinatown, in the midst of a predominantly non-Asian community would be more suitable as a training site, than, for example, an institution in Honolulu, where the Asian-American population is the majority. According to the U.S. Bureau of Statistics, 44.1% of the population in Honolulu in 1970 was of Japanese descent. It would also be undesirable to locate a training program in an area where the ethnic population has acculturated into the general population with little influx of recent Asian immigrants and which does not typify the plight of Asian-Americans in large West and East Coast cities.

One or more ethnic training tracks may be situated in a training institution located, for example, in the San Francisco Bay area, where there are significant Chinese, Japanese, and Korean populations. The requirements for each ethnic training program should be maintained whenever possible, for despite many similarities, there are significant differences and needs among the groups. In addition, the ethnic training programs should be multidisciplinary to include training for Asian psychologists, psychiatric nurses, and social workers within the same institution. For this reason these programs should be in institutions that are affiliated with medical schools or universities.

It can be anticipated that the establishment of a "separate but equal" specialized residency training program that focuses on minority groups will draw protests from certain factions of the psychiatric community, which object to the implication that racism and discrimination exist within our profession. My ex-

perience as a psychiatric educator and frank discussions with Asian and non-Asian colleagues indicate that it is time that we address the issue of psychiatric education for all of the minorities. Unlike the black and native American psychiatric applicants who are now much sought after by training programs, Asian candidates have been ambivalently viewed as a glut on the market and yet desired as a cost-accounting measure for institutional service needs. Their educational concerns have been neglected, and as a consequence they have suffered personally and professionally; most of all, the Asian-American patients have lost out in the long run.

Implementing these training programs will be costly. Although close attention must be paid to the current epidemiological studies to justify the funding of such programs, vigilance must be exercised to ensure that their initiation is not unduly delayed. According to Okura (21), during the period 1968–1972 a total of $50 million was allocated to black and Spanish-surnamed trainees, while not a single cent was spent on Asian-Americans.

Another compelling reason for the establishment of training programs for Asian psychiatrists is the Health Professions Educational Assistance Act of 1976 (Public Law 94-484), which became effective on January 10, 1978. Foreign medical graduates will have to limit their stay in the United States to two years, and all exchange visitors must return home for two years before applying for permanent resident status. Over 70% of all foreign medical graduates were Asian, according to a 1972 HEW report (22), and now the majority of Asian psychiatrists in this country are foreign medical graduates. More than ever, the opportunity now exists to blend Western dynamic psychiatry into an appropriate cultural context for export to Asian nations. For this we need specialized training programs. Finally, skepticism has been expressed about the long-range needs for specially trained ethnically oriented psychiatrists. The assimilation and acculturation of Asians into American society have been slow and historically much opposed processes. The "melting pot" dream has not worked for Asians. In addition, there has been a phenomenal rise in the Asian population in this country because of increased immigration in the past few years from Korea, the Philippines, China, and more recently, Viet Nam. It is projected that by 1995 the Asian-American population will rise to 3 million. Mental health resources have been underutilized by Asian-Americans, and the demand for appropriate and effective psychiatric services will not be s[illegible] long time.

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EDITORIAL

Children of an Idle Brain?

UNLIKE THE fabled vicar who "trod the strait and narrow path between good and evil," students of the dream have generally been divided into two opposing camps. Freud's extensive review (1) of the nineteenth-century literature well documented the polarization between those who disparaged dreams as an anarchy of meaningless images arising from a brain whose higher functions were inoperative during sleep and those who saw in them a creative force giving rise to knowledge inaccessible to the rational waking mind.

Freud's own contributions to the understanding of dreams need not be belabored here. Suffice it to say that (as every educated twentieth-century man and woman knows) he viewed them as the "royal road to the unconscious"—as the symbolic, usually disguised expression of basic human wishes and needs taking their origin from the earliest phases of the individual's life. However, until the posthumous publication of the "Project for a Scientific Psychology" (2) in the 1950s, it was not generally recognized that Freud was from the start concerned with the neurophysiology underlying psychic processes, including dreaming. The fact that he very early on abandoned the attempt to create a neurological model and focused on the psychological aspects of dreaming determined the cast of analytic thinking for decades to come.

The concept that dreams had a method and order of their own and that their meaning could be fathomed by analysis has not been significantly challenged until recently. In the last two numbers of volume 134 of the *Journal*, Drs. J. Allan Hobson and Robert W. McCarley (3, 4) took serious issue with the analytic view of dreams and dreaming. They not only pointed out that Freud's neurological model was archaic in the light of modern knowledge of the brain's function, but they provided evidence that dreams were after all merely the senseless, random accompaniment of the autonomous electrical activity of the sleeping central nervous system.

If controversy—sharp, critical, intelligent controversy—is an editor's fond desire, his dreams came true. The two papers in question provoked more letters to the Editor than the *Journal* had ever received before. Subsequently published, these were merely the opening salvo of response, and the opposing forces, having marshalled their arguments, now return cannon fire for cannon fire in the form of two articles appearing elsewhere in this issue. Dr. Gerald W. Vogel aims his sights at the neurophysiological model proposed by Drs. Hobson and McCarley, suggesting that their hypothesis is not fully supported by what is known about the dreaming brain. Dr. Anthony L. LaBruzza, pointing to the fundamental irreducibility of psychological and physiological concepts and language, one to the other, argues that Hobson and McCarley's *physiological* formulations of dream processes have no logical bearing on the truth or falsity of the *psychological* understanding of dreams and their meaning.

The battle is joined. We are hopeful that, given a forum, scientific dialectic will lead to a reasonable synthesis. Meanwhile, safe in his foxhole, the Editor will remain neutral; he promises to give equal time to both camps and to cut the logomachy short when it begins to pall.

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J.C.N.

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BRIEF COMMUNICATIONS

An Alternative View of the Neurobiology of Dreaming

BY GERALD W. VOGEL, M.D.

The author critiques the recently proposed activation-synthesis hypothesis about the origin and formation of dreams. Many findings do not support the new hypothesis that specific pontine physiological processes, rather than mental processes, instigate dreams and produce their distortion. First, dreaming often occurs in the absence of the pontine processes. Second, forebrain activity (which can have mental correlates) is crucial to the instigation and maintenance of dreaming sleep. Finally, activity of the proposed pontine dream generator, which is claimed to cause dream distortion, is not reliably accompanied by dream distortion.

TWO PAPERS by Hobson and McCarley have integrated a vast array of recent neurophysiological findings to propose a new, non-Freudian theory of dreaming, the activation-synthesis hypothesis (1, 2). The crux of the hypothesis is that physiological processes rather than psychological processes are the primary instigators of dreams and are the primary determinants of their formal characteristics (viz., distortion, bizarreness, discontinuity, and incoherence). Since Freud proposed that psychological processes instigated dreams and determined their formal characteristics (3), Hobson and McCarley concluded that the recent neurophysiological findings refute Freud's dream hypothesis.

This paper represents an alternative to these views of Hobson and McCarley. I will present evidence that their papers did not consider and that I interpret to be inconsistent with the activation-synthesis hypothesis. Furthermore, on methodological grou[nds] that all of the evidence which modern ne[urophysiology] can provide does not and cannot refute [Freud's] dream hypothesis.

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MAJOR PROPOSITIONS OF THE HYPO[THESIS]

The validity of the activation-synt[hesis]—and its corollary, the refutation of F[reud's] hypothesis—rests on the evidence suppo[rting] main propositions (1). These propos[itions] stated as follows:

1. Dreaming occurs only during a c[ertain] physiological state known as the desyn[chronized] state or D sleep.

2. Pontine FTG cells are the sole gener[ators] D state. Augmentation of FTG discharge [is] necessary and sufficient condition for [the] of the D state. Dreaming is produced by [the] activation of the forebrain, which creat[es] using stored memories to make sense of [the] originated in the pons. The dream, ther[efore] synthesis of a pontine activation pat[tern] vious experience—hence the term "[activation-] thesis." Because the neural mechanism [of] the D state is located in the pons and is [genetically] preprogrammed, metabolically determi[ned] dreaming is instigated by a physiolog[ical] Since Freud proposed that dreams w[ere] psychological processes such as uncon[scious] tive wishes, his theory of dreams must [be]

3. The formal characteristics of dream[s] bizarreness, discontinuity, and incoh[erence] caused by the temporally random activ[ation] forebrain by the FTG cells. Barraged [with] stimulation, the forebrain "does the b[est] job" to make sense of such randomness. [Hence] dream distortion, incoherence, etc. Hence [the] tortion is caused by a physiological pro[cess] activation) rather than a psychological pr[ocess]

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the defensive need to disguise or censor unacceptable wishes, as proposed by Freud.

EMPIRICAL CRITIQUE OF THE HYPOTHESIS

What Causes Dream Distortion

I will now examine evidence and methodologic issues related to the activation-synthesis hypothesis that were not considered by Hobson and McCarley. I begin with the proposition that dream distortion is caused by a partially random discharge of neuronal units (FTG cells) in the pontine D state generator.

Hobson and McCarley present no evidence to support this proposition. Indeed, it is an assumption without empirical support. We have no evidence that "disordered" meaning is caused by a disordered (temporally random) discharge pattern of individual neurons. In more general terms, we do not know the neural correlates of particular sequences of mentation, ordered or disordered. We also do not know that the orderliness of mental sequences is determined by the pattern of hindbrain stimulation of the forebrain. It is possible that mentation is relatively independent of hindbrain stimulation and is primarily determined by the response characteristics of the forebrain. In any case, our ignorance about these issues is very great. In view of such ignorance, the claim that disordered (incoherent) meaning is caused by disordered (random) discharge of individual pontine cells appears to be based on verbal similarity rather than empirical findings. Indeed, the empirical findings do not support this claim.

Contrary to the hypothesis, the empirical correlation between irregular (phasic) discharge bursts of the proposed pontine generator and dream distortion is very weak. Relevant studies report that dream distortion, bizarreness, and discontinuity are unrelated or only weakly or unreliably related to indicators of the generator's phasic discharge bursts (4–6). Furthermore, based on the high frequency of random phasic bursts of the generator, the hypothesis would predict that distortion, bizarreness, and incoherence are very frequent characteristics of dreams. However, contrary to the hypothesis, D state dreams usually contain realistic imagery, concern banal everyday events, and display remarkable thematic coherence (7–10). Thus, the correlational evidence does not support the claim that dream distortion is caused by the partially random discharge of a pontine D state generator. Put directly, D state dreams simply do not resemble that kaleidoscopic barrage of unconnected images one would expect from a random generator of mentation.

Furthermore, the activation-synthesis hypothesis cannot explain the remarkable coherence of most dreams by shifting its emphasis from the disordering effect of random hindbrain stimulation to the ordering effect of a forebrain synthesizing response. Such an explanation would mean that although the hypothesis has not predicted dream coherence, it could, post hoc, conveniently invoke just the right amount of synthetic activity to explain the observed coherence of any dream. With such explanatory agility, the hypothesis would be compatible with all degrees of dream coherence but would predict none. In that case, the hypothesis would have no testable empirical consequences and would therefore be empirically meaningless.

The "Dream State Generator"

Next, I will examine evidence on an alternative to the proposition that the "necessary and sufficient" generator of the D state is a pontine mechanism in which the FTG neurons are the specific D state executors. Support for this proposition comes mainly from animal transection, lesion, stimulation, and unit recording studies. My examination of these studies leans heavily on Siegel's recent critical review of the neurophysiology of D state generation (11) and on the related work of Siegel, McGinty, and Breedlove (12–14).

Consider first the transection studies. Cats, decerebrate at the pontine or pontomesencephalic level "exhibit all the brainstem components of the D state . . ." (11). Furthermore, there is no D state in the permanently isolated forebrain of the cat (15). Clearly, the brain stem—and not the forebrain—contains structures whose activity is a necessary and sufficient condition for the brain stem components of the D state. However, these results do not rule out the possibility that in the intact animal, forebrain structures interact with the brain stem D state structures in such a way that the forebrain is crucial for D sleep. This possibility is supported by the McGinty and Sterman finding that lesions of the basal forebrain (which left the brain stem intact) ". . . totally eliminated (the D state) for periods of several weeks" (16). The work of Villablanca and Marcus (17, 18) more directly supports the crucial importance of the forebrain for the D state. These investigators studied sleep and wakefulness over several months in chronic diencephalic cats, i.e., cats without neocortex, striatum, and rhinencephalon. In such cats, non-D sleep was reduced to 1/8 of that in control (nonlesioned) cats, and D sleep was reduced proportionately much more to ". . . less than 1/20 of the controls. In fact D sleep was completely absent in 10 out of 48 sessions (24 hours of observation) while non D sleep was never absent. . . . The normal cyclic distribution . . . of the remaining non D sleep and D sleep could not be demonstrated" (17, pp. 357–358). Moreover, according to my calculations from the other Villablanca and Marcus data (17), the diencephalic cats averaged 2 episodes of D sleep per 24 hours, while the controls averaged 31 longer episodes of D sleep per 24 hours. This evidence indicates that, contrary to the activation-synthesis hypothesis, the forebrain plays a crucial role in the instigation, maintenance, and timing of most episodes of D sleep. Evidence from the Villablanca and Marcus study also indicates that the forebrain affects its own activation pattern during D sleep because the frequency of rapid eye movements (or phasic discharges) during the D state was higher in the chronic diencephalic cats than in the intact control

(17). In the intact animal, the forebrain exerted an inhibitory influence on the D state phasic discharge pattern of the hindbrain. Therefore, contrary to the activation-synthesis hypothesis, the hindbrain activation pattern is not autochthonous, and the forebrain is not simply a passively responding slave of the hindbrain. Rather, the forebrain influences the hindbrain activation pattern. As Villablanca and Marcus put it, "the present finding in 'diencephalic' animals necessitates a revision of [the] concept (that rostral structures do not exert much control over caudal brain stem sleep mechanisms) . . ." (17). "Our hypothesis of the direct, although complex, descending forebrain influence controlling the lower brain's sleep wakefulness structures appears to fit the available data better" (19).

Pontine lesions that eliminate the D state, as in the transection studies, have been interpreted to indicate that the brain stem contains the necessary and sufficient generator of the D state. However, Siegel's critical review offers a different conclusion (11). Siegel emphasized the many shortcomings of the lesion technique when applied to the brain stem. In particular, the specific effects of a localized brain stem lesion cannot be separated from the strong nonspecific effects of the lesion. The latter include serious morbidity (often leading to death), destruction of surrounding blood supply, and death or damage of neighboring neurons and neuronal fibers—all of which occurrences have adverse effects on remote structures. Thus, elimination of the D state by pontine lesions (although consistent with the theory that pontine structures are necessary for D sleep) does not necessarily imply that in the intact animal the pons contains structures sufficient for generation of the D state.

Similar conclusions may be reached from stimulation studies. Although pontine stimulation can instigate the D state, "stimulation of a variety of structures including cortex [20], vagal nerve [21], and the visual system [22] can induce the [D state]" (11). Thus, structures outside the brain stem, including many cortical areas, can instigate the D state.

Finally, on the basis of unit recording studies, Hobson and McCarley have proposed an ingenious pontine mechanism to explain the alternate generation of non-D sleep and D sleep (23, 24). According to these workers, two pontine neuronal groups, a D state inhibitor and a D state generator, reciprocally interact to produce the alternation of non-D sleep and D sleep. The pontine FTG cells are the specific neurons whose augmented activity generates the D state. Hobson and McCarley originally suggested that these neurons were the D state generator because, among other properties, the FTG met the crucial selectivity test, i.e., their firing rates increased selectively during the D state and decreased in the absence of the D state (during wakefulness and slow sleep) (23, 24). More recently, considerable controversy has developed among neurophysiologists about whether the FTG neurons meet the selectivity criterion (11-14, 25). I will present evidence that they do not. In doing so, I am not criticizing the reciprocal interaction model. My po[illegible] cerns the anatomical location of the D sta[illegible] not the mechanism responsible for the alt[illegible] and non-D sleep.

Evidence from two independent labora[illegible] gations indicates the original finding that [illegible] selectively during D sleep was an artifact [illegible] nal experimental procedure (11-14, 25). [illegible] ity for D sleep was originally found in ca[illegible] placed in head restraints to prevent hea[illegible] and to maintain the location of the rigid re[illegible] trodes in the brain (23). Using mo[illegible] trodes, Siegel, McGinty, and Breedlove [illegible] found that in cats without head restrai[illegible] rates of FTG cells during waking hea[illegible] were as high as during D sleep. Ver[illegible] tially the same observation in rats (25). [illegible] cies, the waking FTG discharge during [illegible] not instigate the D state. Agreeing w[illegible] McGinty, I interpret this evidence to i[illegible] mented FTG discharge is not selectiv[illegible] D state and that FTG cells are not the [illegible] D state.

Furthermore, the evidence does not [illegible] expected defenses of the FTG cells as D[illegible] ators. Specifically, there is no evidence [illegible] portion of FTG cells firing during con[illegible] movements is different from the proporti[illegible] ing D sleep, 2) that the firing rates of FTG [illegible] during waking than during D sleep, an[illegible] waking FTG discharge generates only p[illegible] state, such as the phasic or motor com[illegible] Thus, considering all aspects of the issu[illegible] along with Siegel and McGinty, that the [illegible] not meet the selectivity criterion and are n[illegible] ators of D sleep or of any component [illegible] as Siegel points out, despite an intensiv[illegible] pontine neurons have been found that se[illegible] during D sleep (11).

In summary, transection, lesion, stim[illegible] unit recording studies suggest that in the [illegible] both the hindbrain and the forebrain par[illegible] generation of the D state. The hindbr[illegible] structures whose activity is necessary for [illegible] However, contrary to the activation-sy[illegible] esis, in the intact animal the forebrain [illegible] role in the instigation, timing, maintenan[illegible] vation pattern of most episodes of D sleep [illegible] instigators of most D state dreams and [illegible] nants of their formal characteristics are [illegible] processes in the brain stem, where mu[illegible] has no psychological correlates. The fore[illegible] activities can have psychological correlate[illegible] a role in the generation of the D state. [illegible] opens the possibility for psychological ev[illegible] ing and formally shaping D state dreams.

Formal Dream Characteristics

Finally, I will examine evidence about t[illegible] proposition of the activation-synthesis [illegible]

that bizarre, vivid, visual, hallucinated dreaming occurs only during the D state. This proposition is strongly contradicted by empirical data. Studies from different laboratories have found that dreams for all practical purposes indistinguishable from D state dreams occur in the absence of the D state (26-28). For example, during sleep onset dreaming is very prevalent: 50%-70% of awakenings from certain non-D state, sleep-onset EEG stages result in the report of a dream. When compared with D state dreams, sleep onset dreams are not significantly different in bizarreness, length, manifest sexual content, manifest aggressive content, hedonic tone, or dreamlike quality (27, 28). Furthermore, instigators of sleep onset dreams are more closely related to psychological variables than to physiological variables such as EEG stage (27, 29). Dreaming also occurs frequently throughout the non-D sleep of light sleepers (30).

The finding that dreams similar to D state dreams occur frequently in the absence of the D state has two implications. First, the unique physiology of the D state does not explain the instigation or formal characteristics of the non-D state dreams. Second, the similarity of D state and non-D state dreams makes it unlikely that unique D state neurophysiology determines the characteristics of D state dreams. Rather, the similarity of dreams in different states suggests that some properties common to all the dream states determine dream characteristics. We do not know what these properties might be. One plausible speculation is that forebrain response characteristics during sleep are common to all dream states and forebrain activation sources are unique to the different dream states. That, at least, would be consistent with most of the neurophysiological evidence presented above.

Let me clarify the relationship between activation sources and response characteristics with an analogy. Imagine a town with several different independent sources of electrical power. Each night, the temporospatial pattern of lighting will be independent of the power source but will depend on the usual habits of the town's inhabitants and on their unique needs of that day. The power sources obviously represent the forebrain activation sources, the inhabitant's usual habits represent the neural correlates of personality traits, and the unique day's needs represent the neural correlates of recent experiences. In other words, my suggestion is that state generators—like the D state generator—permit dreaming to occur but do not instigate the actual content or determine its characteristics. These, being similar in all dream states, are functions of the response characteristics of the forebrain. This would suggest that psychological phenomena, which are correlates of forebrain activity, can instigate actual dream content and determine its formal characteristics.

Summary of Empirical Critique

This completes the critical review of the activation-synthesis hypothesis. As a whole, the evidence does not support its three propositions. First, dreaming is not limited to the D state. Second, in the intact animal pontine structures, although necessary for D sleep, are not sufficient to account for the generation of most D sleep episodes. The forebrain is also necessary for the instigation, maintenance, and timing of most D sleep episodes. Hence, contrary to the propositions of the activation-synthesis hypothesis, forebrain neural structures with possible psychological concomitants are involved in the instigation of the D state. Third, the correlational evidence contradicts the claim that the formal characteristics of dreams are related to phasic pontine activation. Thus, the evidence does not support the crux of the hypothesis, that (pontine) physiological processes rather than psychological processes instigate dreams and determine their formal characteristics. Furthermore, such an unsupported neurophysiological theory cannot be a sound refutation of Freud's dream hypothesis.

METHODOLOGICAL CRITIQUE OF THE HYPOTHESIS

I have a final comment about Freud's dream theory that is not related to the soundness of the neurophysiological attack on it. Here, I want to show that purely neurophysiological data—such as that used in the activation-synthesis hypothesis—cannot refute a purely psychological theory such as Freud's.

A psychological theory asserts a reliable relationship among psychological variables. Physiological variables can be used to test this psychological relationship only if the physiological variables are empirically reliable correlates of the relevant psychological variables. Conversely, we cannot know that physiological variable A is not the correlate of psychological variable B until we empirically determine the correlation between A and B. Applied to the particular case of dream theory, we cannot know, for example, that pontine activation of the forebrain is not the neural correlate of emergent, primitive, unconscious wishes until we empirically determine the correlation between pontine activation and emergent, primitive, unconscious wishes. That determination has not been made. Hence, we cannot use the physiological variable, pontine activation, to indicate the absence of unconscious wishes. Put in general terms, neurophysiological variables cannot be used to test a psychological theory of dreams (such as Freud's) when the correlation between the relevant psychological variables and the relevant physiological variables is unknown.

Furthermore, even when the relevant psychophysiological correlations are known, the use of physiological variables to test a psychological theory provides only an indirect test of the theory. A direct test would involve only the psychological variables. In the case of Freud's dream theory, a direct psychological test has yet to be made. In a recent detailed examination of the empirical foundation of Freud's dream the-

ory, I concluded that in principle the theory consisted of a structured set of testable but as yet untested hypotheses (31). That a direct psychological test of a psychological theory is more valid than a psychophysiological test can be seen from the following. If the direct psychological test reliably supported the psychological theory and the psychophysiological test did not, we would conclude that the physiological variables were not valid indicators of the psychological variables. This again supports the conclusion that purely neurophysiological data such as that used by Hobson and McCarley cannot refute a purely psychological theory such as Freud's.

CONCLUSIONS

In summary, Hobson and McCarley have provided us with the first theory that genuinely attempts to integrate the recent discoveries in the neurophysiology of dreaming with the peculiarities of dream mentation. One important implication of this theory is that its neurophysiological foundation appears to refute Freud's hypothesis that psychological phenomena instigate dreams and determine their formal characteristics. However, a critical review of additional neurophysiological and psychophysiological evidence indicates that the new theory is not consistent with a number of findings and cannot be used to refute a psychological theory of dreams such as Freud's.

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The Activation-Synthesis Hypothesis of Dreams: A Theoretical Note

BY ANTHONY L. LABRUZZA, M.D.

The author examines Hobson and McCarley's activation-synthesis hypothesis of dreams from the point of view of theory construction and the logic of science. After reviewing pertinent literature, he concludes that modern science has not yet established a well-defined mind-body isomorphism. Therefore, conclusions about the psychological meaning and motive of dreams cannot validly be drawn from neurobiological data.

RECENT RESEARCH in the neurobiology of dreaming sleep by Hobson and McCarley has led to their proposal of an activation-synthesis hypothesis of the dream process (1, 2). Technical advances have allowed investigators to study desynchronized (D) state control in cats by recording from individual neurons in various parts of the cat brain as the sleep cycle progresses. The result has been an impressive and scholarly enhancement of our knowledge of the neurophysiology of sleep and dreaming. In essence McCarley and Hobson have shown that the D state is generated by autochthonous, periodic activation of pontine generator cells with elaboration of the pontine neuronal stimulus by the perceptual, conceptual, and emotional structures of the forebrain during sleep. Unfortunately, these same authors attempt to extend their extensive neurobiological findings to psychoanalytic dream theory without sound logical justification.

The popular press (3, 4) has capitalized on the activation-synthesis hypothesis to make it appear that Freudian dream theory has been "scientifically" invalidated. It is as if we can now rest easy, for our secret, "repressed" wishes can no longer be discovered in the analysis of our dreams. Such theoretical conclusions are unjustified on the basis of McCarley and Hobson's findings. The purpose of this article is to critique Hobson and McCarley's theorizing and to elaborate the need for careful conceptual, logical, and theoretical analysis in drawing conclusions from neurobiological data. The theory of dreams and dreaming will be taken as a model. I have not attempted an exhaustive discussion or literature review; rather, I will examine some fundamental issues of theory building and evaluation.

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FROM PHYSIOLOGY TO PSYCHOLOGY

Freud was well aware of the difficulties in arguing from principles of brain physiology to principles of psychological functioning. Although he set out in the "Project for a Scientific Psychology" (5) "to furnish a psychology that shall be a natural science," he ran into theoretical difficulty that ultimately compelled him to abandon the project. As Strachey explains, Freud "threw over the whole neurological framework" (6) because the neuronal machinery had no means of accounting for the fact of consciousness. Having abandoned his attempt to develop a physiology of the mind, Freud sought in "The Interpretation of Dreams" (7) to define a purely psychological model of mind based on "analogies" and "giving free rein to our speculations." Freud explicitly stated his point of departure: "I shall carefully avoid the temptation to determine psychical locality in any anatomical fashion. I shall remain upon psychological ground . . ." (7). Implicit in Freud's remarks is his recognition of the fundamental principle that "different levels of scientific explanation may involve essentially different frames of reference, and each level may therefore require a different appropriate language" (8). Peterfreund gives a homely illustration of this principle when he points out that if Tom loves Jane ("love" belonging to the language of persons), one does not say that Tom's brain loves Jane's brain, and so on (8).

McCarley and Hobson ignore this principle when they claim that the primary motive for dream language cannot be disguise because the firing of cells in the pons is motivationally neutral. In their own words, "The *primary motive* for the dream language and dream process cannot be disguise if the prime force of dreams is not an instinct or represented 'wish' in need of disguise" (1, p. 1219). Such faulty logic and conceptual unclarity are surprising in an otherwise carefully reasoned and scholarly report. The authors are precise in defining a dream as "a *mental* experience, occurring in sleep . . ." (2, emphasis added). Nonetheless, they frequently fail to distinguish between the dream state (a neurophysiological process) and dreaming as a mental (subjective) experience. There is no objection to their claim that something is happening in the nervous

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system which corresponds to the mental process of dreaming; but that "something" cannot be described in the language of dream meaning and motive, nor can the psychological purpose of a dream be explained in terms of the firing of cells of the pons or forebrain. In contrast to Hobson and McCarley, Freud was firm in his resolve to allow "psychology the right to account for its most common facts . . . by its own means" (9). Horton suggests that Freud's resolve in this matter had to do with "the epistemological judiciousness of mixing frames of reference, of throwing together, willy-nilly and chaotically, the immediate experience of brain cuttings and pathology slides with the immediate experience of emotions, thoughts, and actions" (10, p. 516).

The net effect of such injudicious mixing of frames of reference is the unwarranted implication that Hobson and McCarley's neurobiological discoveries will logically necessitate major revisions in psychoanalytic dream theory. As previously mentioned, reports in the popular press have already picked up on this implication, claiming that the activation-synthesis hypothesis "discounts Freud's psychoanalytic notion of dreams . . ." (3) and that "if the origin of dreams is adequately accounted for by a physiological mechanism, there is no need to hypothesize a psychological motive for dreaming" (4). Statements such as these are glaring examples of the confusion of independent logical orders and need no further comment.

It is noteworthy, however, that psychoanalytic theorists have long been familiar, implicitly or explicitly, with the notion that, logically considered, repressed wishes cannot physiologically initiate the dream state (8, 10–16). Yankelovich and Barrett (11) traced back almost two decades the theoretical misgivings about Freud's theory of forces as opposed to his theory of meanings. It is true that Freud used the term "wish" to represent both a meaning and a physical force, but no serious modern theoretician would make such a claim. In an odd way, McCarley and Hobson are merely confirming what has been accepted by knowledgeable theoreticians for many years: it is not valid to move freely between independent logical systems of description and explanation. One cannot add apples and oranges without appealing to a superordinate concept such as fruit. Likewise, wishes cannot trigger cells, and cells firing in the pons cannot determine the meaning and motive of dreams.

MIND-BODY ISOMORPHISM AND THEORY CONSTRUCTION

In attempting to address the nature of theory construction, we cannot avoid coming face to face with the perennial mind-body problem. How can we reconcile subjective experience with concomitant objective measurements? McCarley and Hobson have provided us with the physiological concomitants of the subjective experience of dreaming. We have seen how they ran into difficulty when they blurred [illegible] between the distinct logical realms of me[illegible] tive on the one hand and causality on t[illegible] mulations about the meaning and motive [illegible] wish fulfillments (or archetypal images[illegible] serve to order the subjective experien[illegible] cally independent of formulations in [illegible] jective experience is treated objective [illegible] causality of the dream state by pontine [illegible]

In short, there exist two discrete [illegible] dreams and dreaming, the neurobiolo[illegible] chological, the "objective" and the [illegible] These two approaches must be coordinat[illegible] multaneously with the logically indep[illegible] explanation. Thus, mind and brain (or [illegible] be treated as two distinct orders, each [illegible] peculiar language, conceptualizations, [illegible] abstraction; to treat them otherwise [illegible] and conceptual unclarity. There is, as y[illegible] ordinate framework or well-defined [illegible] unites these realms of mind and body [illegible] the history of science, data about the [illegible] functioning of psyche and soma are the [illegible] ance, coincidence, and correlation. Cros[illegible] mon explain that the only way to use the[illegible] ent models simultaneously is

> by coordinating them in their application t[illegible] unity of the phenomena under study. For ev[illegible] chophysiological research we work with [illegible] variants, such as the physiological cor[illegible] ported experience. The coordination is an [illegible] ity, which is not equivalent to prov[illegible] explanation for experience and physiolo[illegible] establish a concordance or parallelism [illegible] something about correlations. Only some [illegible] framework, yet to be discovered, could pr[illegible] cal system in which both aspects could be [illegible] (cf. Langer, 1967). In current dream res[illegible] ample, there is a high correlation betwee[illegible] rapid eye movement sleep (REMs). How[illegible] understanding of the *meaning* of dreams is [illegible] the clarification of the REM mechanism [illegible]

In summary, then, analysis of McCar[illegible] son's attempt to construct a theory of [illegible] dreaming has led to a number of im[illegible] tions. There is, to be sure, a general cons[illegible] psychiatrists that mind and body (or [illegible] tute a functional unity. However, to [illegible] psychosomatic issues one must account [illegible] two logically independent realms. One re[illegible] with mind, meaning, motive, and subje[illegible] ence; the other, with brain, body, causalit[illegible] tive measurement. Each realm has its own [illegible] guage, concepts, levels of abstraction, [illegible] and means of determining veridicality.

Relevant to these concluding remarks [illegible] stein's hypothesis of empirical parallelis[illegible] "every phenomenal event is correlated [illegible] rophysiological event" (14). This notion [illegible] parallelism implies that "statements ab[illegible]

correlated events are also correlated; but they are not translatable to one another" (14). Phenomenal and neurophysiological statements belong to different languages and cannot be translated because translation involves transposing without change of reference a statement in one language to a statement in another. By now the reader should be amply aware of the confusion that arises when Hobson and McCarley attempt to translate statements about cells to statements about dream language without regard for the change of reference involved. Further, there is not currently any way to translate statements about cells and dream motive into a common superordinate language. As Reiser observed, "There is no way to unify the two by translation into a common language, or by reference to a shared conceptual framework, nor are there as yet bridging concepts that could serve, as Bertalanffy suggests, as intermediate templates, isomorphic with both realms" (15, p. 479).

It was McCarley and Hobson's assumption of a mind-body isomorphism "in a less exact sense" (1) that got them into theoretical difficulty. The term "isomorphism" has an exact meaning. Either there is an isomorphism or there is not. To make deductions from an isomorphism in a less exact sense is to try to have it both ways. This approach has no foundation in the logic of science.

If by isomorphism "in a less exact sense" McCarley and Hobson mean the use of "analogies" and what Freud termed "giving free rein to our speculations" (7) to generate hypothetical constructs, then they should also agree with Freud that such methodology is "only intended to assist us in our attempts to make the complications of mental functioning intelligible" (7). To speculate from analogy and evocative metaphor is a time-honored tradition for generating scientific hypotheses, but in no way does it constitute scientific proof or explanation as McCarley and Hobson imply. As it now stands, the activation-synthesis hypothesis of the dream process is an impressive theoretical statement about the neurobiology of the dream state; however, its extrapolation to psychoanalytic dream theory is mere speculation, albeit fascinating speculation, and represents a deviation from basic principles of sound theory construction.

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Comparison of the Incidence and Severity of Extrapyramidal Effects with Fluphenazine Enanthate and Fluphenazine Decanoate

BY JOHN KANE, M.D., FREDERIC QUITKIN, M.D., ARTHUR RIFKIN, M.D., AND DONALD F. KLEIN, M.D.

Forty-nine schizophrenic outpatients stabilized on oral antipsychotic medication and procyclidine received 12.5 mg or 18.75 mg of fluphenazine enanthate or fluphenazine decanoate and were examined for extrapyramidal side effects one and two weeks later. Extrapyramidal side effects were present in 30 patients (61%) but were clinically significant in only 11 (22%). Fluphenazine enanthate produced more clinically significant extrapyramidal symptoms, particularly akathisia, than did fluphenazine decanoate.

A SIGNIFICANT PERCENTAGE of schizophrenic outpatients do not take medication as prescribed. Estimates of noncompliance (1–4) vary from 32% to 63%. Long-acting, injectable phenothiazines offer a partial solution to this problem. However, a major disadvantage associated with fluphenazine enanthate, the first long-acting injectable preparation, is its tendency to produce extrapyramidal side effects (EPS). With the introduction of fluphenazine decanoate it became important to compare these agents for the frequency of side effects. Five studies (5–10) have appeared in the literature contrasting the incidence of EPS with fluphenazine decanoate and fluphenazine enanthate.

Kurland and Richardson (5) compared fluphenazine decanoate and fluphenazine enanthate in 19 chronic schizophrenic patients, who ranged in age from 23 to 53, in a 12-week, random-assignment, double-blind crossover study. No prophylactic antiparkinsonian medication was given. It is not clear whether or how often patients were systematically examined for EPS. Five patients developed evidence of EPS, 3 on fluphenazine enanthate and 2 on fluphenazine decanoate. The authors found no significant difference between these agents in producing EPS. However, it is difficult to interpret this study because of variations in injection intervals and lack of clarity regarding the time in which EPS were assessed.

Keskiner and associates (6) reported on schizophrenic patients (age range, 26–51) who received fluphenazine decanoate at two-week intervals in doses ranging from 2.5 mg to 75 mg. Fluphenazine decanoate treatment lasted from 12 to 78 weeks (average, 42 weeks). Single-blind fluphenazine therapy was replaced by fluphenazine enanthate administered in equal dosage at the same intervals for a maximum of 12 additional weeks.

Patients were rated (7) monthly for extrapyramidal side effects. Thirteen patients required antiparkinsonian medication during the fluphenazine decanoate treatment. If antiparkinsonian medication was given, it was continued throughout the entire study. This procedure confounded any comparison between fluphenazine decanoate and fluphenazine enanthate. Of those receiving fluphenazine enanthate, 2 required an increase in antiparkinsonian medication and 2 required the initiation of antiparkinsonian medication. The authors found no significant difference between the side effects of the two drugs. It is difficult to interpret these findings, however, because all patients received fluphenazine decanoate first and most were started on antiparkinsonian medication, which was then discontinued during fluphenazine enanthate treatment. Despite this, 4 patients had an increase in EPS when switched to fluphenazine enanthate.

Itil and associates (8) completed a double-blind crossover study comparing the effects of fluphenazine enanthate and fluphenazine decanoate on chronic schizophrenics (age range, 25–51). Procyclidine was administered, 10 mg/day, and the dosage was increased if necessary. Side effects were rated on the Treatment Emergent Symptoms Scale (9).

The total dosage of procyclidine and the mean values of side effects were higher for the fluphenazine enanthate group than for the fluphenazine decanoate group but not significantly so. The use of unusually high starting doses (1 mg/kg) limits the generalizability of the findings.

Grosser (10) studied 61 female schizophrenic patients (23 of whom had been in the hospital for more than a year) randomly assigned double-blind to fluphenazine enanthate or fluphenazine decanoate for 4

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weeks. The dosage and injection interval were determined by previous medication levels and clinical criteria. Antiparkinsonian medication was given only on emergence of EPS. Additional major tranquilizers, ECT, and night sedation were administered as necessary. Three of 4 patients requiring additional major tranquilizers were in the fluphenazine enanthate group, but no further details are given.

Grosser found significantly more EPS in the fluphenazine enanthate group. He acknowledged that the baseline level of psychiatric symptomatology was higher in the fluphenazine enanthate group and did not compare dosages. It is possible, therefore, that due to greater baseline psychopathology the fluphenazine enanthate group was placed on higher doses than the fluphenazine decanoate group.

Van Praag and Dols (11) reported on 30 inpatients with various diagnoses in a 4-week double-blind random-assignment study using a single 25-mg injection of fluphenazine decanoate or fluphenazine enanthate. In addition, if a patient's condition warranted it, the treating psychiatrist also administered chlorpromazine in a single-blind fashion. If extrapyramidal side effects were observed, orphenadrine was prescribed openly. It is not clear how or with what frequency patients were examined for EPS. In the first week, orphenadrine was prescribed for 11 of the 17 fluphenazine enanthate patients and 3 of the 16 fluphenazine decanoate patients ($p<.05$). During this period the two groups did not differ significantly in the number of patients requiring supplementary chlorpromazine; therefore, it is unlikely that this could account for the differences in EPS.

A review of the studies comparing the effects of fluphenazine enanthate and fluphenazine decanoate indicates a lower incidence of EPS with fluphenazine decanoate in two studies and no difference in three. Most of these studies have serious methodological flaws. The best designed study (11) reported fewer extrapyramidal side effects with fluphenazine decanoate.

The purpose of our study was to determine if fluphenazine enanthate was more likely than fluphenazine decanoate to produce EPS during the first two weeks after their initiation. Our a priori hypothesis was that fluphenazine enanthate would be more toxic.

METHOD

The subjects in our study were 49 recently discharged schizophrenic patients who were attending an aftercare clinic and were stabilized on oral antipsychotic medication and procyclidine, 15 mg/day. The cohort included 29 men and 20 women, with an age range of 13 to 37. We wished to switch these patients to injectable fluphenazine in preparation for a long-term maintenance medication study and took this opportunity to compare the incidence of EPS with fluphenazine enanthate and fluphenazine decanoate. Informed consent was obtained from all patients.

Treatment Design

Because some earlier reports (10, 12) had indicated a high incidence of EPS with fluphenazine enanthate and fluphenazine decanoate, we began with a conservative regimen: 25 patients were assigned randomly to receive 12.5 mg of either fluphenazine decanoate or fluphenazine enanthate and 2 mg of benztropine mesylate I.M. The other 24 patients received 18.75 mg of either fluphenazine decanoate or fluphenazine enanthate without benztropine mesylate. All of the patients continued to take procyclidine, 15 mg/day, and some patients continued to take a fixed dose of oral antipsychotic medication throughout the study. The decision to continue oral medication was based on clinical judgment. Eleven patients who received fluphenazine decanoate and 13 patients receiving fluphenazine enanthate also took oral antipsychotics. In addition, 3 patients in the fluphenazine enanthate group and 2 in the fluphenazine decanoate group were receiving imipramine at a constant dose throughout the trial and 1 patient in each group was receiving lithium.

There were no significant differences between the groups in average age, sex, or average dosage of concomitant medication.

Evaluation

An examination for extrapyramidal side effects was carried out at baseline, 1 week, and 2 weeks after the initial injection by a physician blind to the type of injection received. All of the patients were examined at baseline. The data were incomplete in 11 patients (22%) due to noncompliance. Ten of these 11 patients missed the 2-week examination. There was no significant difference in compliance between the fluphenazine enanthate and fluphenazine decanoate groups.

Evaluations for extrapyramidal side effects were carried out using 8 items of the Simpson-Angus scale (13): gait, arm dropping, shoulder shaking, elbow rigidity, wrist rigidity, glabella tap, tremor, and salivation, as well as 2 additional items to measure dystonia and akathisia and 2 items to measure akinesia (spontaneity of speech and gestures). Items were rated on a 5-point scale, except the 2 measures of akinesia, which used a 4-point scale.

RESULTS

The results are presented in table 1. Of the 49 patients in the study, 30 (61%) developed detectable EPS. Of these 30 patients, 19 experienced a change of one point on the scale. A change of one on this rating scale is of dubious clinical significance, however; since we were interested in determining if either drug had any potential advantage at all the first statistical comparison involved all manifestation of extrapyramidal side effects, whether of clinical significance or not. No significant difference existed when the drugs were contrasted on the 12 individual items nor between the drug

TABLE 1
Patients Experiencing an Increase in Extrapyramidal Side Effects from Fluphenazine Decanoate or Fluphenazine Enanthate

Group	Number of Patients with Increase in Extrapyramidal Side Effects*				Number of Patients W... Developed Sign ...ic... Akathisia	
	Increase ≥1 Point	p**	Increase ≥2 Points	p		
Fluphenazine enanthate, 12.5 mg (N = 12)	8	n.s.	4	.14	3	
Fluphenazine decanoate, 12.5 mg (N = 13)	7		1		1	
Fluphenazine enanthate, 18.75 mg (N = 14)	9	n.s.	5	.17	4	
Fluphenazine decanoate, 18.75 mg (N = 10)	6		1		0	
All fluphenazine enanthate (N = 26)	17	n.s.	9	.03	7	
All fluphenazine decanoate (N = 23)	13		2		1	

Simpson-Angus scale; 6 extrapyramidal side effects were rated on a 5-point scale and the 2 measures of akinesia were rated on a 4-...
One-tailed FE.

groups when all 12 items were considered together. In addition, no differences were found when the same drug was contrasted at the two different dosage levels.

Eleven patients (2%) had a change of two or more on any one item (a change that might be clinically significant). When only these 11 patients were considered, a difference did emerge. Of the 26 patients receiving fluphenazine enanthate at either dose, 9 patients (35%) developed significant EPS, but only two (9%) of the 23 patients receiving fluphenazine decanoate did (p=.03, one-tailed FE).

Akathisia was the most frequent clinically significant extrapyramidal side effect. Of 26 patients receiving fluphenazine enanthate, 7 (26%) developed significant akathisia whereas 1 (4%) of 23 patients receiving fluphenazine decanoate did (p=.04, one-tailed FE). We used a one-tailed test because our a priori hypothesis, based on the literature, was that fluphenazine enanthate was more likely to produce EPS.

At baseline, 9 patients had ratings of two or more on one item, which indicates potential clinical significance. Five of these patients had their oral antipsychotic medication discontinued or decreased at baseline. None of the EPS noted caused subjective distress or interfered in any way with these patients' functioning. Five of these patients received fluphenazine enanthate and four received fluphenazine decanoate. Only 1 of them experienced a worsening of the item that was present at baseline. Only 2 of the 9 patients (22%) with EPS at baseline experienced any significant new EPS. This is the same percentage that was found among patients with no baseline EPS. This would seem to indicate that mild EPS are not necessarily a contraindication to starting long-acting fluphenazine.

Of the 18 patients who experienced a 1-point change on any item, 4 had baseline scores of 1 on that item and therefore manifested a rating of 2 at some point. These patients were evenly distributed, 2 in each drug group, and were not included among those considered to have developed clinically significant EPS.

Of the 26 patients receiving fluphenazine enanthate, 9 experienced no extrapyramidal side effects during either week of the study, as did 10 of the 23 patients receiving fluphenazine decanoate. There were 13 patients in the fluphenazine enanthate group and 6 in the fluphenazine decanoate group who ha... symptoms during the first week but ... patients receiving fluphenazine ena... tients receiving fluphenazine decan... pyramidal symptoms in the second we... tient in each group experienced ext... effects during both weeks. These dat... phenazine enanthate is more likely to ... pyramidal side effects during the first ... tration than during the second (one-ta... p<.03); this is not so with fluphenazine ...

Twenty-four patients continued to ... stant dose of oral antipsychotic medicat... study. The dose and type of concomit... varied among patients but not w... throughout the study. Because the ... phenazine enanthate or fluphenazine ... random it was anticipated that patients ... with medications with a relatively gre... to produce EPS (those with a piper... would be equally distributed between ... ment groups. This was, in fact, the case

DISCUSSION

Our results indicate a relatively high ... EPS with fluphenazine enanthate and ... decanoate, as has been reported. He... studies have not quantified the severity ... measurements indicate that most are of ... cance. Even in those cases where a cha... more occurred on any one item, the sy... usually transient and none significantly ... the patient's functioning or precluded ... of the same or even larger doses after the ... experience with 300 patients in long-term ... medication studies or in transition for ... only 7% have been unable to continue ta... ing fluphenazine decanoate because of ... side effects.

There are several factors that may ... to the low incidence of functionally signif... served in this population. All patients ... phylactic antiparkinsonian medication, so ...

the occurrence of EPS that it did not prevent. Some studies (14–16) have suggested that the routine use of prophylactic antiparkinsonian medication is of questionable value even with the injectable fluphenazines—a conclusion that we consider yet to be established. Secondly, all patients had previous exposure to antipsychotic medication. The occurrence of EPS might be more frequent in patients without previous exposure.

One half of our patients were receiving concomitant oral antipsychotic medication. One might expect that this would increase the likelihood of developing EPS; however, Chien and Cole (12) report a lower incidence of EPS in patients receiving chlorpromazine and fluphenazine enanthate (33.3%) than in patients receiving fluphenazine enanthate alone (52.6%), although it was not statistically significant.

The fact that we examined patients for only two weeks may have also contributed to our findings. However, Ayd (17) reported that 90% of EPS associated with injectable fluphenazine occur within the first week of starting treatment. Certain EPS, however, take longer to develop. Akinesia, a syndrome characterized by apathy, loss of spontaneity, and psychomotor retardation, was observed by Rifkin and associates (18) to occur after several months of treatment.

The trend for fluphenazine enanthate to produce EPS earlier than fluphenazine decanoate is consistent with the findings of Itil and associates (8), who used quantitative EEG investigations, that fluphenazine enanthate has its maximum effect during the first week of administration, whereas the maximum effect of fluphenazine decanoate is reached in the second week. In addition, EEG analysis indicates that the activity of fluphenazine decanoate is longer than that of fluphenazine enanthate, which is consistent with the clinical findings of Van Praag and Dols (11).

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Severe Depersonalization Treated by Behavior Therapy

BY DEBBIE SOOKMAN, M.A., AND LESLIE SOLYOM, M.D.

The author reports the use of behavior therapy in the treatment of two cases of severe intractable depersonalization. Outcome criteria included full psychiatric assessment, patients' self-ratings, and psychometric test scores. Treatment by flooding was highly effective in one case, and associated obsessive symptoms and anticipatory anxiety were substantially decreased in the other. Behavioral techniques may prove especially helpful in cases in which anticipatory anxiety, phobic avoidance, and obsessive perseveration are exacerbating features.

DEPERSONALIZATION occurs when there is a change in any or in all of the four fundamental aspects of self-awareness (1): 1) activity—an awareness that I am the one who wills my activity; 2) unity—an awareness that at any given moment I am unity; 3) identity—an awareness that I have been the same person all the time, that my past experiences belong to me; and 4) separateness—an awareness of myself. The sine qua non of depersonalization is an awareness of internal and/or external (derealization) change experienced as strange or unreal with the simultaneous experience of the opposite, i.e., that no change has occurred. Insight is not lost.

Depersonalization is frequently reported as a secondary aspect of many psychiatric illnesses, the most common of which are depression (2, 3), phobic anxiety states (4, 5), obsessive neurosis (6), and schizophrenia. It may also be a feature of hysteria (7) and temporal lobe epilepsy (8). However, cases in which the syndrome dominates the clinical picture are relatively rare.

Most episodes of depersonalization are transient (9), may be precipitated by identifiable situations, or be accompanied by a particular feeling state, such as hostility, frustration, anxiety, etc., which may provoke the episode. The few articles available indicate that depersonalization is very resistant to treatment (9–12). Although a mild form of it may respond to supportive psychotherapy, this is rarely effective in severe cases (10). Predominant depersonalization tends not to benefit from the treatments to which the associated symptoms of anxiety and depression respond [illegible] does not respond to tranquilizers, [illegible] anticonvulsants (9), or phenothiazines [illegible] ECT may be helpful in cases with clear[illegible] depression (12), Roth (13), and Sargant [illegible] have reported that ECT may exacerbate [illegible] precipitate depersonalization. In severe [illegible] cases, frontal leucotomy has been advo[illegible]

To the best of our knowledge, this pa[illegible] the first reported use of behavior therap[illegible] ment of two cases of severe depersonal[illegible] patients were incapacitated by daily epis[illegible] depersonalization that had persisted for [illegible] and had shown progressive worsening [illegible] ous previous pharmacologic and psycho[illegible] terventions (which in one case included [illegible] apy techniques of desensitization and fl[illegible]

Both patients were assessed on the fo[illegible] sures: 1) an open-ended psychiatric ques[illegible] ing all symptoms on a 0- to 4-point scal[illegible] tom complex consisted of four to six [illegible] mean of which provided the symptom [illegible] patients' self-assessment on a 0- to 4-[illegible] their depersonalization, obsessive-com[illegible] toms, other neurotic symptoms, and soc[illegible] ment; and 3) psychometric test scores [illegible] Obsessive Inventory (15), Fear Surv[illegible] IPAT Anxiety Scale Questionnaire, a[illegible] Personality Inventory.

CASE REPORTS

Case 1. Ms. A, a 48-year-old housewife, [illegible] treatment after 4 years of psychotherapy, gre[illegible] tranquilizing and antidepressant medicatio[illegible] produce any change in her constantly worse[illegible] depersonalization. She complained of feeli[illegible] ity" about herself and the world, which o[illegible] times daily and lasted from a few minutes t[illegible] with no identifiable triggering stimulus; se[illegible] and moderate social phobia; pervading doub[illegible] her incapable of making any decision; obsess[illegible] about trivia, and clinically unimportant ritual[illegible] ing to check her curtains, stove, door, etc. [illegible] dominantly, a depressive mood characteriz[illegible] of interest, feelings of worthlessness, insom[illegible] trate, diurnal fluctuations, and guilt (specific[illegible] ill).

The episodes of depersonalization had been [illegible] years before therapy by a temporary famil[illegible] then, her condition progressively worsened. [illegible]

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quent and intense attacks of depersonalization, derealization, and panic. During the attacks she experienced marked color, visual, and auditory distortions, desomatization, and autoscopy. On assessment she was incapacitated.

Flooding treatment in fantasy was carried out with the use of grossly exaggerated taped narratives of depersonalization episodes based on Ms. A's description of her anxiety-provoking thoughts and sensorial experiences during the attacks; her own words were used as much as possible. She received 2 1-hour treatment sessions per week for a period of 10 weeks in addition to 8 flooding sessions in imagination for her agoraphobia.

Her depersonalization was reduced by 83% on psychiatric ratings and by 67% on self-rating. During the 8th treatment session Ms. A reported that her depersonalization experience was no longer continuous and that each episode was less intense. By the 20th session, the grossly disturbing depersonalization had been almost completely eliminated. She described her experience now as a feeling of "being aware of myself" or "being removed from the situation," and she seldom felt panicky. She felt she had regained her self-control and no longer avoided shops, buses, etc.

On psychiatric ratings, her phobias, particularly the agoraphobia, were reduced by 67%, her anxiety by 33%, and her depressive symptoms by 36%. There was also a substantial decrease in her ruminations (66.7%), horrific temptations (56%), and doubt (57.1%). Her rituals, originally subclinical, increased slightly.

On self-rating, there was also a substantial decrease in her obsessive-phobic and other neurotic symptoms (54.5% and 69.9%, respectively) and a substantial improvement in her social maladjustment (83.3%), especially in the areas of work, extra-familial activities, and expressed self-satisfaction.

At 1-year follow-up, her improvement was maintained. She occasionally experienced a feeling of self-awareness but without panic. She again became depressed but an exacerbation of the depersonalization experiences did not occur. Phenelzine, 45 mg/day for 5 weeks, terminated this depressive episode.

Case 2. Mr. B, a 40-year-old insurance salesman, was admitted to the hospital in a desperate, agitated state with feelings of unreality lasting from a few seconds to several weeks, the shorter episodes occurring up to 10 times a day. During these episodes, things felt unreal or dreamlike, with frequent time distortions, occasional visual and olfactory distortions and déjà vu experiences. He had obsessive ruminations and doubt surrounding his own state of awareness and intense fears of "going into unreality." He maintained a tortuous self-scrutiny in an effort to "keep myself in a state of awareness." Especially intense were his fears of darkness and all dark objects, including his own closed eyes, which he associated with feeling unreal. There was a daily increase in the number of such feared stimuli. Mr. B had an elaborate and incapacitating system of rituals to "get out of unreality" or as a preventive measure to "stay in reality." In order to undo a time distortion, for example, he would wait until the same time on the following day and recreate the situation right down to the same underwear and tie in order to repeat ritualistically and with absolute awareness the previous day's event in a nondepersonalized state. He was terrified of performing the most basic activities—showering, changing his clothes, brushing his teeth, etc.—and on admission was unable to do so.

Other less disturbing obsessive symptoms included a host of horrific temptations, obsessive fears, and all-pervading doubt and indecision. However, Mr. B showed no depressive features and no phobias other than the fear of darkness. One unusual feature was that when he was not depersonalized or ritualistic, he showed an unusually gregarious, friendly, and easy-going personality with very few obsessive personality traits.

Over a 2½-month period, 5 1-hour treatment sessions per week and several prolonged (2- to 3-hour) flooding treatments were given. During the flooding treatment, carried out mostly in vivo, Mr. B was exposed to the situations that provoked his depersonalization experiences, encompassing a large number of stimuli in random patterns to prevent habituation. Some paradoxical intention was employed when he was encouraged to say his dreaded "obscene" thoughts aloud or to try to induce a depersonalization episode.

Mr. B was strongly urged never to perform a ritual to terminate his feelings of depersonalization. In view of the nature of his rituals, self-monitoring was instituted to encourage complete response prevention, since 24-hour supervision was not possible. For his many ruminations a thought-stopping technique—a portable finger-shock apparatus—was used, first administered by the therapist and then by Mr. B during actual depersonalization episodes.

Within the first week of therapy, Mr. B began to extend his activities to include those situations to which he had been exposed during therapy. The flooding treatment seemed to have had very little therapeutic effect on the depersonalization. It was our impression that the episodes were reduced somewhat in frequency and in intensity, but Mr. B did not agree. However, the associated rituals were reduced by 63%. He reported feeling fewer urges to perform his rituals following a depersonalization episode, and the rituals he did perform (e.g., blinking) lasted a few seconds instead of hours. His avoidance behavior was markedly reduced. There was also a 50% reduction in his horrific temptations but only a 25% improvement in his ruminations and pervading doubt. Of greatest importance on the Leyton Obsessive Inventory was a 55% reduction in the interference score. Although the number of obsessive symptoms was only moderately reduced by treatment, their duration and intensity were reduced such that they interfered only half as much as previously.

Mr. B also considered his obsessive and other neurotic symptoms, such as anxiety, to be reduced by over 50%. He rated himself as substantially improved (63%) in all areas of work, leisure, and sexual performance. On returning home, Mr. B was able to resume his work, and at 6-month follow-up he had maintained his progress.

DISCUSSION

Two patients with a main complaint of depersonalization were treated with behavior therapy: In both patients, the depersonalization syndrome was long-standing and highly distressing and previously had shown no response to psychotherapeutic and pharmacologic intervention. However, the personalities of the patients and the other associated symptoms were quite different. In the first patient (Ms. A), depersonalization was connected with severe agoraphobia and mild obsessive symptoms and was not stimulus bound. Ms. A had high anxiety, high neuroticism, and low extraversion scores. This symptom constellation is simi-

lar to Roth's phobic anxiety depersonalization syndrome both in type of symptoms and in personality attributes. In the second case (Mr. B), the depersonalization ruminations, provoked by an almost infinite number of stimuli, led to obsessive rituals. The anxiety, phobia, and neuroticism scores were relatively low; the extraversion score was high. This is a case of long-standing severe obsessive neurosis with atypical personality characteristics. Thus, of the most common psychiatric conditions in which depersonalization is a clinically important feature—phobias, depression, obsessions, and schizophrenia—all but schizophrenia are represented.

The different outcomes of treatment—improvement in depersonalization versus improvement in rituals—may have been due to differences in the treated symptoms as well as to the different personalities and total symptoms of the patients. Flooding in imagination (case 1) proved to be more efficacious than flooding in vivo (case 2) in ameliorating the intensity and severity of the depersonalization episode. The salient factor may be that depersonalization is itself an experience of the imagination and is therefore more effectively treated in imagination.

Despite residual disabilities, both patients were satisfied with the results of treatment. Nevertheless, there remain some reservations about long-term results, as both patients still manifested fairly high posttreatment anxiety scores that may make them vulnerable to relapse.

Follow-up and further clinical application are needed to elucidate the indication for and long-term benefit to be derived from the use of behavior therapy in the treatment of depersonalization. Behavioral techniques may prove to be especially helpful in cases in which anticipatory anxiety, phobic avoid[illegible] sessive perseveration are exacerbating f[illegible]

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Neurobiological Vulnerability to Low-Dose Amphetamine Psychosis

BY MARK S. GOLD, M.D., AND MALCOLM B. BOWERS, JR., M.D.

The authors present a case to illustrate a model of drug-induced psychosis wherein the drug acts as if it fits with some neurobiological predisposition in the patient. Patients with a good interpersonal developmental history but extreme sensitivity to a psychotomimetic drug may provide a model for neurobiological vulnerability to psychosis.

THE WELL-DOCUMENTED clinical similarity between amphetamine-induced and naturally occurring paranoid psychosis (1–7) has led to the proposition that amphetamine produces a model psychosis resembling paranoid schizophrenia in most respects. Support for this model psychosis has come from numerous studies that have been reviewed elsewhere (8–10). These studies have linked antipsychotic drug efficacy, amphetamine stereotypy, behavioral effects, and pharmacologically significant actions of amphetamine to alterations in dopaminergic activity.

Amphetamine psychosis has been produced in volunteer subjects by large cumulative doses of amphetamine (5, 6, 11). An amphetamine psychosis produced by cumulative administration of amphetamine is not a toxic psychosis in the usual sense. However, in another respect this syndrome is "toxic" in that it is limited by the presence of the drug in the body. At very low doses, amphetamine-like compounds can produce psychotic states resembling naturally occurring paranoid psychosis (1). The "low-dose" or amphetamine-precipitated psychosis may continue after the drug effect has dissipated (12–15). It is this low-dose or drug-precipitated reaction that we believe may be more relevant as a model of clinical psychopathology, i.e., a lock-and-key model wherein the drug acts as if it "fits" with some predisposition in the patient. Furthermore, in this low-dose model neither the drug nor the individual contribution is overriding. Both the drug and the individual vulnerability are required.

The model we are using is a component model of psychopathology. With regard to psychotic states, the primary components of this model are vulnerability to the psychotic state and the interpersonal developmental level of the individual. The question of drug-induced models of psychosis may be best scrutinized with this component model in mind. Such a model would predict that vulnerability to psychotic states and interpersonal developmental deficit are separable aspects of psychotic illnesses. It might be possible to isolate this vulnerability, which is unmasked by the provocative drug stimulus, in an individual who has a good interpersonal developmental history but is sensitive to a psychotomimetic drug. In this model the patient carrying such a vulnerability might not develop a prolonged psychosis without the administration of the drug.

To illustrate we will describe a patient with an exquisite sensitivity to amphetamine-precipitated psychosis in conjunction with a history of good interpersonal development. The case is presented as an example of the low-dose psychotomimetic phenomenon and an approach for studying the neurobiology of vulnerability to psychosis as a component of psychotic states.

CASE REPORT

Ms. A, a single 20-year-old woman, was living alone in an apartment at considerable distance from her parents. All of her brothers and sisters had moved out of the home after completing high school, continued their educational or vocational training, and eventually married and had children. Ms. A was the youngest of the four siblings. She was admitted to the Yale-New Haven Hospital Emergency Room and was referred to the clinical research unit with a diagnosis of amphetamine psychosis or schizophrenia. (Urine and blood toxicology tests at the time of admission were subsequently found to be negative.) Neither she nor any member of her family had any previous psychiatric contact, medication, or hospitalization. Three weeks before admission, Ms. A had planned a long drive for a ski weekend. After "a long day at work" she was tired and "borrowed" five 5-mg dextroamphetamine (Dexedrine) tablets from a friend who was under treatment for narcolepsy. She took 5 mg at the beginning of her drive, another 10 mg 3 hours later, and then another 10 mg at daybreak. No further amphetamine was ingested. She describes feeling hyperalert and vigilant throughout her drive but on arriving at the resort felt that she "had died and someone else was in my place." She described intense anxiety, suspiciousness, ideas of reference, hyperactivity, and dysphoria. She was so uncomfortable that she cut her vacation short and returned home. She went to work that next

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Monday but felt that the "F.B.I. had agents watching me." She withdrew from her friends and family, feeling that they were imposters, and spent hours in a noncommunicative state. She demanded that her mother prove her "true" identity and accused her of being an F.B.I. agent. When her mother removed her false teeth as proof, Ms. A concluded that her mother was "a very good imposter but not my mother" (Capgras phenomenon). She began having auditory hallucinations and felt that she was Patty Hearst. She pleaded with her family not to "throw me in the closet." She had vivid dreamlike visual hallucinations, and her family described her as "hyper and always picking at her clothes." She tried to stay at home, but it became impossible when "I felt convinced that the apartment was bugged." She went to live with a sibling who, upon learning of the "drug abuse" and observing bizarre stereotyped behavior, increasing social withdrawal, and communication that at times was incoherent, brought her to the Yale-New Haven Emergency Room for treatment. Her interpersonal developmental level appeared good, and she had a record of age-appropriate adaptive behavior in her sexual adjustment, work, social adjustment, childhood development, and school performance. Interviews with her family members, teachers, employers, and friends failed to document pathological premorbid traits or behaviors. Medical and neurological evaluation was normal on admission.

Evaluation of this patient included measurement of probenecid-induced accumulation of the cerebrospinal fluid serotonin and dopamine metabolites, 5-hydroxyindoleacetic acid (5-HIAA) and homovanillic acid (HVA), respectively, as described by several investigators (16, 17), and serum prolactin, an accessible marker of tuberoinfundibular dopaminergic activity (18). We were looking for evidence of central dopaminergic overactivity in Ms. A before she began pharmacological treatment. During the second week in the hospital (when symptoms were minimal) values for 5-HIAA, HVA, and probenecid were 130 ng/ml, 145 ng/ml, and 17 μg/ml, respectively. During a subsequent drug-free, essentially asymptomatic period serum prolactin was 18 ng/ml. During treatment with chlorpromazine (500 mg/day, dose achieved after 21 days), CSF values for 5-HIAA, HVA, and probenecid were 67 ng/ml, 274 ng/ml, and 25 μb/ml, respectively. Medication was later changed to thiothixene (10 mg/day) at which time serum prolactin was 38 ng/ml.

Ms. A's motor stereotypies, hallucinations, and delusional thinking cleared in sequence. Visual and auditory hallucinations abated 8 days after admission and were followed by a marked decrease in delusional content over the next 72 hours. As she improved, Ms. A began to attend a discharge group and to take passes outside the hospital. After taking a number of these passes and seemingly doing well, Ms. A became quite disorganized and manifested a number of the symptoms seen on her admission. Urine and serum toxicology was repeated and again found to be normal. The EEG was abnormal, with prominent left temporal runs of sharp and spike activity. The EEG became normal on clinical remission 10 days later. After discharge, Ms. A returned to her level of premorbid functioning and was asymptomatic at 1-year follow-up.

DISCUSSION

Cases of psychosis following ingestion of low doses of amphetamine can be found in the literature dating back to the original description of amph[illegible] chosis by Young and Scoville (19). These [illegible] sented a case of paranoid psychosis that [illegible] over 1 year and led them to wonder abo[illegible] innate vulnerability in such a drug reactio[illegible] anecdotal reports suggest that even a [illegible] venous administration of amphetamine-l[illegible] can rapidly provoke or intensify psycho[illegible] in so-called "psychotic" or "borderline" [illegible] occasionally in "normal" individuals. M[illegible] experimental studies suggest that small [illegible] phetamine can temporarily intensify psy[illegible] tomatology in schizophrenic patients (7[illegible] nately, too often amphetamine psycho[illegible] conceptualized as being associated with [illegible] use of amphetamine and clearing within [illegible] after the urine no longer contained ar[illegible] those whose symptoms continued wer[illegible] have probable or latent schizophrenia [illegible] this post hoc argument is the belief [illegible] schizophrenic state can be characterize[illegible] nized in some reproducible way. F[illegible] sistence of psychotic symptoms in drug-[illegible] chotic states has not been found to correl[illegible] premorbid function, symptomatology or [illegible] history, or outcome (15, 17).

Vulnerability to prolonged psychotic s[illegible] by low doses of psychotomimetic drugs [illegible] sarily correlated with the level of interper[illegible] opment. Ms. A had premorbid characteris[illegible] from those usually reported for patients w[illegible] a remitting paranoid state or psychosis s[illegible] cumulative doses of amphetamine (5) an[illegible] regularly reported in schizotypic individ[illegible] tion to being precipitated by a low dose [illegible] mine, Ms. A's symptoms persisted fa[illegible] riod of acute drug effect. This amphetam[illegible] in conjunction with good premorbid fun[illegible] gests a neurochemical vulnerability tha[illegible] compensated for but that can be overrid[illegible] ence of catecholaminergic agonists like a[illegible] In this patient the vulnerability to psych[illegible] associated with premorbid "trait" deficits.

In this case of exquisite vulnerability [illegible] mine-induced psychosis, the question tha[illegible] the nature of such sensitivity. As opposed [illegible] dose psychotomimetic reaction in which [illegible] put is dominant, we must ask what pat[illegible] variables led to this low-dose, prolonged [illegible] amphetamine. Pharmacological data [illegible] phetamine and potent antipsychotic me[illegible] dopaminergic systems and literature on am[illegible] exacerbated psychoses support the role o[illegible] gic hyperactivity or supersensitivity in the [illegible] sis of some paranoid psychoses (9, 10). [illegible] response to a low dose of amphetamine [illegible] sisted for more than 1 month after withd[illegible] precipitating agent, could be compatible [illegible] mine hypothesis. One might postulate tha[illegible] ence of an amphetamine challenge the "[illegible]

nerability might allow for the development of persistent central dopaminergic overactivity and a paranoid state.

Our biochemical measures do not provide definitive information on these points. The CSF HVA value obtained when Ms. A was minimally symptomatic and unmedicated was certainly not elevated (17). The serum prolactin level measured at another asymptomatic, drug-free period was not low. These findings indicate that there was no generalized process at work in this individual which led to increased transmission in the nigrostriatal system (HVA) and/or increased dopaminergic inhibitory pressure on prolactin secretion. That both these systems are functionally intact is demonstrated by the expected responses to antipsychotic drugs in both systems, i.e., an increase in CSF HVA and serum prolactin. It is conceivable that other central dopaminergic systems (possibly limbic) currently inaccessible to direct clinical assessment may contain sites uniquely vulnerable to amphetamine. In this regard, the EEG findings in our patient are interesting. Stevens (21) has reviewed the evidence for the limbic system's involvement in the anatomy of psychosis. It is conceivable that the temporal spike activity in this instance is a reflection of limbic irritability relevant to amphetamine sensitivity and vulnerability to psychosis. Further investigations of patients with a good interpersonal developmental history but extreme sensitivity to psychotomimetic drugs are necessary to determine whether they provide a model for assessing the predictions of the dopamine hypothesis and dissecting out vulnerability to psychosis.

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Enuresis Treatment with Imipramine Hydrochloride: A 10-Y Follow-Up Study

BY PAUL M. BINDELGLAS, M.D., AND GEORGE DEE, PH.D.

The authors followed 29 young adults who had been treated for enuresis with imipramine hydrochloride 10 years earlier. To test beliefs that enuresis is symptomatic of severe psychopathology or of urological conditions, they studied whether the treatment had been followed by psychological decompensation; an inhibition of learning; a predisposition to drug abuse; negative effects on health, growth, weight, and development; or continued urinary symptoms. None of these negative effects was present; in general the subjects were active, well-motivated, and sociable and showed no significant psychiatric symptoms. One patient still wetted but only occasionally.

THERE IS considerable controversy about the use of medication for treating children, particularly when it is for the treatment of enuresis. Fear has been expressed that the long-term effects of medication for enuretic children would lead to 1) psychological decompensation, 2) inhibition of learning, 3) predisposition to abuse drugs as the child grows older, and 4) deleterious effects on health, growth, weight, and development.

Some believe that wetting is a symptom of severe psychopathology in the child and the family and that the removal of this symptom leaves the pathology intact and may result in further psychological decompensation and more severe symptomatology (1).

After reviewing reports by MacLean (2) and others (3) on the positive results of imipramine hydrochloride in the treatment of enuresis, we began a controlled study of its effects on enuretic children whose parents responded to a newspaper article about the study (4). Response to the medication was generally favorable, and a 2-year follow-up indicated that the fears of symptom removal were groundless [illegible] (5). However, the children were still [illegible] to evaluate the long-term effects on [illegible] adults, so 10 years after the start of [illegible] tempted to contact 44 of the children [illegible] pleted our original study to evaluate [illegible] fects of imipramine hydrochloride [illegible] dren.

Revised version of a paper presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2-6, 1977.

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The authors wish to thank Geigy Pharmaceuticals for contributing data about their original study and for supplying technical help and the entire staff of the Jane Wayland Child Center for assistance in this study.

METHOD

We developed a telephone contac [illegible] about the subject's adjustment to sc [illegible] and family; drug, tobacco, or alcoho [illegible] traits; illnesses or accidents; and he [illegible] history of bedwetting. The original fa [illegible] subject were available from the earli [illegible] cluded the subjects' addresses, [illegible] schools, the age at which bedwetting st [illegible] of night that the wetting occurred, th [illegible] bedwetting, the degree of bedwetting [illegible] the family who were bedwetters, the [illegible] of bedwetting, and the child's heigh [illegible] patients' responses to the treatmen [illegible] study were also available.

Due to the long time that had elapse [illegible] tion of the population, only 29 famil [illegible] tacted. Subjects were found throug [illegible] States; 2 were overseas. Patients' ag [illegible] to 25 years (mean, 19 years).

A psychiatrist (P.M.B.) interview [illegible] completed the Hamilton Depression R [illegible] for each of them. A psychologist (G. [illegible] ject's height and weight, administered [illegible] BM from the Thematic Apperceptio [illegible] and the patient completed the Zung Se [illegible] for Depression (8). An additional con [illegible] with 17 families 6 to 8 months after th [illegible] view to check on the constancy of res[illegible]

RESULTS

To measure possible psychologica[illegible] tion, a comparison of emotional and [illegible] lems before and after the original stu[illegible] ing responses to questions asked in the [illegible] terview, the descriptive adjectives c[illegible]

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mother to describe the subject, and data from the original study.

The number of subjects who displayed psychological or emotional symptomatology of any significance was small. We subdivided patients into those who had problems before the study and those who had problems after the study. Eight subjects were reported to have had problems before the study. Four of them indicated they were having problems at the time of follow-up, as did 1 patient who had no reported emotional problems 10 years before. This subject was functioning adequately at home and at school, but his mother considered him "emotional."

In general, the subjects were active, well motivated, and sociable and showed no significant depressive symptomatology, anxiety, or other psychological-emotional problems.

The essential lack of psychological problems was confirmed by the sampling psychiatric and psychological interview that was given to 9 patients. Testing with the TAT pictures, the Hamilton scale, and the Zung Self-Rating Scale for Depression, and the impressions of the psychologist and psychiatrist indicated this same conclusion. An evaluation was completed on these 9 patients using the findings of the original and current studies, and there were no indications that psychological decompensation or more severe psychiatric symtomatology occurred with the removal of the symptoms of enuresis.

School and Work

Of the 29 subjects contacted, 15 were still in school; most of those who were out of school had finished high school and were working, and the few who had not finished high school were all working. The 2 patients who were not in school or working had completed high school the summer before contact. Many subjects were involved in various extracurricular activities in addition to their studies.

While in school, 10 subjects were in the upper third, 12 were in the middle third, and 7 were in the lower third of their class academically. We found no evidence to support the assumption of an inhibition in learning.

Substance Abuse

That using drugs as a child would lead to a predisposition to use drugs is based on the assumption that the subjects would abuse drugs, particularly pills, because of a psychological reliance. We distinguished marijuana use from other drug use and added the use of alcohol and tobacco. Marijuana use, which includes having tried it but not using it regularly, has been estimated to be 20% or higher locally for the age range studied (9).

We asked the subjects to rate their use of marijuana, other drugs, alcohol, and tobacco as "frequent," "some," or "never"; the "some" category covers those who have tried or used a substance occasionally but does not include regular users. Two individuals reported "some" use of marijuana; none reported using it frequently, and 27 said they had never tried it. Of the 2 who did use it, the use was extremely light. Ten of the 29 had used tobacco, but only 5 reported being frequent users. Thirteen reported using alcohol, but only 2 reported fairly frequent use. No family members or patients reported any other form of illicit drug use. As can be seen, this group had remarkably little contact with drugs.

Physical Growth and Development

The original study included data on height, weight, and physical health, and the follow-up also covered these areas. Using Public Health Service height-weight statistics (10) and tables developed by the Metropolitan Life Insurance Company (11), we rated each subject as above average, average, or below average for height and weight at the time of the original study and at follow-up.

The majority of subjects maintained their same growth position in regard to height and weight; 20 maintained their height status, and 23 maintained their relative weight. Six subjects had increased and 3 had decreased their relative height, and 5 had increased and 1 had decreased their relative weight. On the whole this was a fairly well-functioning, healthy group of young adults that was not being significantly hampered by any health problems. There is no evidence to support the assumption of a deleterious effect on health or growth development.

Consistency of Responses

In addition to the 9 subjects who were interviewed by a psychiatrist and psychologist, 17 families were recontacted after 6–8 months to check on the constancy of responses. The responses did not change, which supports the reliability of the telephone interviews.

Urological Symptoms

Arnold and Ginzberg (12, 13) and Mahoney and associates (14, 15) wrote about the high incidence of urogenital symptomatology in enuretic children. They felt that the treatment of choice for most of these children would be urological procedures and that if such procedures were not done, even though many of these children would eventually outgrow their enuresis, the other urinary symptoms such as urgency, frequency, and burning would persist. Therefore, we attempted to check out these contentions. We reviewed the original records of the 29 children in the follow-up; 12 subjects had other urinary symptoms such as frequency and urgency. Four of them had daytime wetting. During our follow-up we inquired as to whether there was any persistence of enuresis and urinary frequency or urgency.

Of the 29 patients, all but 1 reported that they no longer wetted and the 1 patient wetted only occasionally. This patient reported still having symptoms of frequency and urgency. The other 11 who had reported urinary symptoms in the original study were free of urinary symptoms at follow-up. One may speculate

that, just as a psychiatrist or mental health clinic treats many enuretic children with a high incidence of emotional pathology, a urologist may receive a high incidence of referrals with signs indicative of urinary pathology, with the result that both groups tend to get a distorted view of the etiological factors in enuresis.

The results of the 10-year follow-up study support the findings and observations we made at the end of 2 years (5). We again found imipramine hydrochloride to be safe and effective in the treatment of enuretic children. The mere removal of symptoms produced no psychological complications but, rather, had a healthy effect on the child and his or her family.

DISCUSSION

The prevailing view has been that enuresis is indicative of severe psychological and family problems, and, just as with hysteria, if one removes the symptom without treating the underlying cause there is the possibility that the patient will experience further psychological decompensation and even more severe psychopathology. However, most studies that have reported a high incidence of emotional problems in enuretic children have come from child guidance clinics, where one would normally expect to find a high incidence of psychopathology (16). In our original study, in which our patients came in response to a newspaper article about our investigation of the use of imipramine hydrochloride in enuresis, we did not find a high incidence of psychopathology.

Arnold and Ginzberg (12, 13) and Mahoney and associates (14, 15) reported finding many enuretic children who suffered urological conditions. (Arnold and Mahoney are both urologists.) Others have reported small bladder capacity (17), neurological problems (18), and allergic disorders (19, 20) that cause bladder irritability and contraction of bladder size. In addition, there are enuretic children who become enuretic following stressful events in their lives and there are many who are psychologically immature and have severe psychopathology. Thus, we believe that enuresis is a symptom, like a cough, which can have multiple and varied etiologies. We also believe that just because an enuretic child has concomitant emotional problems, one should not assume without further evidence that the enuresis is caused by the emotional problems. We have found that many enuretic children with severe concomitant psychopathology respond well to imipramine hydrochloride and show no evidence of psychological decompensation. In a few instances where for psychological reasons either the child or the family has an emotional need for the enuretic symptom to persist, treatment is discontinued or the child will start wetting again in spite of treatment.

We used the data from the original study (5) to compare and contrast changes, but we also asked the respondents for their memory of the original study. There was a discrepancy between our records and what the parents and the youngster remembered about the effects of the medication, but the discrepancy was greater for those who were contacted twice were closer [illegible] general, it would appear that only when the medication had an immediate and dramatic effect was there a tendency to remember that the medication had a positive effect. Thus, 3 mothers who had been told by the medication indicated that their children stopped wetting almost immediately and were parents of children who were on medication at the beginning of the double-blind part of the study. None of the parents of children who started on placebo had such a reason for the medication. Some parents formed their judgment on the basis of time sequence. When the cause and effect between the medication and the enuresis—wetting was slow and less dramatic, they tended to give the medication less credit and to assume the enuresis stopped because of other reasons.

In general, both parents and patients seemed to consider enuresis something to be ashamed of and often denied it. Thus, although cooperative, most parents made remarks that indicated they wanted to protect the patient from embarrassment at having been the enuretic child. In general, the patients themselves had very hazy recollections of being enuretic, having stopped, or of even having been in the study.

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Sexual Function and Affect in Parkinsonian Men Treated with L-Dopa

BY EDWARD BROWN, M.D., GREGORY M. BROWN, M.D., PH.D., OSCAR KOFMAN, M.D., AND BRUCE QUARRINGTON, PH.D.

Using psychiatric interviews, sexual and affect rating scales, hormonal studies, and neurologic assessment, the authors assessed the effect of L-*dopa treatment on men with Parkinson's disease. Patients demonstrated variable affect changes. Approximately one-half of the patients reported an increased sexual interest that was not related to improvement in locomotor function. Hormonal factors appeared to be involved. The findings suggest that male parkinsonian patients who possess an intact hypothalamic-pituitary-gonadal axis experience increased sexual function related to* L-*dopa treatment.*

THE USE of L-dopa in the treatment of Parkinson's syndrome has been reported to correlate with alterations in affect (1-6) and sexual function (1-5, 7, 8). It remained to be determined if these alterations were related to drug effect or placebo response or were secondary to the reduction of locomotor impairment. In this study the effect of L-dopa therapy was assessed in men with Parkinson's disease by using psychiatric interviews, sexual and affect rating scales, hormonal studies, and neurologic assessment.

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METHOD

The subjects were 7 men with Parkinson's disease who ranged in age from 59 to 69 years (mean=62). They had been ill for 1-7 years (mean=4). All patients were receiving the standard antiparkinsonian medications, benztropine mesylate and trihexyphenidyl HCl, but they had not received L-dopa before this study. Their dosages of benztropine and trihexyphenidyl were unchanged throughout the study.

At the beginning of the study, each patient received a thorough physical examination as well as a psychiatric assessment. Neurologic status was assessed at intervals. Each patient was asked to complete self-administered sexual interest and affect questionnaires each day at the same time.

On the sexual interest rating scale, each patient was asked to give a single numerical response (1 to 9) that corresponded to a specific question reflecting drive-intensity (e.g., 1=no interest in sexual matters, 5=some things are of sexual interest, 9=readily excited sexually). In addition, each patient was asked to respond to questions about specific behaviors (e.g., masturbation, intercourse without ejaculation, intercourse with ejaculation); responses tended to confirm the validity of the sexual interest rating scale. On the affect rating scale, each patient was asked to give a single numerical response (1 to 9) that corresponded to a specific question reflecting affect (e.g., 1=extremely unhappy, 5=not happy or unhappy, 9=extremely happy). Patients were also asked to respond to questions regarding specific behaviors (e.g., feeling like crying, hopelessness, energy level, wanting to be alone); again, results tended to confirm the validity of the rating scale. Mean values were calculated for each patient on the various measures at each phase of the study (baseline, placebo, stable L-dopa dose). Findings were then subjected to analysis of variance.

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Plasma luteinizing hormone (LH) and prolactin[1] were measured by radioimmunoassay with methods and materials supplied by the National Institute of Arthritis, Metabolism, and Digestive Diseases. Blood specimens were collected weekly in the early morning of the same day each week.

The following sequence was followed for each patient in a single-blind method: period 1 (baseline)—3 weeks in which no placebo or L-dopa was given; period 2 (placebo)—3 weeks in which placebo was given; and period 3 (L-dopa treatment)—a period of changing L-dopa dosage, followed by a period of stable L-dopa dosage (3–18 weeks).

RESULTS

Psychiatric Interviews

The psychiatric assessments were done before the baseline study period. Patient 2 failed to complete the psychiatric assessment but continued to participate in all other aspects of the study. Patient 3 exhibited moderate anxiety symptoms with some depression. Patients 5, 6, and 7 were moderately depressed. Patients 1 and 4 were within the normal range of affect.

The patients fell into three categories of sexual function. Patients 3 and 4 reported marked reduction in sexual desire and erectile and ejaculatory capability that preceded the onset of Parkinson's disease. Patients 1, 6, and 7 reported a marked reduction in sexual desire and erectile and ejaculatory capability that coincided with their parkinsonian symptoms. Patients 2 and 5 reported a satisfactory level of sexual desire and sexual potency, which did not change with the onset and progression of their symptoms.

Sexual Interest and Sexual Activity Ratings

Patient 1 had a significant increase in the sexual interest rating during the drug period ($p<.001$), but his masturbation frequency showed a nonsignificant decrease during this period. His frequency of intercourse showed little change.

Patient 2 showed a significant increase in sexual interest ($p<.001$) during the placebo period but not during L-dopa administration. However, his intercourse frequency decreased during the placebo period and rose markedly ($p<.004$) with L-dopa.

Patient 3 showed a decrease in sexual interest ($p<.001$) during the L-dopa period. This patient developed side effects that necessitated a number of dosage changes. He reported no sexual activity throughout the study.

Patient 4 showed a progressive decrease in sexual interest throughout the study ($p<.001$) and reported no sexual activity.

Patient 5 showed little change in sexual interest. He reported no masturbatory activity until he began to receive L-dopa. The Fisher exact test comp[illegible] batory frequency in the placebo pe[illegible] stable drug dosage period showed no [illegible] ference. This was probably due to the fa[illegible] cebo period was only 20 days long. W[illegible] and placebo period combined were c[illegible] period of stable drug dosage, the diffe[illegible] cant ($p<.01$).

Patient 6 showed a marked increase [illegible] ual interest through the placebo perio[illegible] period. This increase could not be [illegible] drug alone since it appeared during th[illegible]

Patient 7 showed no changes in any [illegible] throughout the study.

Thus, 2 patients showed evidence [illegible] ual functioning that was probably rel[illegible] patients showed increased sexual in[illegible] be attributed in part to the drug but in [illegible] tors as well, 2 showed reduced sexu[illegible] showed no change. In patient 3, [illegible] seemed to be related to L-dopa, pro[illegible] verse side effects. The reduced sexua[illegible] ed by patient 4 was part of a trend thr[illegible] od in which he was studied. Factor[illegible] drug were operating, perhaps as part [illegible] thexis related to the subject's chronic [illegible] to which he succumbed a few month[illegible] period.

Affect Ratings

Mean values for affect for each of t[illegible] periods were determined for each pati[illegible] termined the mean values (±1.5 SD) f[illegible] control subjects (men aged 57–67 in good [illegible] with no psychiatric history).

Patient 2 showed a baseline affect s[illegible] mal limits. During the placebo period, th[illegible] nificant increase in his affect ratings th[illegible] tained during the period of stable dr[illegible] patient therefore appeared to have a [illegible] sponse to placebo.

Patients 3, 5, and 6 showed baseli[illegible] ratings at or below the control va[illegible] showed no significant changes with [illegible] Patient 5 improved significantly to ne[illegible] ing L-dopa administration. Patient 6 sho[illegible] cant level of improvement with placeb[illegible]

Patients 1, 4, and 7 showed no sig[illegible] over the three periods and all values w[illegible] mal limits.

Statistical analysis (Pearson correlat[illegible] of data for all patients as a group duri[illegible] L-dopa treatment indicated a significan[illegible] tive correlation between affect a[illegible] ($r=.1716$, $p<.002$).

Pearson correlation coefficients bet[illegible] terest and affect scores were calculate[illegible] a group for the combined baseline and [illegible] ods, the period of L-dopa treatment, and [illegible] ods combined. Sexual interest was sign[illegible]

[1]Data were insufficient for analysis of prolactin changes.

to the affect score only for the combined study periods ($r=.73$, $p<.001$).

The data suggest that affective state did not play a consistent role in determining sexual interest, particularly in subject 1, who showed an increase in sexual interest without a corresponding change in affect.

Neurologic Findings

Patient 1 had tremor with very mild rigidity that improved with treatment. With the exception of minimal residual tremor, his pattern returned to a relatively normal state.

Patient 2 had significant tremor in the right arm with very slight rigidity. With L-dopa treatment the tremor improved somewhat but did not cease. There was no alteration in the mild rigidity.

Patient 3 initially had mild to moderate bradykinesia with very slight tremor and mild rigidity. The bradykinesia improved somewhat but was still present. The rigidity and tremor were unchanged.

Patient 4, who had bradykinesia, tremor, and moderate rigidity, showed little essential change with L-dopa treatment.

Patient 5 had mild bradykinesia, significant tremor in the left arm, and a very slight tremor in the right arm. There was mild rigidity in the left arm. Following treatment there was considerable improvement in the tremor, with mild residual tremor in the left arm only. The bradykinesia improved substantially.

Patient 6 had moderate bradykinesia and moderate tremor, particularly in the left arm and leg. The severity of these symptoms was mild after treatment.

Patient 7 had pronounced tremor and mild rigidity in the left arm, slight tremor in the right arm, and mild bradykinesia. With treatment the bradykinesia disappeared; tremor and ridigity persisted but were less pronounced.

The data did not permit intraindividual statistical analysis. However, there were sufficient data to perform statistical analysis for patients as a group for baseline-placebo versus L-dopa periods. There were no positive correlations between neurologic parameters and the ratings for sexual interest or for affect. This would suggest that the alterations in sexual interest and affect in parkinsonian patients treated with L-dopa are independent of changes in neurologic function.

Luteinizing Hormone

There were significant changes ($p<.05$, analysis of variance) in LH in patients 2, 5, and 6. Patient 5 had no significant baseline-to-placebo change but showed a significant increase during L-dopa treatment. Patient 2 demonstrated an increase during placebo but only a slight further increase with L-dopa, indicating the involvement of factors other than L-dopa treatment. Patient 6 showed a significant drop in LH from baseline to placebo and from baseline to L-dopa treatment. The 4 other patients showed no significant changes in plasma LH throughout the study. These findings suggest that factors other than L-dopa influenced plasma LH. Patients 2 and 5, who showed significant increases in sexual activity with L-dopa treatment, also showed significant increases in plasma LH.

DISCUSSION

In this study, 4 of 7 men with parkinsonism reported increased sexual interest or sexual activity related to treatment with L-dopa. (One of these men also reported increased sexual interest with placebo.) In other studies (1–3), in which patients were not specifically questioned about their sexual function, the incidence of increased sexual interest or activity was consistently low—about 4%. In studies wherein the patients were questioned (4, 7, 8) approximately 30% reported increased sexual interest or activity with L-dopa treatment. Further review of these studies revealed that three times as many men as women with parkinsonism reported increased sexual function with L-dopa. Recalculation of rates from other studies for men only, taken together with our own findings, would indicate that the incidence of increased sexual interest or activity among male parkinsonian patients receiving L-dopa is approximately 50%.

It has been suggested (9) that L-dopa may act via the hypothalamic-pituitary-gonadal axis to induce a hormonally related increase in sexual function. The majority of female parkinsonian patients reported in the literature were probably postmenopausal and therefore were less likely to possess an intact hypothalamic-pituitary-ovarian axis. The findings in the present study are compatible with this hypothesis. Those patients with the longest history of impotence showed no increase in sexual function with L-dopa. Those who had no history of impotence (patients 2 and 5) and were more likely to have an intact hypothalamic-pituitary-gonadal axis showed an increase in sexual activity with L-dopa. Moreover, it was the latter patients who also showed an increase in LH with L-dopa. Intrapsychic and interpersonal factors such as the fear of failure in reestablishing sexual behavior after prolonged abstinence must also be considered.

Our neurologic findings are in agreement with those of Bowers and associates (8), who found no significant correlation between sex drive and improved locomotor function in parkinsonian patients treated with L-dopa.

Three of the patients in this study showed evidence of moderate depression before treatment. This is consistent with the findings in other studies (4, 10), in which approximately 50% of patients with Parkinson's syndrome were found to be depressed.

In studies involving parkinsonian patients receiving L-dopa, the affect responses have varied. Jenkins and associates (3) reported that 14 of 90 patients receiving L-dopa became depressed. Marsh and Markham (11) reported no change in Minnesota Multiphasic Personality Inventory depression scores with L-dopa, al-

though there was significant improvement in locomotor functioning. Other studies (1, 3-6) have reported an apparent hypomanic response to L-dopa.

This variable affective response to L-dopa has been observed in studies involving nonparkinsonian patients. Goodwin and associates (12) reported that only patients with retarded depressions improved with L-dopa (administered with a peripheral decarboxylase inhibitor); most of the endogenously depressed patients had no change or became worse. Murphy and associates (13) found that 8 out of 9 nonparkinsonian bipolar depressed patients in their study developed a hypomanic response to L-dopa. Only 1 of 12 unipolar depressed patients manifested a hypomanic response to L-dopa.

This variable affective response to L-dopa was demonstrated in the present study. Only two of the three moderately depressed patients showed an improvement in affect related to L-dopa. One patient (patient 2) demonstrated a hypomanic response to placebo.

CONCLUSIONS

Our study and the review of other findings have indicated that there is a high incidence of sexual impotence and depression in male patients with Parkinson's disease. With L-dopa treatment, parkinsonian patients demonstrated variable affective changes. Two patients with a history of reduced sexual function reported an increase in sexual interest with L-dopa treatment (one also reported increased interest with placebo) but did not report any change in sexual activity. Two patients with no history of impaired sexual function reported an increase in sexual activity with L-dopa treatment. The improvement in sexual function for the group as a whole was not related to improvement in locomotor function. Hormonal factors did appear to be involved in the L-dopa-related increase in sexual activity. These findings suggest that parkinsonian patients who have not undergone an interruption of sexual activity and who possess an intact hypothalamic-pituitary-gonadal axis may undergo an increase in sexual activity relative to L-dopa treatment.

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CLINICAL AND RESEARCH REPORTS

This section contains 1) new research findings, including preliminary data from pilot studies, either clinical or laboratory; 2) worthwhile replication studies; 3) case reports that describe a truly new syndrome or cast new light on established ones; and 4) case reports that indicate a new therapeutic procedure of potential value or call attention to adverse effects of drugs or previously unreported complications of therapeutic interventions. Program descriptions and literature reviews cannot be printed in this section. Criteria for format are listed in "Information for Contributors" in each issue; papers that do not adhere to these criteria will be returned to the author.

Computerized Tomography and Skull X Rays: Relative Efficacy in Detecting Intracranial Disease

BY LUKE TSAI, M.D., AND MING T. TSUANG, M.D., PH.D.

Computerized tomography (CT) is an important new noninvasive neuroradiologic method for detecting intracranial disease (1). It provides as much information about intracranial structures as any other single investigative procedure currently available. However, many hospitals still routinely take skull X rays in addition to using CT scan when organic brain syndrome is suspected. The purpose of the present study is to evaluate whether routine use of both procedures is justified.

Subjects and Method

One hundred and twenty-five patients admitted to the inpatient unit of the Iowa Psychiatric Hospital from 1973 to 1976 received both CT scans and plain skull X rays in addition to initial neurologic and psychiatric evaluation. The age of these patients ranged from 5 to 82 years, with mean ages of 44 for women (N=62) and 35 for men (N=63). The reasons for CT and X ray referral included evidence of memory deficit or deterioration of intellectual function on clinical and/or psychometric testing, persistent or unexplained confusional state, focal abnormality or neurologic examination suggestive of intracranial pathology, focal or generalized seizures, focal or generalized EEG abnormality, evidence of mental retardation, history of poorly explained rage attacks or aggressive behavior, and unusual headache history. The CT scans were performed and interpreted by the department of radiology of the same medical center. We compared the results of the skull films with the CT findings to determine the efficacy of these diagnostic procedures.

Results

Table 1 shows the results of this comparison between skull film and CT scan findings for the 125 patients. In 6 of the 125 patients both CT scans and skull films were positive. In 1 of these patients the skull film showed a shift of the pineal to the right. This diagnosis was confirmed by CT, which showed enlargement of the ventricles. In another case the skull X ray detected intracranial calcification caused by tuberous sclerosis, while CT detected not only the calcification but also the precise location of the lesions. In a patient with a history of a gunshot wound to the head, both skull film and CT located the shotgun pellets. Another patient's skull film indicated Paget's disease and acromegaly, while CT detected cortical atrophy combined with ventricular enlargement. In 2 patients the skull films showed only calcification around the sella turcica, whereas both CT and EEG demonstrated that the cerebral atrophy was a contributory cause of organic brain syndrome.

Of the 29 patients with positive CT scan and negative skull X rays, 12 had cortical atrophy, 4 abnormal ventricular size, 8 cortical atrophy with ventricular enlargement, 4 cerebral infarction, and 1 demyelinating disease.

Five patients had positive skull X rays that indicated possible intracranial tumors, but CT scans were normal. The skull film of 1 of the patients, a 44-year-old woman, showed meningioma. A 21-year-old woman's film showed upward shift of the pineal, and that of a 29-year-old man demonstrated thinning of the dorsum sella. Another patient, a 23-year-old woman, showed

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TABLE 1
Results of Comparison of CT Scan and Skull Films in 125 Patients

Outcome Combinations	Number	Percent
Abnormal CT/abnormal X ray	6	4.8
Abnormal CT/normal X ray	29	23.2
Normal CT/abnormal X ray	5	4.0
Normal CT/normal X ray	85	68.0

increased density of the sphenoid on the skull film. The last of these patients, a 16-year-old girl, had questionable increased intracranial pressure. Radionuclide scans and EEGs done on these 5 patients were normal except that the 23-year-old woman showed a left-right shift of slow waves over the temporal region on her EEG. However, this EEG finding is not compatible with intracranial tumor.

Discussion

The CT scan measures small differences in tissue absorption of X rays, permitting the identification of normal and abnormal structures of the brain, ventricles, and subarachnoid space. These changes in density also make it possible to detect brain edema and hemorrhage and to distinguish them from cerebrospinal fluid, as well as determining ventricular size or displacement. Mori and associates (2) found no false positives when they compared the detectability of lesions by good CT scans with lesion size as measured pathologically. Current data suggest that CT has an overall accuracy rate of 90%–98% in the detection of abnormalities (3) compared with the skull X ray, which has perhaps the lowest yield (50%) of any of the neurodiagnostic methods (4).

CT has several unique advantages over skull films in the location and characterization of tumors that can lead to more accurate planning of complex treatment. CT is extraordinarily effective—100% accurate in several series (3)—in detecting intracranial hemorrhage (cerebral, cerebellar, intraventricular), whereas skull X rays are rarely helpful. One study reported 85% accuracy of CT scans in detecting vascular abnormalities larger than 1.5 cm, including arteriovenous malformation and aneurysm (5); skull films barely detect these abnormalities. CT identifies significant nonacute disease (i.e., cerebral atrophy, porencephaly, old infarction) and clearly defines both normal and abnormal ventricular size and configuration, things that skull X rays cannot do. Demyelinating diseases and changes in the infectious disorders of the brain are evident on CT, whereas skull films are rarely helpful in such cases.

In Delaney's review of 2,943 cases (6), skull films revealed some abnormalities in 17.3% of the patients and significant abnormalities in 2.4%. Seven patients had skull film diagnoses of enlarged sella turcica, but after pneumoencephalography and other medical investigation only 3 of them were considered for surgery. The skull films of 5 other patients with clinical signs of increased intracranial pressure, but subsequent tests did not reveal any neurologic abnormalities. The author concluded that the small yield of significant findings does not seem to justify the effort and expense of routine skull films (6). Bull and Zilkha reported that the great majority of skull films taken for conditions such as migraine or headache, parkinsonism, vertigo, Ménière's disease, and epilepsy, in the absence of clinical signs, did not contribute materially to the diagnosis (7). In the diagnosis of dementia, Bull and Zilkha concluded that skull films were wasteful and unrewarding.

In our study, skull films detected significant abnormalities (intracranial calcification and sella turcica changes) as contributory causes in 2 patients, but CT scans detected these findings. In addition, the skull X rays of 5 patients indicated possible brain tumors, but CT scans, radionuclide scans as well as EEG failed to support these findings. It is apparent that even when positive findings, skull films rarely contribute to the confirmation of intracranial pathology.

With the ever-increasing cost of health care, the goal of clinicians must be to maximize the use of simple diagnostic tests in order to use the fewest possible that are most likely to give the correct diagnosis with the least risk to the patient and the most cost-effective use of the patient's or third-party payer funds. CT results and those of other studies indicate the efficacy of skull X rays in detecting neuropathology is limited. Thus, the routine use of both skull X rays and CT scan is not justifiable in terms of patient benefit and increased cost.

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Oral-Buccal Dyskinesia Symptoms Associated with Low-Dose Benzodiazepine Treatment

BY SEYMOUR R. KAPLAN, M.D., AND CHARLES MURKOFSKY, M.D.

This case report describes the temporal correlation between low-dose benzodiazepine treatment and the occurrence of oral-buccal dyskinesia similar to the oral symptoms associated with neuroleptic drug-induced tardive dyskinesia. We have been unable to find other reports in the literature describing oral-buccal dyskinesia associated with benzodiazepine treatment.

Case Reports

Mr. A, a 63-year-old man, was seen because of hypochondriacal complaints and depressive symptoms. His internist reported no physical findings other than a mild hypertensive condition, which was controlled by hydrochlorothiazide. The patient had a family history of psychiatric illness, and his son had suffered from tics in early adolescence.

Mr. A initially obtained relief from his depressive symptoms with amitriptyline, 150 mg/day. He subsequently was sustained on a dosage of 50 mg/day, except for intermittent exacerbations of the depressive affect that required resumption of the higher dosage. The patient's somatic anxieties and insomnia continued, although lessened in intensity. These symptoms were ameliorated by diazepam (15 mg/day) and flurazepam (30 mg/q.h.s.).

During an exacerbation of depressive symptoms that required the higher amitriptyline dose, Mr. A began to have rhythmic involuntary oral-buccal movements (1 per second) that were "fish-like"; i.e., he pursed his lips and made sucking motions and then protruded his tongue. There were no involuntary movements of the extremities, trunk, or neck.

Because Mr. A had no history of neuroleptic treatment and there have been reports of the association of imipramine with tardive dyskinesia (1, 2), amitriptyline was discontinued. However, the involuntary movements did not diminish.

Mr. A remained on diazepam and flurazepam in the same dosage for 13 months after amitriptyline was discontinued. Both drugs were then stopped, and the involuntary movements unexpectedly ceased within 72 hours.

Because of Mr. A's marked discomfort following the discontinuation of the benzodiazepines and the uncertainty about the specific relationship of diazepam and flurazepam to the involuntary oral-buccal movements, he was instructed to take one 5-mg tablet of diazepam that evening and two 5-mg tablets the next day. He was asked to call about his condition after he had taken the second dose. He reported that the involuntary movements had returned. The benzodiazepines were discontinued and the movements again stopped within 72 hours.

Amitriptyline (150 mg/day) was resumed the day his involuntary movements stopped, after a 1-year hiatus. There was no recurrence of the dyskinesia. Mr. A showed moderate improvement in his depressive symptoms within 8 days, when he had a pleasant and relaxed evening. However, he relapsed the next morning and made a serious suicidal attempt. He ingested at least 20 tablets of flurazepam (15 mg) and an unknown amount of codeine, chloral hydrate, and alcohol. The blood alcohol level on hospital admission was 0.5%. A respirator was required because of the effect of the drugs on the central nervous system.

Two days after his ingestion of flurazepam, there was a recurrence of the oral-buccal dyskinesia. The movements stopped 4 days later at which time Mr. A again complained of marked agitation and perspiration, severe muscle aches, and insomnia. On examination, he had a fine tremor of the hands and memory lapses for the day's events. In addition, he showed uncharacteristic fearfulness and ideas of reference. The symptoms were considered to be part of a drug-induced withdrawal reaction. A neurologic examination and an EEG conducted the next week showed no abnormalities.

Discussion

This case illustrates the temporal association of prolonged low-dose benzodiazepine treatment and the development of oral-buccal dyskinetic movements, similar to the symptoms of neuroleptic-induced tardive dyskinesia. This is the first known report of a possible causal relationship between benzodiazepine treatment and dyskinetic symptomatology. It must be balanced with the fact that benzodiazepines are the most widely prescribed drugs today, with 44,280,000 prescriptions during 1974–1975 (3).

There have been several reports in the recent literature describing dyskinetic symptoms associated with the use of drugs other than neuroleptics. In 1973, Crane observed that "a variety of drugs, including amphetamines and L-dopa . . . cause involuntary movements in all areas of the body, including the mouth" (4). The association of tricyclic treatment to oral dyskinesia has been reported (1, 2), and a recent publication described the occurrence of these symptoms with the prolonged use of antihistaminic decongestants (5). Crane noted that dyskinesias associated with drugs other than neuroleptics tend to subside once the drugs are discontinued. He also pointed out that organic conditions such as Huntington's chorea should be considered in the differential diagnosis of dyskinetic symptomatology (4).

Research on neuroleptic-induced tardive dyskinesia indicates that it is caused by dopamine hypersensitivity due to prolonged neuronal blocking. L-Dopa and amphetamines directly potentiate central dopaminergic

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activity. In addition, the anticholinergic property of psychotropic drugs, especially the tricyclics, is considered a factor in the etiology of dyskinetic symptoms because of their influence on the dopaminergic/cholinergic balance within the brain.

Studies of brain mechanisms associated with benzodiazepines are less conclusive, but there have been indications of interaction with neurotransmitters, including γ-aminobutyric acid (GABA), glycine, norepinephrine, and serotonin. For example, benzodiazepines may mimic GABA's presynaptic inhibition at the serotonin nerve terminal (6). One might speculate that a compensatory reaction to this inhibition could develop after prolonged benzodiazepine treatment. Further research is necessary to determine the mechanisms involved in the actions of benzodiazepines on the CNS. However, we thought it important to report this unusual occurrence of tardive dyskinesia-like symptoms during ongoing low-dose benzodiazepine treatment. It is suggested that clinicians consider this possibility when treating cases of involuntary oral-buccal movements in patients whose drug regimen [illegible] zodiazepine treatment. Further considera[illegible] tionship between tardive dyskinesia-lik[illegible] and benzodiazepine treatment requires c[illegible] clinical observations.

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Hyperkinesis and Diet: A Replication Study

BY FLORENCE LEVY, M.B.B.S., M.R.A.N.Z.C.P., M.P.H., AND GARY HOBBES, M.COMM.

There has been considerable recent controversy about the claims of Feingold (1) regarding the effects of artificial colors and food additives on hyperactivity in children (2, 3). A controlled study by Harley (4) failed to show any differences between children on the Feingold diet (known as the K-P diet) and a placebo diet. A double-blind crossover trial conducted by Conners and associates (5) found a significant reduction in symptoms of children on the K-P diet as rated by teachers.

Goyette and associates[1] have studied a sample of children selected by the following criteria: 1) a reduction of at least 25% in symptoms as rated by the mother on the Conners Hyperkinesis Index (6), 2) a behavioral and clinical diagnosis of hyperkinesis, and 3) baseline scores of at least 15 on mothers' and clinicians' ratings of the Hyperkinesis Index.

In the Goyette and associates study, eight subjects (mean age=5.3 years, range=3.4–8.4) met the full criteria, and five other children were border[illegible] ers. All subjects participated in a single 2[illegible] over trial with daily ratings by parents for a [illegible] riod after subjects ingested cookies cont[illegible] food colorings currently approved by t[illegible] Drug Administration or placebo. These [illegible] found a significant challenge effect in a Gr[illegible] × Challenge analysis of variance.

Levy and associates (7) reported a [illegible] crossover trial of the Feingold diet in whi[illegible] a tartrazine challenge. The children were [illegible] and after 4 weeks on the elimination die[illegible] weeks of tartrazine or placebo challenge. [illegible] scale scores and objective tests showed [illegible] differences in the behavior of the childre[illegible] during the challenge period. However, [illegible] thors tested a subgroup of the children se[illegible] basis of a 25% reduction in symptoms[illegible] there was a significant challenge effect.

Method

The present study attempted to r[illegible] Goyette and associates unpublished stud[illegible] same selection criteria, rating scale (the [illegible] Index), and instructions to parents on ra[illegible] dren. Challenge or placebo cookies wer[illegible] domly over 14 days. One difference from [illegible]

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[1]Unpublished data from C.H. Goyette, C.K. Conners, T.A. Petti, et al.

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and associates study was that the challenge consisted of tartrazine only (yellow dye number 5), whereas Goyette and associates used a mixture of food colorings approved by the FDA. The children ingested 4 cookies containing 1 mg of tartrazine each.

Results

Eight subjects met the study criteria. There was 1 dropout, a child whose parents claimed he became worse after ingesting the challenge cookies for 3 days. The average age of the children was 5 years, 2 months. There were seven boys and 1 girl. A Subjects × Treatment analysis of variance indicated no significant interaction. The mothers' scores on the Conners Scale were an average of 2.6 points higher during the challenge. However, this treatment effect just failed to reach the .05 level of significance. Global assessments by the mothers showed no significant differences in distinguishing active from inactive cookies. Thus the study did not replicate the significant challenge effect found by Goyette and associates. However, the 2.6-point difference in mothers' scores on the challenge versus placebo represented a 13% drop on placebo cookies from the initial average score of 20.5.

Discussion

The results may have been affected by the sample size—it was difficult to obtain a large sample of children who met all the selection criteria. In addition, the use of a symptom rating scale for assessing small or transient differences in hyperkinetic children has inherent problems; the differences rated by parents may be statistically significant but may not represent important clinical effects, or vice versa.

A further study reported by Conners and associates (5) found no significant differences in parental ratings during double-blind testing. However, an objective tracking test of attention showed a trend toward deterioration 1–2 hours after subjects ingested challenge materials. It is important that future studies of diet effects (and medication effects) use objective tests rather than the less reliable rating scales.

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Tricyclics, Bioequivalence, and Clinical Response

BY ROBERT B. OSTROFF, M.D., AND JOHN P. DOCHERTY, M.D.

Tricyclic antidepressants are used widely in the treatment of depression (1). Much evidence has accumulated linking plasma levels of these drugs to therapeutic response (2–4). This evidence highlights the importance of comparing the bioequivalences of currently available tricyclic preparations both among various brands and within specific brands. Bioequivalence refers to the equivalence of the target biological activity, e.g., postsynaptic neuronal stimulation. In terms of the clinical use of tricyclics, bioequivalence, which is used interchangeably with the term "bioavailability," reflects the rate of absorption and the amount available for absorption of the active ingredient into the circulation (5). The following case illustrates the clinical importance of plasma drug levels and bioequivalence in the use of tricyclic antidepressants.

Case Report

The patient, a 56-year-old married man, stated that he came to the psychiatric clinic "because my doctor sent me." He had had one brief psychiatric hospitalization 38 years previously for depression. In the intervening years he had been without symptoms and without psychiatric contact. In the initial interview he was noted to have marked motor retardation, with constriction of affect and concrete thinking. He had had significant stressful familial and marital problems and complained of a 2-month history of anorexia, a 4.5-kg

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weight loss, early morning awakening, difficulty falling asleep, crying spells, feelings of hopelessness, and suicidal thoughts. He was not delusional.

He was started on 50 mg/day of amitriptyline and seen subsequently in couples therapy. Three days later, when no adverse drug effects were noted, the dosage of amitriptyline was increased to 100 mg q.h.s. He reported an immediate improvement in his sleep disturbance. At the end of week 2, his dosage was increased to 100 mg q.h.s. and 50 mg q.a.m.

At the end of week 4, the patient appeared at a couples session with a smile on his face. His expression was animated, the motor retardation was gone, and he reported improved appetite and complete amelioration of his dysphoria. At that session his prescription for amitriptyline was renewed. Three weeks later he came to a session appearing as depressed as he had initially. He complained of early morning awakening, difficulty falling asleep, loss of appetite, depression, and feelings of hopelessness. He admitted that he had taken an extra amitriptyline tablet during the week, but both he and his wife confirmed that this was the only such instance. There were no dynamic stresses. His amitriptyline was increased to 150 mg q.h.s. and 50 mg q.a.m., but his symptoms remained unchanged a week later. He was taking no other medications, including antacids, and had not been ill. He denied gastrointestinal disturbances and diarrhea. The couple's therapist (R.O.) was puzzled about the change in the patient's requirement for medication.

The amitriptyline dosage was increased to 200 mg q.h.s. and 50 mg q.a.m. One week later he reported a complete amelioration of his symptoms. At that point, the hospital pharmacy's record was reviewed. It was discovered that the pharmacy stocked two different manufacturers' brands of amitriptyline, one in 25-mg tablets (brand A) and the other in 50-mg tablets (brand B). The patient's dosage had been increased in increments of 25 mg (brand A) until he had a therapeutic response. When he renewed his prescription, he was given the 50-mg tablets. He required 250 mg of this product to achieve the response he had on 150 mg of brand A. A plasma level was obtained and the medication was changed back to brand A, 150 mg/day. One week later plasma levels were again determined. Plasma levels were determined on both doses and brands, with samples taken 8 hours after his last dose. The levels on the 250-mg dose of brand B were 18 ng/ml of amitriptyline and 23 ng/ml of nortriptyline. Levels on 150 mg of brand A were 29 ng/ml of amitriptyline and 45 ng/ml of nortriptyline. Six weeks later the patient remained symptom-free on 150 mg of brand A amitriptyline.

Discussion

There is substantial evidence that a minimum plasma level of a tricyclic compound is essential to therapeutic response (4). If the bioequivalence of tricyclics varies from manufacturer to manufacturer for the same compound, or from batch to batch produced by one manufacturer, then patients will achieve different plasma levels even though they are being given identical amounts of the active ingredie[nt] ... the Food and Drug Administration propo[ses] ... lations to establish a bioequivalence re[quirement] ... certain oral drugs in this category, inclu[ding] ... monly used amitriptyline and imipramin[e] ... rivatives. In the proposal it is stated th[at] ... data suggest that the various marketed ... same oral tricyclic antidepressant may ... parable therapeutic effects" (6). Many ... pharmacists to substitute various brand ... agent. The case we have reported sugg[ests] ... the digitalis preparations, tricyclics s[hould] ... scribed by brand name. The prescrib[er] ... should be aware that bioequivalence ma[y] ... between compounds and that, unless s[pecifically] ... structed not to do so, pharmacists in ma[ny] ... substitute preparations containing the ... agent. Also, the bioequivalence for ... turer's preparation may vary if the ine[rt] ... are changed. One must be sure that the ... company variability in the preparation o[f] ... pound. Additionally, in terms of cost ... dosage requirements must be considere[d] ... cost effectiveness rather than simply ... cost. At the present time, when plasma ... readily available and the correlations a[mong] ... plasma levels, and clinical response are ... the patient may be best served by havi[ng] ... adjusted and maintained on a given pro[duct] ... pound.

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Baclofen in the Treatment of Tardive Dyskinesia

BY N.P. VASAVAN NAIR, M.D., RAMSES YASSA, M.D., JOAQUIN RUIZ-NAVARRO, M.D., AND GEORGE SCHWARTZ, B.SC.

Tardive dyskinesias are involuntary stereotyped hyperkinesias that may develop during and after long-term treatment with neuroleptics. Although the exact pathogenesis of tardive dyskinesia is unknown, the current hypothesis is that neuroleptic drugs block striatal dopamine receptors (1), rendering these chemically denervated receptors hypersensitive (2).

Gamma-aminobutyric acid (GABA) is a major inhibitory neurotransmitter spread throughout the central nervous system. There is increasing evidence that the gabergic neuron system inhibits the dopaminergic nigrostriatal system (3). In animal studies, GABA agonists have been shown to inhibit the impluse flow in the nigrostriatal dopaminergic neurons (4) and to inhibit the release of dopamine into the synaptic cleft (5).

Baclofen (Lioresal, 4-amino-3-(4-chlorophenyl-butyric acid) is a GABA derivative that crosses the blood-brain barrier. It acts as a GABA agonist in inhibiting the striatal dopamine system (6). Korsgaard (7) and Gerlach and associates (8) found baclofen effective in tardive dyskinesia, whereas Simpson and associates (9) found it ineffective. The purpose of this study was to determine the efficacy of baclofen in a 3-week placebo-controlled crossover clinical trial.

Method

The subjects were 10 chronic schizophrenic patients (5 men and 5 women), ranging in age from 40 to 64 years (mean=56), who had been hospitalized for at least 20 years (mean=29) and had moderate to severe neuroleptic-induced tardive dyskinesia. Informed consent was obtained from the patients or their relatives.

All neuroleptic medications were withdrawn for at least 3 months. Patients were assigned randomly under double-blind conditions to receive baclofen or placebo for 3 weeks; they were then given a 2-week washout and a 3-week crossover. Baclofen dosage began with 30 mg/day, increasing gradually to 90 mg/day. The main evaluative instrument used was the Abnormal Involuntary Movement Scale (AIMS), which was recorded on videotape before and at the end of each 3-week phase of the study. In addition, the Clinical Global Impression (CGI) scale and the Brief Psychiatric Rating Scale (BPRS) were completed at the same time as the AIMS. Three psychiatrists rated the videotapes blind, and the mean AIMS scores were computed. Vital signs were taken daily and laboratory assessments were completed weekly.

Results

No statistically significant changes were found between baseline and the baclofen or placebo periods (Wilcoxon matched-pairs signed-ranks test). The mean sum of the first 7 movement scores of the AIMS before and at the end of placebo administration was unchanged at 8.8; mean scores increased slightly (from 10.9 to 11.5) in the baclofen condition. No significant changes were noted on the BPRS or CGI.

One woman patient developed severe loss of muscle tone toward the end of the baclofen period. No other side effects or significant changes on any of the laboratory measures and vital signs were noted for the group.

Discussion

Our finding that baclofen was not effective in the treatment of tardive dyskinesia is in agreement with that of Simpson and associates (9). Korsgaard (7) and Gerlach and associates (8) did find significant improvement in their patients' dyskinetic movements when baclofen was administered in combination with neuroleptic medications.

Our finding of a low incidence of side effects is also similar to those of Simpson and associates (9); Korsgaard (7) and Gerlach and associates (8) reported more side effects, but their patients were older and had various diagnoses, including dementia, which may have contributed to the increased incidence of side effects.

It appears, therefore, that baclofen when administered alone is ineffective in the treatment of neuroleptic-induced tardive dyskinesia in chronic schizophrenics.

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Baclofen and placebo were supplied by Ciba-Geigy of Canada, Ltd.

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Adult Minimal Brain Dysfunction and Schizophrenia: A Case Report

BY LEIGHTON Y. HUEY, M.D., MARK ZETIN, M.D., DAVID S. JANOWSKY, M.D., AND LEWIS L. JUDD, M.D.

Evidence exists that minimal brain dysfunction (MBD) may continue into adulthood in a variety of clinical manifestations (1-3). One group has reported that some adult patients with MBD respond favorably to psychostimulants, but patients with schizophrenia were excluded from that study (4). In addition, a subgroup of schizophrenics has been identified who are clinically responsive to psychostimulants (5) and who, unlike other schizophrenics, do not show an intensification of psychosis with such drugs (6).

The following case may be representative of a subgroup of individuals diagnosed as schizophrenic who have histories of childhood MBD. We postulate these individuals may have an adult variant of the MBD syndrome rather than schizophrenia per se and may be clinically responsive to treatment with psychostimulants rather than conventional neuroleptics.

Case Report

A 23-year-old man was admitted to the hospital with delusions of reference, complaints of auditory hallucinations, and feelings of confusion. He had a history of multiple hospitalizations during which he was diagnosed as schizophrenic and treated with neuroleptics, which were minimally effective. Past antipsychotic drug treatment had made him feel worse, and at no time did his auditory hallucinations disappear completely.

In childhood, the patient was seen by psychologists for behavior problems and hyperactivity, but he never received pharmacologic treatment. As a child he had been described with nervousness, inability to sit still, stubbornness, difficulty concentrating, temper tantrums, failure to complete tasks, fighting, bad handwriting, poor coordination, and general troublemaking.

In recent years, the patient had complained of restlessness, emotional overactivity, moodiness, poor concentration, impulsiveness, sloppiness, and poor coordination. He had occasionally used amphetamines because they seemed to have calmed him and made the hallucinations tolerable.

The clinical admission diagnosis was chronic undifferentiated schizophrenia. Using a structured diagnostic interview employing diagnostic criteria of Feighner and Spitzer, he was assigned a diagnosis of probable chronic undifferentiated schizophrenia. His full-scale IQ was 119.

Because of the patient's childhood history of MBD, he was enrolled (after giving informed consent) in a double-blind, placebo-controlled study to assess the effects of intravenous methylphenidate (0.5 mg/kg) on his psychiatric condition. The patient received placebo on day 1, followed by a 1-day drug washout and active methylphenidate on day 3.

He was interviewed on days 1 and 3. During the initial interview (placebo), the patient did not relate well to the physician, complained of active auditory hallucinations, and was anxious. He felt he was not "with" himself. His affect was markedly blunted. He displayed tremulousness in his extremities and in his facial movements. This tremor continued throughout the first session. During the interview on day 3, the patient initially appeared anxious and reported active auditory hallucinations, which were encouraging. After injection with methylphenidate, he stated the hallucinations faded into "babbling" and "noise." He became less anxious and was in better contact with the interviewer. He felt calm and more confident and reported feeling more able to focus on the tasks required during the session.

Four days after this interview, the patient was started on a double-blind trial comparing the effects of 4 days of oral placebo methylphenidate to 4 days of oral methylphenidate (10 mg b.i.d.).

Results

The results of the intravenous and the oral methylphenidate conditions are presented in Table 1. The oral

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TABLE 1
Effects of I.V. and Oral Methylphenidate on Mood, Behavior, and Performance (Modified BPRS)

	I.V. Methylphenidate							
	Placebo			Active			Oral Methylphenidate	
	Baseline	+30 Minutes	+60 Minutes	Baseline	+30 Minutes	+60 Minutes	Placebo	Active
Beigel-Murphy Manic Rating Scale (total)	18	19	18	21	9	7		
NIMH Behavior Rating Scale (total)	55	50	46	51	36	32	43	38
POMS scales								
Tension-Anxiety*	61	59	58	64	30	32	52	43
Depression-Dejection*	40	39	38	40	40	40	40	36
Anger-Hostility*	49	47	46	50	37	37	49	37
Vigor*	44	38	38	45	44	45	41	36
Fatigue*	38	38	37	43	43	45	37	37
Confusion-Bewilderment*	66	58	61	64	35	35	55	45
Elation	2	2	0	2	5	3	3	4
Friendliness	6	5	5	7	11	10	8	8
Trailmaking Test (seconds)	64	59		66	31		29	28
Digit Symbol Test (number completed)	45	41		41	65		66	77

*Scores presented in T score form (mean=50, SD=10).

were some marked changes between the placebo and active intravenous trials. Changes were less pronounced for the oral phase of the study, perhaps because of the relatively low dose of methylphenidate. On the Beigel-Murphy Manic Rating Scale (8), the patient showed less distractability, irritability, and activity, and displayed better impulse control when challenged with active methylphenidate. During active medication phases, the NIMH Inpatient Behavioral Rating Scale indicated decreases in anxiety and tension, disorganized speech, hostility, excitement, hyperactivity, distractability, and increased affect. There appeared to be an increased sense of depression and uncertainty about the future.

On the Profile of Mood States there were decreases in scores on items measuring tension-anxiety, anger-hostility, and confusion-bewilderment during active drug administration. The patient also showed improved performance on the Trailmaking and Digit-Symbol Tests of the Halstead-Reitan Battery.

The changes seen with the oral methylphenidate, although less dramatic, paralleled those seen with the intravenous drug. The patient reported improved concentration and increased calmness during the oral active methylphenidate phase. He frequently read novels for long periods; when he was not receiving the drug his behavior was characterized by anxiousness and pacing.

The patient has been treated effectively for 4 months as an outpatient on 25 mg/day of oral methylphenidate. He reports improved ability to maintain good interpersonal relationships. On one occasion, the patient's methylphenidate was stolen and he was off the drug for 2 weeks. During that time, he reported a return of the auditory hallucinations, worsening concentration, and sleeplessness. Reinstitution of methylphenidate resulted in improvement of these signs and symptoms. During the outpatient program, the patient has never asked for an increase in his dose of methylphenidate or for extra medication.

Discussion

This case suggests it may be important to distinguish between those individuals who have schizophrenia and those who appear schizophrenic but may have an adult manifestation of MBD. Effective treatment for these groups may be different, with conventional neuroleptics being efficacious for schizophrenics, and psychostimulants, tricyclic antidepressants, or lithium having efficacy in those with adult MBD. Quitkin and associates (9) have described a group of schizophrenics, characterized by "soft neurologic signs," who showed improvement on imipramine. Furthermore, Handford (10) has hypothesized that individuals who experience brain hypoxia perinatally will be at risk for having minimal brain dysfunction in childhood and for subsequently developing schizophrenia as adults, possibly secondary to damage to dopaminergic pathways.

It has been suggested that minimal brain dysfunction can persist well into adulthood and may be concealed by other diagnoses, including sociopathy, explosive personality, hysterical character, and labile personality (4). We believe schizophrenia could be added to this list and propose that some adult patients diagnosed as schizophrenic may have a variant of the MBD syndrome. We speculate that some of the schizophrenics who show favorable responses to psychostimulants may be those having childhood histories of MBD or an adult variant of MBD.

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Phenelzine and Delusions of Parasitosis: A Case Report

BY MICHAEL R. LIEBOWITZ, M.D., ERIC J. NUETZEL, M.D., ANSTISS E. BOWSER, M.S.W., AND DONALD F. KLEIN, M.D.

A delusion of parasitosis is the firm, fixed (mistaken) belief that one is infested by parasites. The phenomenon occurs with the use of psychostimulants—particularly amphetamines, cocaine (1), and methylphenidate (2)—and as a form of monosymptomatic hypochondriacal psychosis (3). Similar phenomena occur in various disease entities such as temporal lobe epilepsy, schizophrenia, and delirium tremens (4).

A variety of adverse effects have been reported with monoamine oxidase inhibitors (MAOIs), including psychosis, mania, and delirium (5), but we are unaware of any reports of delusions of parasitosis associated with this class of drugs. In fact, MAOIs have recently been advocated in the treatment of such delusions (6). The following case illustrates delusions of parasitosis in a patient taking MAOIs.

Case Report

Ms. A, a married office-worker in her late 20s, sought outpatient treatment for depression and marital difficulties. Her history included recurrent depressive episodes in response to feeling rejected. These nonautonomous episodes were characterized by tearfulness, social withdrawal, suicidal thoughts, decreased energy, overeating, and oversleeping, comprising a syndrome termed hysteroid dysphoria (7).

The patient had no history of mania or psychosis, although she had experienced transient visual and tactile hallucinations while smoking marijuana. She denied sedative abuse and use of hallucinogens or cocaine, and she had avoided marijuana for several years before we saw [illegible] phetamine use had consisted of up to four [illegible] several months 8 years earlier. Her alcohol u[illegible] ly light, and she had a stable work history. Wh[illegible] depressed, Ms. A was outgoing and vivacious [illegible] history and results of a physical examination [illegible]

Ms. A was treated with psychotherapy and [illegible] phenelzine in a maintenance program for hys[illegible] ics after the procedures were fully explained [illegible] informed consent to participate. The phenelz[illegible] increased to 60 mg/day over the next several [illegible] reduced to 45 mg/day during the 8th week of [illegible] Ms. A became overstimulated. She appeared [illegible] functional and happy on this dose for the ne[illegible]

During the 18th week of treatment, while [illegible] 45 mg/day of phenelzine, Ms. A reported to h[illegible] her body and hair were infested with small bl[illegible] had first noticed a painful itching all over her [illegible] followed by the sensation of things crawling [illegible] She said tiny black objects fell from her hair, [illegible] her clothing, burrowed into her skin, and ca[illegible] her pores. She consulted a dermatologist, w[illegible] festation. She also saw long, black, hairlike [illegible] in her stool and consulted an internist, who [illegible] fibers. During psychotherapy sessions, [illegible] scratched and picked at herself, sometimes [illegible] aware of this behavior and at other times dis[illegible]

Ms. A's mental status at this time was char[illegible] line only in that she was less depressed and [illegible] tactile hallucinations and a delusional belief [illegible] She was oriented in all spheres, had no cog[illegible] ment, and continued to work throughout the [illegible] phenelzine was discontinued, the itching dec[illegible] biting, crawling, and pinching sensations sto[illegible] Ms. A continued to see small black objects fa[illegible] but she now believed they were particles from [illegible] ing or vent. Over the next 3 months her itch r[illegible] of the scalp, visualization of small black ob[illegible] cern with environmental pollutants gradually [illegible] ing this period physical examination and lab[illegible] ing were normal, except for borderline vitam[illegible] serum B_{12} levels, which were treated with ora[illegible]

After 4 months off medication, Ms. A was a[illegible] and was restarted on phenelzine, 15 mg/day [illegible]

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she reported the feeling that bugs were crawling on her skin and scalp, and she saw numerous black moving spots when looking at light-colored surfaces. The experience was identical with the previous one, except that this time it was attributed by the patient to medication. Serum vitamin levels were normal, and the symptoms disappeared within several days after she was switched to placebo phenelzine in a single-blind manner.

Two nonhydrazine MAOIs were then tried on a nonblind basis with 10-day drug-free periods before each. Tranylcypromine, 10 mg/day, produced severe abdominal cramping and constipation after 3 days and was discontinued. She was then given pargyline, 10 mg/day, and after 4 days reported seeing gray-black spots and feeling crawling and pinching sensations of scalp and pubic regions. This made her wonder whether she was infested with lice. An antidepressant effect was noted on day 6, but the drug was discontinued the next day because of its side effects.

Discussion

A variety of mechanisms have been offered to explain psychostimulant-induced psychoses. Amphetamine potentiates several catecholamines, but attempts to assess relative dopaminergic and noradrenergic effects through behavioral and neurochemical studies have produced conflicting results (8). Post and Kopanda (9) suggested an interaction of catecholamine potentiation and pharmacological kindling or sensitization of limbic structures to explain cocaine's effects. Ellinwood (1) argued that amphetamine-induced delusions of parasitosis are an extension of stereotyped grooming and picking responses in association with abnormal skin sensations that may be peripherally mediated. Finally, Siegel (10) speculated that cocaine hallucinations evolve from drug-induced tactile sensations and visual phenomena.

Our patient's delusion of parasitosis, evolving in a clear sensorium, was indistinguishable from those previously reported with psychostimulant use. Phenelzine is clearly implicated: the delusions appeared after 4½ months of phenelzine use, gradually waned when the drug was discontinued, reappeared soon after it was reinstituted, and disappeared after a single-blind switch to placebo. The reappearance of similar symptoms when the patient was taking a low dose of pargyline implies that the effect is not dependent on hydrazine toxicity.

Several inferences can be made with regard to pathophysiology. This patient's periods of behavioral overstimulation and delusional belief were temporally distinct, which indicates that they were mediated by different mechanisms. That she also showed a reverse tolerance to the drug supports the involvement of a kindling mechanism. Most significantly, her delusion of parasitosis seemed elaborated from a combination of abnormal skin sensations and visual phenomena, similar to the hypothesis proposed by Siegel for cocaine (10).

One can also speculate on other mechanisms. Peripheral nerve effects may play a role, although repeated assays failed to confirm suspected vitamin abnormalities. Trace substances such as phenylethylamine may accumulate, adversely affecting a patient who has substantially inhibited MAO activity. Finally, this patient may have had an underlying vulnerability to visual and tactile hallucinations, as evidenced by her past reactions to marijuana.

The case is presented to elucidate further the etiology of psychostimulant-induced psychoses and to alert physicians who prescribe MAOIs. We recommend careful monitoring of any patient with a history of even brief psychotic experiences and discontinuation of the drug should these symptoms develop.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Associat[illegible] requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes [illegible] regarding publication. Letters must be typed double spaced throughout and should not contain [illegible] words and 5 pertinent references. Criticisms of published articles will automatically be sent to the [illegible] response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we [illegible] writers of the disposition of letters or return those which are not printed.*

Policy of the APA Ethics Committee

SIR: In the February 1978 issue Robert A. Moore, M.D., published his interesting and informative article, "Ethics in the Practice of Psychiatry." The paragraph on due process may be misunderstood and is in need of clarification. In that paragraph, Dr. Moore advocated that ethics committees investigate complaints and hold hearings according to the adversary model of criminal justice, including confrontation and cross-examination of the complainant. Although that article did not appear in the buff pages (reserved for official actions of APA) in the *Journal* and therefore should be clearly understood to reflect only Dr. Moore's personal opinion, at the time the article was published Dr. Moore was Chairperson of the American Psychiatric Association Ethics Committee and, in fact, made such procedures the policy of the APA Ethics Committee when it acted in the capacity of initial investigating body. Thus, some might (and indeed at the Ethics Workship, Course 7, at the annual meeting, one lawyer did) justifiably but mistakenly think Dr. Moore's statement reflected not merely his personal opinion but the policy of APA as well.

In fact, the APA Constitution and By-Laws and the Procedures for Handling Complaints of Unethical Conduct (Revised 1977) make no statement prescribing or recommending a formal "hearing." Rather, they state that the complainant must file a complaint in writing and that the investigating body "permit both the defendant and complainant to be heard," allowing greater flexibility for ethics committees to tailor their procedures to the needs of individual cases.

The official position of APA is consonant with that of the American Medical Association Judicial Council Opinions and Reports (1) (by which APA would not be bound, since we are a separate organization) and is supported by legal precedents concerning disciplinary proceedings by professional societies and other membership organizations. For example, to quote the California Supreme Court from the Pinsker decision mentioned by Dr. Moore (in which exclusion determined as equivalent to expulsion was at issue):

> The common law requirement of a fair procedure does not compel formal proceedings with all the embellishments of a court trial . . . nor adherence to a single mode of process. It may be satisfied by any one of a variety of procedures which afford a fair opportunity for an applicant to present his position . . . The [illegible] themselves should retain the initial and pri[illegible] sibility for devising a method which provi[illegible] quate notice of the 'charges' . . . and a re[illegible] portunity to respond. In drafting such pro[illegible] determining, for example, whether an appl[illegible] given an opportunity to respond in writing [illegible] sonal appearance, the organizaiton should [illegible] nature of tendered issue and should fashi[illegible] dure to insure a *fair* opportunity for an app[illegible] sent his position. (2)

Thus, the procedures advocated by Dr Mo[illegible] required nor recommended by the official pol[illegible] by the AMA, or by legal precedent.

The Washington Psychiatric Society and A[illegible] (on whose behalf I am writing) strongly su[illegible] policy as it now stands. The adversary proc[illegible] mended by Dr. Moore, which are neither p[illegible] requirement, in our opinion do not represent [illegible] or ideal model. From our experience, we w[illegible] number of complaints filed would plummet [illegible] plainant had to attend an adversary heari[illegible] plainants, many of whom are patients in a pre[illegible] mental health, would be willing (nor do we be[illegible] willing) to subject themselves to cross-exami[illegible] gressive opposing attorney (and in his article [illegible] ommends the complainant not have the right t[illegible] legal counsel present). Furthermore, it has no[illegible] pression that following the adversary model [illegible] tice helps in uncovering the truth. Rather, i[illegible] experience that the more we have veered tow[illegible] sary model, the more we have become enme[illegible] dural technicalities and the harder it has be[illegible] the ethical concerns and recommend appropr[illegible]

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2. Pinsker v Pacific Coast Society of Orthodont[illegible] 116 Calif RPPR 245, 526 P 2d 253

ROY M. Co[illegible] M.D.
Wash[illegible]

Dr. Moore Replies

SIR: While APA does have established procedures for the routing of ethical complaints and for appeals, it does not have procedures for how hearings should be conducted. To avoid any misunderstanding, my article should not be construed as an official position of APA.

Two years ago, the APA Ethics Committee held a workshop on hearings for its own education with a faculty consisting of the APA legal counsel, an American Civil Liberties Union attorney, and an attorney from the American Arbitration Association. The results of this workshop have been circulated to the District Branches and discussed at the last two workshops on ethics at the annual meeting. As in the article, this information has been presented for consideration as a hearing model, not as a mandate. Of course, situations differ so that no one model will be correct in all cases.

The issue of the right to challenge someone who brings charges against another is well established in due process. What constitutes "fairness" in a fair procedure is less clear. While we are urging greater activity in self-policing, this must not lead us to disregard what is "fair" for one of our members. I believe that an accused member has the right, in all fairness, to personally challenge his or her accuser. That right does not belong to the hearing body nor the accuser but to the accused. He or she may well decide to forego that right, especially if the accuser is a patient who might be harmed by such a confrontation.

A more important issue is whether we are erring by moving to more formal procedures. Unfortunately, in earlier years, our efforts at peer review and discipline were often subterranean and had a "clubby" quality that led our critics to say we were doing nothing and therefore they should do it for us. Further, accused members themselves, exercising their legal rights, have challenged such informality. The result has been greater formality and visibility to demonstrate we are capable of protecting the integrity of our profession without denying our members their rights.

While there may be differences of opinion, I am most impressed by the dedication to ethical principles and to their protection shown by Dr. Coleman and the Washington Psychiatric Society.

ROBERT A. MOORE, M.D.
San Diego, Calif.

Ethics in Military Medicine

SIR: I feel it is necessary to emphasize that the comments by Ransom Arthur, M.D. ("Reflections on Military Psychiatry," July 1978 supplement) on ethics and military medicine are his own highly personal conclusions and represent only one opinion on an issue worth studying more closely. Dr. Arthur's comments, in part by focusing on a reductio ad absurdum, would have us believe that all ethical questions regarding military psychiatry are "ethical fastidiousness," "ethical hypersensitivity," and "scrupulosity."

Dr. Arthur states that "it was only with the arrival of a war of stupendous unpopularity involving a nation possessed of unprecedented comfort and affluence that moral sensitivity arose about a number of war-related issues." Here he appears to lump genuine concern about ethical issues with radical chic. He neglects the growing legitimate (to my mind) questioning of inappropriate governmental activities in this country. This questioning has been more frequent since World War II, in part because some members of a new generation both experienced the McCarthy era and heard Eichmann say, "I was just following orders," and do not wish to see those mistakes repeated.

In addition, I would like to call into question one of Dr. Arthur's statements of fact. He states that "moral dilemmas of military medical practice were mentioned in works dealing with military psychiatry in both of the world wars but were quickly dismissed." Yet Lord Moran wrote in 1943, describing his feelings as a medical officer with the British Army in World War I:

> but what, I wonder, became of pity in those ruthless years? When I look back I see that I was caught up in the atmosphere of the trenches. . . .
>
> I was perturbed at that time not by any difficulty in shaping opinion in the battalion, but by a gnawing anxiety lest the hard temper of the hour should drive men beyond what is fair and just. . . . Even now after twenty years my own conscience is troubled by the summary judgements passed on some poor wretch in those days, and by my own part in those verdicts. (1, p. 177)

This statement was made by a distinguished physician who served as Churchill's doctor during World War II.

Finally, Dr. Arthur states that "in any case, with the ending of America's direct participation in war, the moral issues of combat psychiatry are now moot." This is untrue. It is precisely now, some time removed from "the atmosphere of the trenches," that we can reflect more soberly on these issues. We need to sharpen our thinking in this area, not only to be prepared for future wars, but also because we need to be aware of the potential role conflicts that inevitably occur in any psychiatric setting (2).

The issue is too emotion-laden and too complicated to be dealt with merely by part of an article and by an exchange of letters to the Editor. It deserves a more extensive look.

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MICHAEL S. PERLMAN, M.D.
Northampton, Mass.

Dr. Arthur Replies

SIR: I had hoped that my comments on the changing ethical climate of military psychiatry would provoke some thought and further ideas. I am pleased to see that they have done so.

The interpretation that I implied that all ethical questions regarding military psychiatry are merely the results of ethical hypersensitivity is a misreading of the text. If the topic of ethics in military medicine did not have real substance, it would not have been included in the essay.

I stand behind my statement that the Viet Nam war and the recent tumult about ethics in government have produced a vastly different social and intellectual climate for the consideration of such questions than was the case during and after World Wars I and II. There really were no organized

general, and explicit statements of moral opposition to the provision of medical services per se (including psychiatric services) to the military, during either world war. Naturally there were, as always, individual acts of protest against war itself (Bertrand Russell's, for example) but certainly no "movement" against military medicine as such. A rereading of *The Anatomy of Courage* leads to the interpretation that the remarks quoted by Dr. Perlman are reflective of Lord Moran's experience with effects on morale of gas warfare rather than an indictment of military physicians. In fact, he indicates on a subsequent page, "But in this life the individual shrinks to nothing, he has no longer the right to an opinion, only the regiment matters" (1). During World War I, Lord Moran served as the battalion medical officer for the First Royal Fusiliers (the initial regular battalion) from 1914 to 1917. Although he was obviously ambivalent about war—admiring the courage and example of units like the Brigade of Guards but deploring the misery, squalor, and loss of life attendant upon armed struggle—he never puts forth any conceptualization of an explicit conflict between his role as a physician and his role as an officer of the Fusiliers. Whatever his private doubts may have been, he did his duty as a medical officer throughout his active service on the Western Front. On the other hand, the Viet Nam war occasioned overt public criticisms of military medicine. During that war, publicity was given to possible moral conflicts between one's duty as a physician and one's duty as a member of a nation's armed forces.

Of course, there should be rational debate on these issues, but discussion can be unnecessarily envenomed by the intense emotion that still colors any thoughts about that horrid war in Southeast Asia. I recently reviewed a book dealing with certain psychiatric problems of the Viet Nam war. A striking feature of many of the excellent essays was the sense of general rage expressed by the authors. There was a tone of vituperation directed toward all manner of institutions, which could not be more different from the tone of works like *Psychiatry in a Troubled World* (2), which dealt with the events of World War II. I think we should be discussing the ethical constraints asked by the modern world of a military physician. However, genuinely fruitful discussion will be difficult until some of the indignation has diminished and a certain degree of dispassion and objectivity is attained.

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RANSOM J. ARTHUR, M.D.
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Dangers of Indirect Data

SIR: In the June 1978 issue there was an extremely useful and informative article on "The Opiate Receptor and Morphine-Like Peptides in the Brain," by Solomon H. Snyder, M.D., who is famous for contributions to neurobiology, particularly the biochemical aspects of psychiatric disorders. We all know that collecting meaningful data from patients with psychiatric disorders is very difficult—patient characteristics cannot be controlled like those of experimental animals, and human considerations make it almost impossible to collect data from the living, functioning [illegible]. I [illegible] ce[illegible] sequence, there has developed a large bod[illegible] lite[illegible] based on indirect data.

There is a potentially misleading passage in [illegible]rap[illegible] illustrates some typical consequences of the [illegible] of [illegible] data. The passage begins as follows: "A leu[illegible]-com[illegible] β-endorphin molecule has been reported in [illegible]ey[illegible] schizophrenics undergoing hemodialysis (75 [illegible]Wa[illegible] ker, personal communication)" (p. 650). The[illegible] m[illegible] of findings in controls, so it is not clear whe[illegible] om[illegible]h specific to schizophrenia was observed. The [illegible] ues, "Wagemaker and Cade (personal com[illegible] [illegible]tic[illegible] ported that repeated hemodialysis increases [illegible]deg[illegible]e clinical improvement in schizophrenics." In [illegible]ss. of the *Journal* there was a paper (1) in whic[illegible] [illegible]sp[illegible] data from many centers showed no positive [illegible] fects of hemodialysis on 50 schizophrenic pa[illegible] failure. Perhaps Dr. Snyder was not familiar [illegible] os[illegible] ings, but the Editorial Board of the *Journal* c[illegible] municated them to him. There is great interes[illegible] on hemodialysis, and it is important that nega[illegible] communicated rapidly. (I should note that I [illegible] skeptical about the hemodialysis findings fro[illegible]

The author then notes, "Chemical analys[illegible] from these patients revealed the presence of [illegible] amino acid sequence was established as bein[illegible] that of β-endorphin except that the methioni[illegible] of β-endorphin was replaced by leucine. The f[illegible] id, could imply a single gene mutation in sc[illegible] The data given are not sufficient to propose [illegible] is irresponsible to add to the many works [illegible] schizophrenia that have already been writte[illegible] in the way of definite data. Dr. Snyder must [illegible] data are necessary to postulate such a genetic [illegible] note that "whether schizophrenic and nor[illegible] differ reliably in levels of this substance has [illegible] ously established." Given this lack of data, I [illegible] passage should not have been published. T[illegible] passages like this in the literature, and some[illegible] tirely constructed of indirect and/or uncont[illegible] should stop publishing this uninformative ar[illegible] leading material.

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C. Van [illegible]
Groningen, [illegible]

Dr. Snyder Replies

SIR: Dr. Van den Berg raises several very [illegible] about my review of the opiate receptor and [illegible] Basically, Dr. Van den Berg is unhappy th[illegible] studies showing improvement of schizophr[illegible] following hemodialysis and the reported isol[illegible] β-endorphin from dialysates of schizophrenic[illegible] Berg is certainly correct that these findings [illegible] tive and have not been adequately controlled [illegible] other laboratories and on those grounds fe[illegible] have discussed them. However, these finding[illegible] considerable amount of attention from the sc[illegible]

eral public, particularly through the news media. Because I thought the readers of the *Journal* were probably familiar with these findings and associated claims, I felt obligated to discuss them and to point out their very tentative nature. I also felt obliged to point out the logical implication of a possible single gene mutation reflected in the specific abnormality of leucine-β-endorphin in schizophrenic blood. Obviously, one would not draw this conclusion until the data on which the speculation is based were shown to be replicable.

I was not aware of the survey of schizophrenics given hemodialysis who did not show any clinical improvement when I wrote the review.

SOLOMON H. SNYDER, M.D.
Baltimore, Md.

Depressive Cognitions

SIR: In "Maladaptive Cognitive Structures in Depression" (May 1978 issue) Maria Kovacs, Ph.D., and Aaron T. Beck, M.D., outlined in a precise and scientific fashion which has long been known about some of the phenomenologic aspects of depression. These observations are consistent with empirical observations by psychiatrists and physicians and more or less intuitive general knowledge about depression.

At this point, however, the authors interpret in a causative role what others have considered to be merely the symptoms or effects of depression. Some effort is made to affirm that these cognitive patterns and mechanisms are present in depressed patients, and this is done rather convincingly.

The "proof of the pudding," however, would seem to await a study showing that what the authors have labeled "depressive cognitions" are present in patients with histories of recurrent depressions while their depressions are in complete remission. Presumably, this would not be the case with a matched control group of nondepressed normal people. Such a finding would more convincingly establish these depressive cognitions as part of the patient's underlying personality structure rather than symptoms resulting from the depressive syndrome itself.

I hope that such a study will be forthcoming from the authors.

MARK T. SCHREIBER, M.D.
St. Louis, Mo.

Drs. Kovacs and Beck Reply

SIR: We appreciate Dr. Schreiber's positive comments about our article and would like to respond to two interesting issues he raises regarding the cognitive model of depression.

First, Dr. Schreiber alludes to the traditional concept that depressive cognitions are basically "symptoms or effects of depression," as opposed to the more salient role we emphasized. Second, he says we have not shown depressive cognitions to be part of the depression-prone individual's personality (or cognitive) organization.

The first question relates to the primacy of cognitive factors in the depressive syndrome. In many contemporary descriptions, depression is regarded as an affective disorder, pure and simple, and its cognitive manifestations are largely ignored. However, we have attempted to make some sense out of the highly diverse symptoms by arranging them in a coherent logical sequence.

In taking a "cross-section" of depressive symptomatology, we look for the primary psychopathology in the peculiar way the individual views himself, his experiences, and his future, and in his idiosyncratic way of processing information. The postulate that the patient's negative construction of reality is the first link in the chain of symptoms provides a range of hypotheses, many of which have been tested and supported in over two dozen studies (1). A recent review of cognitive behavior therapy in the treatment of unipolar depression (2) cited 10 additional outcome studies which report that techniques derived from this model appear to be more effective than comparison treatments. (We are aware, of course, that the efficacy of therapy does not prove the underlying theory).

Therefore, at this stage, we can state with some minimal level of confidence that the cognitive model has a greater explanatory power of the phenomena of depression than other psychological models presented to date. The model, however, does not address the ultimate etiology or cause of unipolar depression, which may encompass hereditary predisposition, faulty learning, brain damage, biochemical abnormalities, or any combination thereof.

In regard to the role of underlying maladaptive personality traits, we base our case on logic and speculation. It does not seem plausible that the aberrant cognitive mechanisms are created de novo every time an individual experiences a depression. It appears more credible that an underlying predisposition (personality trait or cognitive structure) becomes activated when depression is precipitated by psychological stress, biochemical imbalance, hypothalamic stimulation, or whatever. The manner of activation of the depressogenic schemata has been formulated previously (3). In our article, we cited two relevant studies. Hauri (4) found that compared with controls *remitted* depressed patients showed a preponderance of characteristic negative dream themes and a predominant past time orientation. Weintraub and associates (5) reported that over a course of 5 testings, cognitive content was remarkably stable compared with affect. Based on the Weintraub and associates study, it would be a logical conclusion that in the organization of personality, a factor that is stable over time is more likely to have a causative role then an unstable one such as affect.

We certainly agree with Dr. Schreiber that the "proof of the pudding" resides in further investigations. There is an urgent need for longitudinal studies of depressed patients in remission. It would also be desirable to compare the cognitions of two groups of remitted depressed patients, one given cognitive therapy and one treated with chemotherapy. We could then test the hypothesis that cognitively treated remitted depressed patients are less likely to evidence the so-called depressogenic assumptions and cognitions than the comparison patients.

In summary, Dr. Schreiber's points are well taken, but his suggestion that the cognitive distortions result from an independent syndrome labeled depression has not yet received any empirical support.

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MARIA KOVACS, PH.D.
AARON T. BECK, M.D.
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Nutmeg Abuse

SIR: The case report in "Spice Cabinet Intoxication" by Robert A. Faguet, M.D., and Kay F. Rowland, Ph.D. (July 1978 issue) was of special interest to me as I had reported a similar case elsewhere (1). The patient reported by me had ingested two tablespoons of nutmeg and demonstrated an acute confusional state with visual hallucinations. The authors suggested that myristicin was the psychoactive ingredient responsible for the symptoms. However, it appears that the hallucinogenic properties of nutmeg are not caused by myristicin but by other volatile principles present in it, like eugenol, isoeugenol, geraniol, safrol, borneol, and linalool (2).

The usual toxic dose of nutmeg is about 5–15 g of powdered nutmeg, and toxicity in a majority of cases is usually accompanied by severe physical distress. Nutmeg abuse is not as rare as the authors suggest. Nutmeg poisoning was first documented in 1576, and since then there have been a number of reports of toxicity following intentional or accidental excessive use of nutmeg (3, 4). "Nutmeg high" was well known to the youth of the 60s, and convicts have always abused nutmeg.

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Effects of Diagnostic Criteria on Incidence Figures for Schizophrenia

SIR: Michael Alan Taylor, M.D., and Richard Abrams, M.D., noted in "The Prevalence of Schizophrenia: A Reassessment Using Modern Diagnostic Criteria" (August 1978 issue) that narrow, rigorous criteria for a diagnosis of schizophrenia may result in a low incidence of schizophrenic admissions to a psychiatric inpatient service. However, at the Medical College of Virginia, we have noted a much higher admission incidence of schizophrenia than that reported by Drs. Taylor and Abrams.

As part of the clinical trials of *DSM-III*, we studied 67 inpatient admissions, aged 15–95 years, all of whom were interviewed by at least one of us. There was 10[illegible] agreement on diagnosis for all patients who were interviewed by [illegible] us. Of the 67 patients, 15 (22.4%) received [illegible] schizophrenic disorder. Overall, 15 of the 91 [illegible] diagnoses (16.5%) were of schizophrenia.

These data may be considered further evi[illegible] the low incidence of schizophrenia reported by D[illegible] and Abrams is solely a function of diagnostic rigo[illegible] for schizophrenia in the proposed *DSM-III* a[illegible] between Kraepelinian-Schneiderian narrowness [illegible] rian broadness. However, this is unlikely in [illegible] cause most of the schizophrenics had Schneid[illegible] symptoms and most met the research criteria [illegible] Taylor and Abrams.

Another explanation of the differences in in[illegible] the results are an artifact of the patient popu[illegible] Our hospital is a public urban teaching hospital [illegible] ly serves individuals in Hollingshead-Redlich [illegible] III–V. There is generally a higher rate of sc[illegible] these social classes than in social classes serv[illegible] urban center the authors studied. In addition, [illegible] atric hospitals in our locality "siphon" off pa[illegible] fective disorders, which leaves a patient poo[illegible] incidence of schizophrenia and which leads t[illegible] mission incidence of schizophrenics.

KENNETH SOL[illegible], M.D.
PRAKASH G. E[illegible], M.D.
LUCY JANE[illegible], M.D.
ROBERT J. RES[illegible], Ph.D.
Ric[illegible]

Drs. Taylor and Abrams Reply

SIR: Dr. Solomon and associates' report th[illegible] of inpatients satisfied *DSM-III* criteria for schiz[illegible] is interesting and encouraging. Their figure is one [illegible] could expect using *DSM-II*, and their 100% diag[illegible] ment suggests that for the first time America[illegible] will have reliable official diagnostic criteria.

Their figure, however, is higher than not [illegible] but also all recent reports in which researc[illegible] been applied to samples of consecutive hospit[illegible] We agree that it is unlikely that the differenc[illegible] result of *DSM-III*'s differing from publishe[illegible] though it would be interesting to know what [illegible] their 67 patients satisfied our criteria and othe[illegible] teria for schizophrenia. It is also unlikely that [illegible] are due to different populations served, since [illegible] 8 different studies, which included public and [illegible] tions across the eastern half of the United St[illegible].

We agree that the difference could be an [illegible] patient population sampled, but we would nee[illegible] manner of admission selection before comm[illegible] possibility.

It is not surprising, however, that criteria [illegible] general use are less precise than research cri[illegible] theless, Dr. Solomon and associates' experien[illegible] *III* is reason for optimism, and should the ot[illegible] prove the criteria equally reliable, progress [illegible] curred.

MICHAEL A. TAY[illegible], M.D.
RICHARD ABR[illegible], M.D.
North C[illegible]

Editorial Double Standards?

SIR: The recent exchange of letters between Joseph Wolpe, M.D., and Silvano Arieti, M.D. ("Research Methodology," August 1978 issue) concerning my criticism of the publication of Dr. Arieti's article "Psychotherapy of Severe Depression" (August 1977 issue, and letter in the January 1978 issue) forces me to conclude that we have all left the real culprits off the hook. I write of journal editors. Despite lengthy missives to the contrary, Dr. Arieti did not define his patient group in reproducible terms, did not use reliable and valid pre- and post-treatment measures of behavioral change (or any other change), did not use a comparison control group, and did not have an "unbiased" rater evaluate his patients after treatment.

My quarrel is not with Dr. Arieti; it was and is with the editor of this and most other psychiatric journals. If I were to submit an article about my favorite treatment modalities that had the same failings as Dr. Arieti's paper, I would receive a rejection letter, not to mention nasty reviews. This is a double standard. If I cannot get away with bad science, why can Dr. Arieti? Dr. Wolpe wrote about the "disdain some psychotherapists display for the rules of scientific research." If such disdain exists, it is because journal editors have allowed it to exist. Let the Editor end this double standard and explicitly state the rules of scientific research to which each article, no matter what its content, must adhere. I look forward to such a policy in the *American Journal of Psychiatry*.

MICHAEL A. TAYLOR, M.D.
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Editor's Note. Although not certain that the writer's conclusions about editors were reached through the scientific procedures he espouses, we endorse the standards he implicitly proposes for the direction of the *Journal*. We will pursue them diligently, secure in the knowledge that when the fallibility of human judgment leads us into an occasional falling from grace, it will not be met by silence.

J.C.N.

Cause and Effect in "Alcoholic Tremor"

SIR: Many patients and physicians point to alcohol toxicity as the cause of a tremor, and "alcoholic tremor" has been well described. However, it is important to note that some alcoholic patients began drinking alcohol because of a tremor. We have seven patients who have definitely stated that they started drinking alcohol because alcohol relieved their tremor. As the need seemed to be continuous, the drinking became more and more essential.

Benign essential (familial) tremor is a common condition manifested by trembling or involuntary rhythmic movement of the body, usually upper limbs or head. It most commonly starts in adulthood (1), affects both sexes, and may progress slowly. Often, there is evidence of a dominant inheritance pattern (2). No consistent pathology has been found (3).

The rate of the tremor is generally in the range of 4–12/second, and it may be an exaggeration of normal tremor (4). It is present at rest and may be inhibited temporarily by volition. However, it becomes worse when the patient is under stress or is being observed and improves when he or she is tranquil or in repose. Fatigue usually increases the tremor. Neurologic examination is otherwise normal. These patients often report improvement after alcohol intake. Treatment consists of various drug therapies (e.g., propranolol) or simply reassurance.

We feel that preexistent benign tremor may play a role in some patients' initial use of alcohol and that not all tremors in alcoholics are due to excessive alcohol intake.

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GEORGE PAULSON, M.D.
Columbus, Ohio

Speech Blockage in Young Patients Taking Tricyclics

SIR: In their recent article "Speech Blockage: A Tricyclic Side Effect" (May 1978 issue) Alan F. Schatzberg, M.D., Jonathan O. Cole, M.D., and Dietrich P. Blumer, M.D., reported on five patients who developed a previously undescribed side effect of tricyclic antidepressants, "speech blockage." In their communication they described this as an inability to find and translate words into speech, leading to pauses in conversation which could be confused with the "blocking" seen in schizophrenic patients. Four of their five patients were over age 40 and the fifth patient was 36, so they suggested this side effect to be more common in older patients. They attempted to explain this phenomenon as resulting from a central anticholinergic effect on higher cortical functions or, alternatively, from "a relative decrease in cholinergic activity secondary to a tricyclic-related increase of noradrenergic activity."

I wish to report another case of speech blockage, this occurring in a 25-year-old man. The patient, a married man, sought treatment for a depression of several months' duration characterized by insomnia, loss of appetite with slight weight loss, diminished interest in usual activities, headaches, abdominal discomfort, palpitations, poor concentration, and depressed mood with feelings of hopelessness and worthlessness. He was begun on amitriptyline alone, 75 mg/day, which was increased to 100 mg after one week. After another week he experienced what he described as difficulty in "getting my words out" and feeling like his "mouth muscles wouldn't work" for a few seconds at a time. The "blockage" occurred unpredictably in conversation, sometimes in midsentence. Sometimes he could only articulate one syllable of a word but after trying a few seconds he would be able to complete the sentence. He reported no difficulty finding or thinking of words but rather in translating the word from thought to speech. When the amitriptyline was increased to 125 mg, the effect became worse and the medication had to be discontinued. Within 2 days, his speech returned to normal. He was then started on imipramine and increased to 150 mg over 10 days, during which he experienced one transient episode of speech blockage. This did not recur again while

he was continued on 150 mg, and his depression resolved completely over the next 2 weeks.

Unlike the cases reported by Schatzberg and associates, this patient was young, in his 20s. Also unlike the reported cases, the patient denied difficulty "finding" words but rather could not convert a conceived word into actual speech. In agreement with their description, however, this patient showed no evidence of a toxic-confusional state, nor was his speech slurred. He had no problem articulating or pronouncing words; rather, there was brief but complete cessation of the speech process.

It is of interest to note that along with speech blockage, he also experienced occasional difficulty with recent memory, an effect previously reported with tricyclics (1). Schatzberg and associates did not state whether their patients also experienced memory impairment, but this might be important in view of their description of difficulty with word retrieval as part of speech blockage. It is not clear, for example, whether speech blockage might be related to a transient disturbance of memory, or to an aphasic or apraxic phenomenon.

Although I have treated hundreds of patients with tricyclic drugs, this is the first patient in my experience to report this side effect. Whether this reflects a lack of awareness or a low incidence of its occurrence is unclear. As this case illustrates, however, clinical awareness of this side effect of tricyclic antidepressants must now be extended to younger patients as well.

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ALAN J. SHOLOMSKAS, M.D.
New Haven, Conn.

Drs. Schatzberg and Cole Reply

SIR: We were pleased to read Dr. Sholomskas' report of another case of speech blockage. His patient showed some difficulty with memory that our patients did not demonstrate. We have seen and reported on mild memory difficulty with tricyclic antidepressants in another group of patients (1). It is difficult to determine whether the symptom reflects a central hesitance in thinking or a hesitance in translating the "cognitive thought" into words. This is in part a matter of differentiation. Our patients did not appear to be aphasic. Dr. Sholomskas' observation that the symptom can occur in a young patient should alert us to look for such symptoms in patients on tricyclics. We believe the symptom is more common than it may appear. Since our report I have seen several other cases and other clinicians have reported cases to me. One last point, which we raised in a previous letter, regards the question of some patients with stammering having myoclonic jerks of facial musculatry. This is not clear from Dr. Sholomskas' letter but may have been a contributing factor in that the patient felt like "his mouth muscles wouldn't work." Was this a descriptive statement of the symptom or did the patient in fact demonstrate some involuntary facial jerks or difficulty in phonation? We believe that our patients' difficulties were primarily related to the central anticholinergic effects of the drugs. However, to prove this is difficult.

Again, we wish to thank Dr. Sholomskas for his contribution of reporting another case of "speech blockage."

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ALAN F. SCHATZ[illegible] M.D.
JONATHAN O. [illegible] M.D.
Bel[illegible] Mass

Treating the Impaired Physician

SIR: The American Medical Association's C[illegible] of Ethical Ethics make it clear that physicians are [illegible] help faltering colleagues. It is less clear how [illegible] this.

Impaired physicians may benefit from syst[illegible] plemented, practical programs of casefindi[illegible] vention, in which psychiatrists actively pa[illegible] Michigan Psychiatric Society recently urged [illegible] APA program aimed at three groups: the seriou[illegible] those troubled but still capable of practicing [illegible] trainees (1). Your readers may be interested i[illegible] mendations outlined below, since the burden o[illegible] turbed colleagues falls most heavily on psych[illegible]

The seriously impaired physician. He/she us[illegible] ous, on the brink of professional ruin, and re[illegible] rehabilitative effort, including the following:

1. Crisis teams with psychiatric direction, t[illegible] ry out intervention.
2. Financial advice and emergency loans.
3. A *locum tenens* roster (perhaps of recent[illegible] still effective physicians) to replace a faltering [illegible] can get back on his feet.
4. A national network of residential treatm[illegible] impaired physicians, with free services for th[illegible] pay (2).
5. Health insurance adequate to cover both [illegible] outpatient therapy.
6. Emotional support and guidance for t[illegible] family.

The troubled but still practicing physician. [illegible] those in this category include the following:

1. Improved casefinding and highlighting o[illegible] ty of voluntary, confidential help for the phys[illegible] family.
2. Information and assistance to the spous[illegible] the key to obtaining diagnosis and treatment [illegible] cian.
3. A roster of physicians in each state wh[illegible] consult with impaired physicians and their f[illegible] cians recovered from disability could be unic[illegible] this effort. (APA and the district branches [illegible] leadership.)
4. Self-awareness experiences and mutual [illegible] to explore stresses of medical practice on fam[illegible] hance the family's effectiveness as *the* suppo[illegible]

The medical student and resident. Medic[illegible] training programs should be encouraged to

1. Improve students' stress-handling tech[illegible] crease awareness of the physician's vulnera[illegible] tional disturbance and of available help.
2. Provide readily available psychiatric c[illegible] treatment.
3. Teach doctors to ask for help.

Most of these suggestions are already in pl[illegible]

locations. However, only a comprehensive effort will overcome the social and emotional barriers that still separate the faltering physician from effective help (3). It has been reported in a western state that 25% of physicians whose fitness to practice has been questioned by the medical licensure board end as suicides. Attempts to secure public funding of programs to counteract such tragedies often are met with condescending variations on the theme "physician, heal thyself." Others may report more encouraging results, but I believe that if the troubled physician is to have a better fate than the proverbial shoemaker's child, it is clear that we will have to take care of ourselves. Material from the third national AMA conference on the impaired physician (Sept. 29–Oct. 1, 1978) should clarify how we are to do this.

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Nonconfusional Paranoid Psychosis Secondary to Anticholinergics?

SIR: I read with interest the article "Levodopa-Induced Psychosis: A Kindling Phenomenon" by Charlene Moskovitz, M.D., Hamilton Moses III, M.D., and Harold L. Klawans, M.D. (June 1978 issue). In that article they noted that 6 out of 43 patients "exhibited psychiatric side effects while on anticholinergics and/or amantadine alone. These symptoms were not qualitatively different from those seen in patients on levodopa or combination therapy." As I have not previously read any report of a nonconfusional paranoid psychosis secondary to anticholinergics I would be very interested to know if one of their patients on anticholinergics alone did indeed develop such a condition.

DAVID P. MOORE, M.D.
Louisville, Ky.

Dr. Klawans Replies

SIR: I am afraid that Dr. Moore is correct and that our statement is somewhat ambiguous. What we observed in patients who were on anticholinergics or amantadine alone were hallucinations that were not in any way different from those seen in patients on levodopa alone. We, too, have not seen any report of a nonconfusional paranoid psychosis secondary to anticholinergics. The point we wished to make was merely that the hallucinogenic state brought on by anticholinergics appeared to be identical to that of hallucinogenic states brought on by dopaminergic agents.

HAROLD L. KLAWANS, M.D.
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Testosterone Levels and Women's Sexual Preference

SIR: I have received many inquiries regarding an article I wrote with D. Lynn Loriaux, M.D., Ph.D., and Thomas N. Chase, M.D., on "Plasma Testosterone in Homosexual and Heterosexual Women" (October 1977 issue). Colleagues have asked whether I concluded that homosexual women demonstrated testosterone "abnormalities" compared with heterosexual women.

I would like to emphasize that the testosterone ranges for the two groups of women (homosexual and heterosexual) overlapped considerably. In fact, only 5 homosexual women had testosterone measurements above the range for the heterosexual women. Furthermore, all testosterone measurements—for the homosexual as well as the heterosexual women—fell within the range considered normal for women who do not have endocrine problems. (The normal range of <100 mg/100 ml was determined by Hazelton Labs, Inc., a laboratory under contract with the National Institutes of Health to perform radioimmunoassays.) Therefore I would not conclude that homosexual women have abnormal testosterone measurements.

Persky and associates (1) recently demonstrated that high-normal baseline testosterone levels in heterosexual women—levels that were equivalent to the highest levels measured in our homosexual women—were significantly correlated with high self-rated gratification scores and the ability to form positive interpersonal relationships. It would be interesting to determine whether high-normal plasma testosterone levels in homosexual women are correlated with similar sexual, social, or interpersonal advantages.

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NANETTE GARTRELL, M.D.
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Clinics, Private Practice, and National Health Insurance

SIR: Donald G. Langsley, M.D., ended his article on "Comparing Clinic and Private Practice of Psychiatry" (June 1978 issue) by saying, "It seems fair to conclude that . . . private psychiatric treatment can be successfully defended and financed." However, if I were a member of the United States Congress formulating plans for mental health care coverage under national health insurance (NHI), Dr. Langsley's article would lead me to believe instead that clinics such as the community mental health center (CHMC), in addition to providing services which are not available in the private sector, can and do provide treatment services identical to those provided by the private practitioner. I would conclude that the clinic system constitutes the preferable mental health care delivery system.

Whether it is true or not, legislators today are of the general opinion that the CMHC working in tandem with health maintenance organizations (HMOs) will best serve the mental health care needs of the nation, providing services at the lowest possible cost. In fact, under all NHI plans currently before the Congress, the role of the private practitioner of psychiatry is greatly restricted, especially in providing out-

patient treatment. (The most liberal plan allows only 20 "private" outpatient visits per year.)

I think Dr. Langsley misleads us when he implies that clinic and private psychiatric practice are roughly equivalent. Could he have reached his conclusions because the clinic psychiatrists he reports on were actually working in private clinics rather than in HMOs or CMHCs? I cannot find such a demarcation. Could it be that the "majority" he reports on are unrepresentative of clinic and private psychiatrists taken as a whole?

Dr. Langsley makes a mistake, I think, by not examining the functioning of the clinic as a whole, but rather isolating the function of the psychiatrist within the clinic. Clinic staffs as a whole engage in more supportive psychotherapy than private practitioners, many (but not all) of whom choose to focus their treatment on more in-depth, reconstructive psychotherapy. This, I think, is a major point distinguishing the private practitioner of psychiatry from the clinic practitioner (all breeds), especially in the CMHC. It is one definite reason why the private psychiatrist should be entitled to provide care under NHI, or rather why the patient should be entitled to receive care from him or her.

In connection with Dr. Langsley's article, I would like to make a few general comments about mental health care in the public and private sectors. Unfortunately, by their design public clinics (such as CMHCs) do not usually afford the patient the option of treatment by a psychiatrist. Even if they do, there is not much choice for the patient as to who his or her psychiatrist will be. We all know that this is a crucial choice because a patient can only work well with a psychiatrist with whom he or she can establish a strong rapport.

There is a strong case to be made to the effect that the individual patient is more likely to receive in-depth comprehensive treatment from the private practitioner of psychiatry. Public clinics are oriented toward treating the patient in a brief number of visits because they operate under broad economic pressures that really do not take into account length or depth of treatment. Consequently, a clinic's treatment goals may well differ from the patient's own treatment goals. By contrast, when a patient is treated by a psychiatrist in private practice, he or she is able to work with the psychiatrist until both feel that a "cure" or the optimum possible readjustment has been reattained (provided, of course, financial resources are available to support treatment such as this).

We do have statistics that bear out the differences between treatment the patient receives from a public clinic versus the private practitioner. The average number of visits per patient at the CMHC is only 5.3 (1) in comparison with an average of 46 treatment visits per year to psychiatrists in private practice (2). (Even though the median of the latter type of visits is much lower, 25 visits, we must acknowledge that there is a group of patients, not necessarily more sick, for whom in-depth treatment is likely to produce a better result in terms of overall adjustment.) We would all acknowledge that for the most part, only very superficial mental health care can be delivered when a patient is treated only five times. This leads to one assumption: namely that the CMHC—and mental health care within the public sector in general—is oriented toward resolution of patient crises. It would seem, on the other hand, that the private practitioner of psychiatry works with his patients not only to alleviate the acute presenting problem, but to enable the patient to change his behavior so that his crises do not repeat themselves ad infinitum. In the HMO, too, I think we shall find significant economic disincentives to the practice of such "private-style" open-ended psychotherapy, as well as limited cho[illegible] for the patient.

Unfortunately, the private practitioner of [illegible] not justify his existence under NHI unless [illegible] what he does is different, important, and [illegible] effective. Either that, or he can appeal to th[illegible] enterprise.

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DAVID [illegible], M.D.
[illegible]

Dr. Langsley Replies

SIR: Perhaps the best response to Dr. Upt[illegible] him of my concluding sentence: "... *private* [illegible] treatment can be successfully defended and fi[illegible] good enough to begin his letter with that [illegible] theme of his concern is a comparison of all tre[illegible] by mental health clinics as compared to that [illegible] vate psychiatric practitioners. That was not t[illegible] The data available to me concerned only the [illegible] atrists in clinical or private settings. I have [illegible] the same concern about the quality of treatm[illegible] nity mental health centers that is implicit in L[illegible] ter. Those concerns are based on the lack of [illegible] CMHCs where, during the period between [illegible] the number of psychiatrists per center dropp[illegible] while the number of psychologists doubled a[illegible] of social workers increased by 50%.

The purpose of the study was to focus only [illegible] trist (not on the total clinic). I think it demon[illegible] psychiatrist in a clinic setting in these two are[illegible] does the same kind of work as his colleague i[illegible] tice. The study also provided evidence that [illegible] myth about private practice and gave data to [illegible] vate practitioners do not neglect the serious[illegible] vide a variety of treatment according to the [illegible] tient. If the study had any theme at all, it [illegible] defended the position of the private as well as [illegible] chiatrist and suggested that private psychi[illegible] should be available under government insura[illegible]

Our patients, and we as professionals, hav[illegible] from the position that psychiatric practice is [illegible] that quality treatment can take place in priva[illegible] tings. The criticism of clinics has more to do [illegible] psychiatrists than with how psychiatrists w[illegible] settings actually practice. Our political eff[illegible] aimed toward increasing psychiatric involv[illegible] manding the quality care that can be provid[illegible] ficient number of psychiatrists and the app[illegible] their skills.

DONALD G. LAN[illegible], M.D.
Chic[illegible]

BOOK REVIEWS

The Other Generation Gap: The Middle-Aged and Their Aging Parents, *by Stephen Z. Cohen and Bruce Michael Gans. Chicago, Ill., Follett Publishing Co., 1978, 283 pp., $10.95.*

The generation gap discussed in this book presents a real and pressing problem for some families and bids fair to become equally important for many others as the life span for increased numbers of the elderly lengthens. More and more middle-aged families find themselves unprepared for the "real and often destructive feelings of anxiety and guilt because of their parents."

The main purpose of the book, the authors state, is to pull together materials and insights that might equip middle-aged sons and daughters to view their parents' physical, social, and emotional needs within the larger context of aging. To this end the authors have compiled information, guidelines, and suggested techniques from medical, psychological, and geriatric standpoints and present them from the point of view of the middle-aged individual. The authors rightly believe that understanding the dynamics of aging will help readers strike a better balance between their own needs and those of their parents. Wisely, they also believe that efforts should be made to encourage the parents' independent functioning and to postpone the time when they become a burden.

Unlike in Europe where the different generations of a family often live close to one another, generations in the United States frequently live apart. Thus, when the problems of elderly parents fall on the middle-aged they are often unprepared to handle them. Not infrequently they find themselves in a bind between trying to understand their own adolescent children on one hand and their elderly parents on the other.

A large number of the elderly fail to accept the physical changes taking place within them. However, these individuals are not as hard to care for as are those who magnify every bodily change and pester their children with their complaints. This can arouse resentment along with an urge to withdraw in the middle-aged children. In some cases children and parents have had poor interrelationships to begin with.

Although in some cultures the elderly are regarded as precious and their longevity enviable, in our culture the elderly are equated with uselessness. Women have lost their beauty and are not expected to be attractive. Elderly men are overly sensitive to the physical impression they make. The need to be noticed and desired is something many individuals never lose, no matter what their sex or age.

Disabilities, retirement, death of a spouse, and death of friends are all punishing blows for the elderly; often enough these events hint that death is not far off. Therefore, the elderly cling to a sense of continuity while their world steadily shrinks.

These facts require understanding on the part of their children, as do the elderly parents' methods of coping with their problems. By understanding these phenomena the offspring will learn how best to approach their parents when the need arises. The middle-aged children should also recognize that their parents' doctor is often regarded as the most important person in their lives and that they would do well to become acquainted with him. They might also take a long hard look at their own emotions over the years, considering the feelings they had about their parents and judging how much conflict might arise if they lived in close proximity to one another.

A genuine understanding of the inevitable changes happening to the aged is an essential element in the parent-child relationship. The habits of elderly parents are fixed and they are not going to change—in fact, they may become even more pronounced with age. The independent functioning of elderly parents should be prolonged by whatever combination of means necessary. The authors advise the formulation of a set of feasible limits to the child's direct involvement and determination as to whether his or her activities arise from a sincere desire to help or are motivated by feelings of guilt and obligation. They advise against martyrdom.

There is a discussion of nursing homes and the need for their inspection and for getting the advice of a skilled social worker about them before placing a parent in one. If a parent can live alone, independently, that is usually best. Most elderly individuals do not want to be a burden, and a quick reaction on the part of the son or daughter out of a sense of guilt and reawakened childhood worries can be destructive. The authors warn that rearranging one's own life because of such a reaction might well turn into a bitterly resented obligation. One must evaluate one's own qualifications and balance one's parents' needs with one's own while keeping the Biblical injunction regarding parents in mind.

F.J.B.

The Golden Cage: The Enigma of Anorexia Nervosa, *by Hilde Bruch, M.D. Cambridge, Mass., Harvard University Press, 1978, 150 pp., $8.95.*

Dr. Bruch describes anorexia nervosa as a disease that befalls the young, rich, and beautiful. She has made extensive contributions toward the understanding of this illness in the past and acknowledges that the disease is not really new. However, she suggests that its incidence has greatly increased in recent decades. The contention that the disease selectively afflicts the rich and beautiful, however, may be an artifact of patient selection rather than its true epidemiology.

The Golden Cage is a distillation of knowledge gleaned from many years of work with anorectic patients and their families. It addresses a wide audience, both lay and professional. Although it is only 150 pages long, its content and substance are substantial. The title evokes the imagery described by one of the patients, who saw herself as a sparrow in a golden cage, too plain and simple for the luxuries of her home but deprived of doing what she truly wanted to do.

Even though English is not her mother tongue, Dr. Bruch

has that rare capacity for beautifully and accurately conveying what she wants to say. In her characteristically incisive style she places into sharp focus the nature of the disease, its antecedents, the development of the illness, its psychological characteristics, and the process of treatment. Throughout the lively text are interlaced case vignettes and anecdotes. She integrates the best of both worlds: appreciation of the importance of physiological changes attending starvation and a thorough understanding of the psychodynamics underlying the psychological conflicts in the patient and operating within the family.

The book will be informative, educational, and helpful to anorectic patients and their families. It will aid them to understand better the disease and to evaluate the adequacy of their treatment. At the same time it will be of immense value not only to the beginning therapist but also to the experienced one. The last chapter is a masterful exposition of psychotherapeutic technique. Dr. Bruch states, "The task of psychotherapy in anorexia is to help a patient in her search for autonomy and self-directed identity by evoking an awareness of impulses, feelings, and needs that originate within her." The principles of treatment Dr. Bruch espouses have much broader implications and applications for disorders other than anorexia nervosa.

ALEXANDER R. LUCAS, M.D.
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The Way Out Must Lead In: Life Histories in the Civil Rights Movement, *by William R. Beardslee, M.D. Atlanta, Ga., Emory University Center for Research in Social Change, 1977, 169 pp., no price listed (paper).*

This book is a significant contribution to psychiatry and to the history of the civil rights movement in the United States. Through a series of open-ended interviews with representative leaders in the struggle, Dr. Beardslee has recorded vignettes of personal commitment and involvement in a cause that are not easily understood in purely psychological terms.

All 11 interviews are presented with some editing, in vivid conversational style. This allows the reader to hear the stories in the interviewee's own words. The nuances of dialect and background are all there, capturing some of the flavor and beauty of the civil rights movement experience.

Of the 11 historians, 10 are black and one is a white minister. The author interviewed three black women whose roots are in the South. The seven black men are of varying ages, and have backgrounds in both the North and South. This small sample thus represents a spectrum of the leadership involved in the movement. While moving from one interview to the next the reader experiences a sense of the excitement and anticipation of personal drama. However, one wonders why the author gives fictitious names to all but three of the historians, since most of them are easily identifiable as leaders and public figures.

Dr. Beardslee's approach of interviewing leaders is probably the best way to capture the spirit of the movement in this short book. The historians are articulate and insightful. However, the reader must not forget that these individuals do not represent the typical civil rights worker. The workers Dr. Beardslee chose are those who made a long-term commitment, perhaps a lifetime commitment, to the cause of furthering human rights. They are the ones who stuck it out during the worst times. All experienced periods of profound personal stress but, nonetheless, remained in the struggle. In addition, the activists represented do not incl[ude those] either became disenchanted or whose spirit w[as broken] ing the days of violent and frustrating confr[ontations] racist southern authorities during the 1950s a[nd] civil rights workers dropped out either after a [summer] to two years. The group that is not represent[ed] given us a broader picture of the forces that [led] become involved in that historic movement. [Further,] was quite impressed with the author's caut[ious] the personal drives and ideologies that propell[ed] ers and reflected the spirituality and courage [that] them on the firing line.

What makes one stand up and continue to f[ight] under fear of imprisonment, violence, and ev[en] author realizes that such questions are ofte[n] not easily understood. Except for brief refe[rences] Erikson's theories of identity formation, [he] avoids a strictly psychiatric analysis of the r[eal] does not look for explanations in negative an[d] does he suggest that the workers were acting [out] suicidal impulses, or working through oed[ipal] Many will recall that civil rights workers w[ere] lyzed in this fashion by psychiatrists in the [1960]s.

Dr. Beardslee recognizes that the workers [experienced] ner psychological changes of great dimension[s. Much] change was a direct result of their involven[ent] workers in a just cause. People in the movem[ent] ing *with* others *for* others. This sense of co[mmunity] atmosphere of sacrifice and sharing was the s[trong] for most of the workers described in this b[ook. Most] already grounded in a strong religious spirit [now] hanced in a real human struggle to make all m[en] brothers and sisters on earth as well as in he[aven]. That activists actually brought about radical ch[ange] ca to take us one more step to the Promised L[and] the most sustaining factor in their ability to [persevere] ing on." Civil rights workers displayed a g[reat] tuitive insight when they spoke of being [on a] high."

In a closing chapter Dr. Beardslee provide[s an] analysis of the experience. Like his contemp[orary] ert Coles, he has not contented himself with [the] chair psychiatrist. He has gone into the [field] source—to learn a bit more about the sociops[ychology] heroic fighters of the civil rights movemen[t. How] nately and too quickly drifted into anonym[ity] will find this book educational as well as an [inspiration]. ly warding epic of human strength and struggl[e] the most important periods in American his[tory].

ALVIN F. POU[SSAINT, M.D.]
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Manic-Depressive Illness: History of a Synd[rome], *ed[ited by] Edward A. Wolpert, M.D., Ph.D. New [York]. International Universities Press, 1977, 587 pp., $2[5].]*

According to his introduction, Wolpert [has] this volume "to present papers historically [significant] development of our understanding of . . . [manic-depressive] illness." To this end, he included the follow[ing] by Kraepelin, two by Abraham, and two by [Lewis] cally oriented papers by Katan, Jacobson, [Lehrman, Lo]tan, Fromm-Reichmann, and Cohen and ass[ociates]. papers by Cade, Schou, and Stallone and [Lange].

netic paper by Reich and associates; sleep research papers by Hartmann and Kupfer and associates; two "biosocial attempts at synthesis" by Schildkraut and Kety and by Akiskal and McKinney; and two papers on manic-depression in children by Anthony and Scott and by Feinstein and Wolpert. Ten of these 22 papers were originally published in the decade before 1977, so Wolpert was clearly relying heavily on his own prophetic skills in determining that they are of historical significance.

The criteria for inclusion were evidently diverse. Some of the papers are widely cited classics, familiar to most readers if not in their original form then from secondary sources. Some were probably included because they are typical of a genre, some because of the historical surveys they include. The quality of the papers is variable.

Reading the hundred pages of Kraepelin's writings included here gives one an awareness of why Kraepelin was so influential: his statement is lucid, comprehensive, and has a ring of authority to it. Although some passages consist of speculations that now seem implausible, other passages would be difficult to improve on in a contemporary text.

Reading both the 1912 and the 1924 papers by Abraham, one cannot help but be aware of his preoccupation with orthodoxy. Abraham seems at least as interested in whether his views converge with those of other analytic writers as in whether his views have clear clinical support. Reading next the familiar papers by Freud, one finds the kind of clarity of thought and expression noted in Kraepelin's writings, and one knows that Freud's dominance among early analytic thinkers was not due solely to his seniority in the club. Katan's paper, to a much greater degree even than Abraham's, seems addressed only to a very limited audience of the faithful. It seems almost to embody in caricature form the common weaknesses of analytic writings—treating constructs as if they were realities and inferences as if they were observations and assuming that dynamic elements deemed present in a disorder must have etiological import.

Jacobson's paper is immeasurably more comprehensible, perhaps in part because some acknowledgment is given to real life events, with a focus not so exclusively on patients' fantasies and the theoretical propositions of other analytic writers. Freeman's paper has some of the attributes of each of the two previous ones.

The remaining three dynamically oriented papers—Levitan's, Fromm-Reichmann's, and Cohen and associates'—are excellent, readable papers. The latter two are already classics, and I agree with Wolpert that Levitan's paper will (or should) become one; it has more fresh observations in it than most of the rest of the book put together.

Of the papers on biochemistry, genetics, and sleep research, only one—Cade's 1949 paper on lithium—is clearly of historical import. The rest seem merely typical of a genre of research report and not much more important than a myriad of similar papers. The best papers in the lot, those by Schou and by Schildkraut and Kety, are somewhat dated research reviews. I would prefer to have read current statements on the topics (lithium and biogenic amines, respectively). The paper on biogenic amines also lacks a focus on manic-depressive disorder.

Anthony and Scott's contribution on childhood manic-depressive psychosis is one of the few gems that made a careful reading of the book worthwhile. Its historical, systematizing approach make it the kind of paper I wish were available for adult manic-depressive disorder, in which case it would nearly obviate the present volume. The paper also provides a context in which the case report presented in the Feinstein and Wolpert paper can be seen to be of considerable import.

I cannot imagine a situation in which I would tell anyone that he or she ought to read Wolpert's book on manic-depression. A person claiming competence in psychopathology should be familiar with most of the issues raised in it, and students in any field should, from time to time, sample the early masters directly rather than relying solely on secondary sources. However, both of these goals can be met in ways that are simpler and less expensive than buying and reading this book. On the other hand, there are few papers in the book that I would say are a waste of time to read, and there are a few papers, in particular Levitan's and Anthony and Scott's, that even very well-read psychopathologists may find new and provocative.

PAUL H. BLANEY, PH.D.
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A History of Aggression in Freud. Psychological Issues Monograph 39, *by Paul E. Stepansky. New York, N.Y., International Universities Press, 1977, 196 pp., $12.50.*

To trace and to understand the place of aggression in Freud's thinking is a difficult task given the complexities and turns which that thinking took. It is remarkable indeed that this history of Freud's views on aggression was originally formulated and drafted as a senior thesis by the author while he was an undergraduate at Princeton.

Stepansky approaches the problem from two points of view. The first is that of aggression as a metapsychological issue in psychoanalysis. The second is more hazardous: acknowledged speculations about the psychohistorical, psychobiographical, political, institutional, and personal reasons Freud might have had for his changing and often confusing positions on aggression.

Psychoanalysis as a therapy led Freud to formulate his earliest ideas about aggression. It was in his efforts to understand the hysterical and obsessional disorders that aggression had to be taken into account, but these earliest efforts were not fruitful. Eventually Freud's self-analysis and dreams about the deaths of parents and siblings as well as dreams that depicted aggressive wishes and acts for professional revenge had to be understood and explained. In "Three Essays on Sexuality" (1) aggression is depicted as a sadistic component of sexuality, but in *Jokes and Their Relation to the Unconscious* (2) aggression is given an instinctual status of its own. Stepansky holds that Freud then rejected the existence of an independent aggressive instinct because of his hostile relationship to Adler and because Adler presented a threat to psychoanalysis. Further, Stepansky repeatedly focuses on Freud's proposal of a death instinct. Few psychoanalysts will disagree that any clinical implications can be drawn from a biologically determined death instinct. More certain to stir disagreement are the suggestions that Freud's views about war were intended to avoid dealing with an innate, individual aggressive instinct, that he broke with Adler because Adler proposed an independent aggressive instinct first, and that Freud's own aggressive tendencies caused interference because of the anxiety and guilt they provoked.

Controversy about the place of aggression continues to the present day. Some contemporary psychoanalysts believe that aggression occurs in response to stimuli that evoke it. Other contemporary psychoanalysts consider it innate. Although I agree with the latter group, I would hesitate to ex-

trapolate in the way Stepansky does about the former. In some ways he joins Wittels (3, 4), Roazen (5), and others in weaving together psychoanalysis as theory and the person of its founder. This will almost certainly draw responses of the kind that Eissler (6) made to Roazen. That is to say, Stepansky's interpretation of the complex internal and external forces impinging on Freud will be challenged. His exposition of the vicissitudes of aggression in Freud's work is in a way separate from that interpretation and is a significant contribution.

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Biochemistry of Mental Disorders: New Vistas, *edited by Earl Usdin and Arnold J. Mandell. New York, N.Y., Marcel Dekker, 1978, 268 pp., $29.50.*

This volume, the latest in the Modern Pharmacology-Toxicology series, reports the proceedings of the 1975 Intra-Science Foundation Symposium. At the meeting Dr. Seymour Kety was awarded the 1975 Intra-Science Foundation Gold Medal for his fundamental contributions to our understanding of the biochemistry of mental disease. Forty-four psychiatrists and neuropharmacologists presented their views, reviews, research, and speculations concerning new vistas in this area of research. This volume includes both historical perspectives of the early research in mental disease as well as data using such modern techniques as electron spin resonance in the study of monoamine oxidase. Unfortunately, the book is far from unique in the library of the neuropharmacologist, which currently contains many proceedings of similar symposia.

As in all such proceedings, the chapters vary greatly in their quality. The chapters by Drs. Kety and Felix are very entertaining and help put current research in mental disorders into perspective. Dr. Bloom's paper on the role of the electrophysiologist in the investigation of psychiatric disorders and the discussion that follows are excellent not only for their historic insights but also for Dr. Bloom's view of the complex electrophysiological properties of catecholamines. He demonstrates that norepinephrine cannot be classifed simply as inhibitory or excitatory on postsynaptic cells but, rather, that its synaptic effects depend on the simultaneous activity of various other pathways. Reviews by Drs. Bunney, Aghajanian, Goodwin, and Dement are short, concise summaries of the state of the art as it existed in 1975. Several other papers are less useful because the studies are very preliminary and the data are not statistically convincing. Furthermore, no new or innovative techniques [illegible] These chapters include the genetic study of Dr. [illegible] lithium study of Dr. Judd and associates, and [illegible] study of serotonin metabolites in psychotic p[illegible]ts.

It was disappointing to see no discussion [illegible] nonbiogenic amine transmitters in psychiatric [illegible] no data pertaining to the relationship of per[illegible] and endocrine abnormalities to mental disea[illegible] research on these topics has been done since 1[illegible] but other psychopharmacological volumes th[illegible] such a delay in publication contain this inform[illegible] probably represent a more rational investmen[illegible] ent publication.

ANNE B. YOU[illegible]
Am[illegible]

The Whipping Boy, *by Beth Holmes. New Yo[illegible] ard Marek, 1978, 458 pp., $10.00*

The author of *The Whipping Boy* describes [illegible] book about emotionally disturbed children:

> *The Whipping Boy* exposes one of the [illegible] most terrible secrets of our legislators, jud[illegible] yers. They have a problem they pretend [illegible] ist—because they don't know how to cope [illegible] problem is that more children are totally [illegible] parental emotional abuse than by physical [illegible]

Despite the fact that the book is really not [illegible] ally abused children but, rather, about a host [illegible] psychiatric issues, it is of interest to mental [illegible] and the lay public. *The Whipping Boy* is the [illegible] derline/psychotic boy and his seriously ill mo[illegible] It chronicles his symptomatology, his family [illegible] interaction, and their cross-country search [illegible] help. An astute clinician would say that the [illegible] toms were not consistent, the family patte[illegible] nomonic, and the diagnosis, prognosis, and t[illegible] offered by the child psychiatrists questi[illegible] theless, despite its clinical inaccuracies, the [illegible] ping, and it does reveal some of the problem[illegible] sion. That is to say, for seriously disturbed [illegible] diagnose illness and recommend treatment [illegible] quate treatment facilities, carefully staffed w[illegible] able clinicians, are not always available or are [illegible]

Unfortunately, despite the clinical conditio[illegible] onist (Timmy), which is ambiguously presente[illegible] ters that surround him—his mother, his fathe[illegible] brother, the child therapists—are either good [illegible] such, lack credibility. An in-depth look at [illegible] environment is really an exercise in putting [illegible] ther rather than mother, and it is clear, at l[illegible] the protagonist is a victim of "poppism."

Despite its many flaws—in characterizat[illegible] and literary style—I found the novel gripping [illegible] a detective story with psychological insights [illegible] with the clinical accuracy and richness of no[illegible] (1) or *I Never Promised You a Rose Garden*[illegible]

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ELISSA P. BENEDEK, M.D.
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Student's Rorschach Manual: An Introduction to Administering, Scoring, and Interpreting Rorschach's Psychodiagnostic Inkblot Test, revised ed., *by Robert M. Allen, Ph.D. New York, N.Y., International Universities Press, 1978, 353 pp., $15.00.*

Described as "an expanded and updated edition" of a book first published in 1966, this revised volume totals 30 chapters, 3 of which are new. Two of the new chapters pertain to perception theories and the teaching of projective techniques in universities and practicum agencies. The third new chapter briefly discusses the Rorschach as a measure of a child's cognitive development in lieu of formal IQ testing. There is also a useful gossary of Rorschach terms and symbols and several pages on audiorecording of the Rorschach that are not found in the earlier book. Unfortunately the current text's reference section includes a mere handful of Rorschach literature citations published after the 1966 manual, omitting such comparatively recent books as Aronow and Reznikoff's (1), Exner's (2), and Goldfried and associates' (3). Thus, the present volume is not substantially different from its 1966 predecessor.

Allen gears his text to teaching the basics of administering, scoring, tabulating, and interpreting the Rorschach. He has largely succeeded in writing an eminently readable and comprehensible manual for the Rorschach neophyte. The book is organizationally sound, with the scoring and interpretive elements of the various Rorschach location areas and determinants interwoven in a meaningful fashion. The author uses illustrative material very effectively, even including a diagram of the best seating arrangements for the administration of the test. His sample protocols are well chosen and presented with clarity. Very appropriately, Allen has elected to include a "normal" case.

Even allowing for the fact that the book is intended as an introductory text, however, Allen's discussion of Rorschach content is too narrow in scope. No mention is made of the many content scales found in the literature, some of which are highly encouraging with respect to reliability and validity factors. Notable is Schafer's personality themes (4) and Elizur's anxiety scale (5). On the other hand, Allen's treatment of the content components of each of the 10 Rorschach cards, discussed individually, is very informative yet properly circumspect so as not to stimulate flights of speculative overinterpretation.

In sum, this text is, for the most part, a very reasonable choice for an elementary Rorschach course, both for teaching the fundamentals of the technique and underlining the need for the beginner to seek more advanced training.

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MARVIN REZNIKOFF, PH.D.
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Short Forms of the MMPI, *by Thomas R. Faschingbauer and Charles S. Newmark. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1978, 179 pp., $15.00.*

It is universally agreed that a short form of the MMPI is preferable to a full-length MMPI only when a full-length MMPI cannot be obtained. A full-length MMPI involves endorsing 550 statements as either true of oneself or false. This is a time-consuming task that can also be tedious; consequently, the use of the full-length test is often impractical in many research designs, especially ones that require multiple administrations of the test. In clinical settings the full-length test may not be obtained without a certain amount of stamina and willingness to cooperate on the part of the person taking the test.

Short forms of the MMPI have been developed primarily by finding the fewest items necessary either to yield a profile that matches the profile generated by the full-length test or to yield a profile from which statements about the person may be made that are useful and accurate when compared with some nontest criterion. The short forms were developed primarily among three populations: college students, psychiatric inpatients, and psychiatric outpatients. The clinical usefulness of a short form would be primarily in cases where patients are unable or unwilling to complete a full-length MMPI.

Short Forms of the MMPI renders a thorough rundown of the construction and the strengths and weaknesses of five well-developed short forms plus information on two efforts that extend or improve on a short form. The authors compare the short forms in a variety of populations and also discuss critical issues involved in the use of new forms of the MMPI that may risk the integrity of the original time-honored test. The short forms given the most attention are Kincannon's 71-item Mini-Multi (1), Dean's Midi-Multi (2), Hugo's short form (3), Faschingbauer's Abbreviated MMPI (4), and Overall and Gomez-Mont's MMPI-168 (5), which uses only the first 168 items of the standard MMPI. Two abbreviated MMPIs that are also described in this book were developed independently to improve on the Mini-Multi. These were developed by McLachlan (6) and Spera and Robertson (7); both are titled Maxi-Multi.

A clinician who respects the MMPI and is experienced in reading MMPI profiles appreciates the abundance of information it provides. Well-developed short forms are welcome when a full-length MMPI may not be obtained. However, each of these short forms is going to have to make it on its own. The short form is a related but separate test from the original. This book is a thorough guide to short forms and one would do well to read it before choosing a short form and before using any short form at all.

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Assessing Personality Through Tree Drawings, *by Karen Bolander. New York, N.Y., Basic Books, 1977, 412 pp., $20.00*

Attitudes toward projective testing always underscore vividly the regrettable divorce between clinicians and researchers. That books on assessing personality can be of interest exclusively to one group or the other is a sad reminder that the two worlds remain dissociated. In the case of *Assessing Personality Through Tree Drawings*, most clinicians will accept the idea that personality, too complex and subtle for scientific or statistical methods, can be revealed in a drawing of a tree. Personality researchers like myself will be skeptical, but the book is not written for researchers.

On the basis of her experience collecting and studying the tree drawings of thousands of people, Dr. Bolander concentrates her presentation on how to interpret tree pictures for clues and insights concerning an individual's experiential history and psychological state. Intended to instruct the novice arboritor, the text is written clearly, and the lengthy method section is organized logically. The basic information necessary to interpret central themes in a drawing is given in four key schematic figures. These schemata define the meaning of the space in which the tree is drawn and the meaning of the various tree components. The crown represents mental life, the trunk represents emotional life, and the roots represent instinctual life. Details toward the left side are feminine, details toward the right are masculine. Specific content like branches, leaves, shading, groundline, knotholes, and type of tree are described separately and allow increasingly detailed inferences. Figures and illustrations of all of these elements, individually and in combination, are plentiful.

The basic schemata and many of the interpretive guidelines are intuitively appealing—overemphasized root systems indicate being firmly rooted in reality, dead trees often indicate the feeling of complete victimization by external forces, knotholes and scars indicate traumatic experiences. The problem here, as with other similar interpretive systems, is determining whether there is any usefulness to the schemata beyond their intuitive appeal. Moreover, even if the basic schemata are accepted, when do interpretive guidelines and generalizations apply to a particular case? Unfortunately, despite the wealth of data available to the author, very little evidence is presented to substantiate any of the method. The book is written with the understanding that the answers to these questions lie ultimately in each user's own evaluation of the method. This position is not unreasonable, given the current state of clinical assessment, but it again illustrates the wide gap between clinical and research perspectives.

It would be unfair to judge this work against [illegible] lems of clinical psychology, psychiatry, and personality assessment. This is a perfectly appropriate and [illegible] for clinician users of projective tests and for [illegible] especially. Used in conjunction with other sources of information about an individual, as the author recommends, many practitioners will find this interpretive system helpful.

Despite the skepticism and the critics, projective approaches to personality assessment survive. [illegible] human personality will always tolerate and test attempts at understanding a little more.

RICHARD J. H[illegible], PH.D.
Bet[illegible]

Interpreting Psychological Test Data, Vol. I, *by [illegible] bert, Ph.D. New York, N.Y., Van Nostrand [illegible] 1978, 237 pp., $15.95.*

This is the first of two volumes of this text, [illegible] now in preparation. The primary purpose of both [illegible] to associate personality and behavior with [illegible] tained from the four tests that are most often used [illegible] porary psychodiagnostic evaluation: the Bender Gestalt Test, the Human Figure Drawing Test, the Wechsler Adult Intelligence Scale (WAIS), and Rorschach Psychodiagnostic Inkblot Test.

This volume "abstracts and synthesizes [illegible] books regarded as standard references in the [illegible] pects of behavior which are assumed to be related [illegible] psychological test productions" (p. vii). The "[illegible] erences" abstracted are certainly authoritative [illegible] the tests included. Several equally applicable [illegible] reference sources are excluded, however. This [illegible] is readily admitted by the author: "For the [illegible] foundations provided by the cited references [illegible] suffice" (p. vii). Unfortunately, the limitations [illegible] volume will apply to the forthcoming second volume. The author promises that volume II "will bring together in summary form the diagnostic syndrome of behavioral attributes suggested by test responses" (p. ix).

If, as the author states, one of his future objectives is to expand the present volume (p. vii), the contemporary usefulness of the second volume faces limitations similar to obsolescence. Volume II will refer to diagnostic syndromes based on criteria used in the first and second editions of *DSM III*. The fact that the second edition of *DSM* (1) is currently being revised (2) adds further to the obsolescence of these volumes. Another example of this dilemma is noted in the chapter in which the author devotes approximately 50 pages of pagination to the interest content of the Rorschach. The entire text on interest content of this test has been recently published (3).

Another major critical issue is the exclusion of [illegible] sources from texts that would assure adequate [illegible] sample of data for specific age groups, especially [illegible] This particularly applies to texts on the Bender-Gestalt test. Koppitz' work (4), which is entirely devoted to normative data for children in different age groups, is not [illegible] From a contemporary point of view, the Bender is extremely useful in learning disorders and certain [illegible] tion. The author also fails to include the Wechsler Intelligence Scale for Children (WISC-Revised) [illegible]

of only the WAIS limits the use of this reference source for people who work with children.

Almost two-thirds of this volume is devoted to the Rorschach test. This is justifiable in light of the frequency of its use in psychodiagnostic procedures. However, half of this portion of the book concerns interest content. Since volume II purportedly will relate these data to diagnostic nomenclature, more objective and normative Rorschach data will be needed. Likewise, the author's effort to equate the several Rorschach scoring systems (p. vii) has already been accomplished in another text (5).

The author's question of "whether the present volume fulfills a useful role in the profession" (p. vii) is certainly open-ended. Perhaps the introduction is most pertinent in expressing the author's awareness of the critical theoretical issues that limit the valid and reliable usefulness of this volume. One purpose of this text—"to provide a rapid useful reference source" (p. vii)—has been accomplished. However, the scope of the sources used limits even this.

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Depression: Biology, Psychodynamics, and Treatment, *edited by Jonathan O. Cole, Alan F. Schatzberg, and Shervert H. Frazier. New York, N.Y., Plenum Press, 1978, 242 pp., $22.50.*

This book consists largely of material presented at a one-day symposium on depression held in March 1976. No doubt those who attended that symposium were favorably impressed with the relative newness of what the speakers said. A more than two-year delay in the publication of this material has aged it, compelling me to assert that now these pages contain little that is new or helpful for the basic scientist or the clinician. In fact, a good portion of this volume has been published in either journals or books, including the proceedings of other symposia on depression. Furthermore, the book has many of the deficiencies of multi-authored symposium proceedings. It is neither comprehensive nor an in-depth coverage of the subject matter; therefore, it is very difficult to decide for whom this book should be recommended. It is already outdated. It is a historical record of the views of some American experts on various aspects of depression in 1976. Its contents do not justify purchase by students of psychiatry, and it certainly is not designed for those working in the field of depression.

FRANK J. AYD, JR., M.D.
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The Child Protection Team Handbook: A Multidisciplinary Approach to Managing Child Abuse and Neglect, *edited by Barton D. Schmitt, New York, N.Y., Garland STPM Press, 1978, 407 pp., $24.50.*

In their scrupulous effort to avoid omitting essential data, most handbooks and compendia, including this one, tend to exaggerate the value of vigorous adherence to the practices and procedures the authors have found comfortable. If and when such handbooks become the standard, to take unsanctioned shortcuts is deemed slipshod, to be casual is thought uncaring, and to vary from the procedures may be malpractice. When the interdisciplinary approach is overemphasized, as it is in this handbook, the procedures outlined become cumbersome and time-consuming. The careful delineation of the role of the different disciplines, respectful as it is to the contribution that each discipline can make, becomes role padding when these different perspectives add little new dimension to the solution of problems. Similar insights derived from different sources are no less repetitious than when they come from a single source. What such role clarification does for the child-abusing parent is conjectural. On the one hand, it may be flattering to have so many important individuals preoccupied with him or her. On the other hand, the help of so many specialists focused on his or her many difficulties may make the parent feel like an insignificant figure in a bureaucratic maze.

Some of the statements in this text are more likely to raise eyebrows than receive enthusiastic affirmation. That 80% of these parents are treatable and that most of them love their children are questionable theses, no matter how loosely we define treatment or love. The necessity of parenting does not make parenting an automatic virtue. If child-abusers could accept parenthood as neither virtue nor vice, they might be less likely to exaggerate the investment parenthood entails with less fruitless anxiety about their lack of preparation for parental responsibilities. The hostility of many of these parents to their children derives in part from their ignorance and unsophistication. Since many of them are unprepared for the struggles and complexities of interpersonal relationships, it is logical that they see their children as their equals and hence willful opponents to their wills. In a sense, child and parents are both in the "flyweight" class. To make "heavyweights" of these parents, to reduce their arrogance and stubbornness, requires not only instruction in childrearing but some understanding of interpersonal dynamics.

The authors have made a laudable and conscientious effort to develop a team approach to the diagnosis and treatment of child abuse. They have collected and collated much of the technical information necessary for the child abuse teams now proliferating in this country. At times they overemphasize tact, which becomes indistinguishable from timidity. The emphasis on tact undoubtedly reflects the authors' obvious respect for their colleages and their patients.

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Orange, N.J.

Ecologic-Biochemical Approaches to Treatment of Delinquents and Criminals, *edited by Leonard J. Hippchen, Ph.D. New York, N.Y., Van Nostrand Reinhold Co., 1978, 392 pp., $16.95.*

No book that recognizes a biochemical contribution to antisocial behavior can be all bad—at least so I thought until

I read *Ecologic-Biochemical Approaches to Treatment of Delinquents and Criminals*. Of course, until then I was not aware that corporal and capital punishment were biochemical treatments.

This book consists of 20 chapters written by individuals from a variety of disciplines, including a "licensed medical hypnotist," an optometrist, and an "orthomolecular family physician." Topics range from the etiology of learning disabilities ("children with learning and behavior disorders are vitamin B_3-B_6 dependents. A second major cause of perceptual changes is food allergies" [p. 14]) to the treatment of penitentiary inmates ("In my opinion we need capital and corporal punishment more now than ever before, because more people are getting into this mindless state where life means nothing" [p. 277]). The author of these words clearly felt that "life means nothing."

Topics also include the orthomolecular treatment of antisocial behavior ("The three most common forms of criminal malnutrition are . . . the vitamin B dependencies, . . . the saccharine diseases [the relative hypoglycemias], . . . the cerebral allergies" [p. 4]) and the effect of tinted sunglasses on behavior ("Three students constantly wore 'hot-pink' sunglasses, and a check on faculty ratings indicated that these same students were also considered to be the most psychologically disturbed students in the college" [p. 109]). The obvious fact that the choice of a particular style rather than its effect on behavior might be associated with maladaptation is not appreciated. Most unfortunate, in light of the emphasis in this book on so-called cerebral allergies, is the failure of contributors to distinguish between allergic and toxic reactions to substances. For example, one of the authors cites the following as an example of neuroallergy: "Befuddled thinking or an acute episode of mental illness may occur upon exposure to petroleum fumes, carbon tetrachloride, the fumes of a gas range, or the odor of a magic marker" (p. 239). This is not allergy!

A critical reader of this book might wrongly discount the possible contribution of malnutrition to maladaptation. There are reputable investigators in the field of nutrition and behavior whose work, although not included in this book, deserves attention. Unfortunately, the contributions of Cravioto and Delecardi, Wurtman, Pollitt, and others are by and large ignored.

Unfortunately, claims in this book are so grandiose (e.g., "The evidence suggests that recidivism rates among prison populations could be reduced 25–50% with the addition of orthomolecular treatment" [p. 16]), any kernels of truth this book might contain are lost. The association of malnutrition and maladaptation is made to look ridiculous rather than worthy of further scientific investigation.

It would seem that faith rather than scientific knowledge guided the pens of the authors. Perhaps this book would have been reviewed more appropriately under the category of religion or science fiction.

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John of God: His Place in the History of Psychiatry and Medicine, *by Ruben D. Rumbaut, M.D. Miami, Fla., Ediciones Universal, 1978, 70 pp., $4.00 (paper).*

The saints of all religious or spiritual traditions are interesting, not only in theological or cultural terms but also in psychological—one might also say psychiatric—terms. Juan Ciudad Duarte, who became known to history as Juan de Dios, is certainly no exception. His story is told in these pages simply and sparely: his birth to a poor Portuguese family, his running away from home with an itinerant clergyman at the age of eight, his subsequent adventures as a Moorish soldier, his return to his home years later to find the household in grief and tragedy precipitated by his departure, the subsequent misfortunes leading him to increasingly desperate straits, and, finally, the crucial conversion at the hands of Juan de Avila, the famous Apostle of Andalusia. Hearing Avila's fiery sermon apparently served as a cathartic experience, plunging Juan into a psychotic episode that appeared predominantly depressive in character.

The second part of the story starts with the post-conversion transformation of Juan Ciudad Duarte into Juan de Dios. Dr. Rumbaut then provides an account of Juan de Dios' saintly works and the founding of the distinguished Order of the Hospitallers of St. John of God and their worldwide work of caring for the sick, particularly the mentally ill and neurologically afflicted.

The story is well told and worth the retelling. There are certain interesting correspondences with the life of Juan's contemporary, Inigo de Loyola, who became known to history as Ignatius of Loyola, the founder of the Society of Jesus and the architect of the Counterreformation. Ignatius was born about the same time as Juan and served as an officer in the Spanish army. In fact, he was one of the garrison, in which Juan de Dios is said to have been a foot soldier. One wonders if these two men might have crossed each other's paths. In any case, Ignatius also had his trauma, his psychotic transformation, and he emerged from this soul-wrenching experience in Manresa with a new spiritual identity.

Such marvels of the human spirit excite the greatest imagination of the psychologically minded, but the present work offers us little more than a simple recounting of the events. There is little in the retelling that allows a distinguish between hagiographical mythology and authentic biography. There is no attempt to scrutinize or understand the psychological journey of this fascinating man. Moreover, the somewhat innocent suggestion that the famous Apostle served Juan de Dios in somewhat the role of a psychotherapist hardly matches the details of the account to the effects of the apostle's sermon on the distraught and tormented Juan. Anyone who undertakes to read this delightful account (the book is a bilingual edition printed in English and Spanish so that the whole story is told in less than 30 pages) will find in it a memorial to one of the great saints. However, if one looks here for any deeper understanding, any exploration of the dark continent of human psychology and spiritual experience, or even any attempt to illuminate the unique conversion experience of Juan de Dios, he will seek in vain.

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Mothers, Fathers, and Children: Explorations into the Formation of Character in the First Seven Years, *by [illegible] and Sidney Axelrad, with Ethel Horn, Marshall Lebovich, and Marvin Taylor. New York, N.Y., International Universities Press, 1978, 659 pp., $27.50.*

This book is a follow-up and expansion of an earlier one, titled *Anxiety and Ego Formation in Infancy* (1[illegible]

the authors reported a study of 131 mothers and their infants during the first year of life. For various reasons, the authors decided to follow 127 of the mothers and their children from the original study. Their intent was to determine whether the original patterns of mother-infant interactions continued relatively unchanged or varied during the ensuing years until the onset of latency. The authors are psychoanalytically oriented, and they felt that direct interviews with mothers and observations of children could contribute to our knowledge beyond the analytic process.

The book begins with an overview of the earlier study. Part one goes into detail about the methods used in that study. The mothers could have been divided into at least seven subgroups but, for statistical reasons, were separated into two types, A and B. The type A mothers were good parents who provided consistent emotional nurturance and support to their infants into the later years of childhood. Type B mothers were lacking in many of the ingredients of what one might consider good motherhood. The authors are quick to state that this does not separate the A and B mothers into good and bad, but the B mothers had more difficulty in adapting to the maternal role and were overprotective, underprotective, defensive, disinterested, or showed a variety of other patterns not conducive to good ego and personality development in their infants and children.

After their explanation of the method used, the authors present nine case reports of mothers and their infants. Part two, The Longitudinal Study: Mothers and Children, deals primarily with the follow-up of the original group of infants. The research group found that the children of type A mothers were better adjusted both at home and at school and had more adequate personality development than did the children of type B mothers. With the exception of more age-appropriate drawings in the A group, many of the four- and five-year-old youngsters in both groups gave similar impressions to the researchers at the height of the oedipal period. The group A children subsequently reassumed more normal overt development than did the group B children.

The study seems to indicate that some assumptions about changeability in parent or child are not necessarily valid: the concept that parental adequacy depends on the ability to shift from one to another type of identification with the child when the youngster enters a new developmental phase, the idea that insufficient or inappropriate parental responses during one phase of growth can be made up for in a subsequent period because of the child's resilience, the idea that a "good fit" between mother and child is important not only to their ongoing relationship but the child's progress. Although the authors agree that all of these points are of importance, they may not be quite so important as has been believed. In the summary of this portion of the book, the authors say,

> It is within the child's capacity very gradually to form a post-ambivalent connection to the parents and through them to more distant objects; to integrate the early affective bonds with motor, cognitive and language development— . . . to build an intact body ego and broaden capacities for gratifying experiences with a whole world of objects.

Part three, Fathers and Children, begins with a chapter on variations in paternal behavior and attitudes and then presents in some detail 10 case reports of mothers, fathers, and children. As with the preceding portion of the book, the cases are divided between parents who are more adequate and those who are less so.

This book is a report of a time-consuming study of prelatent children and their parents. The authors rightfully point out several times that it has some scientific flaws in methodology; nevertheless, it is basically a useful book. Perhaps it could have been written without such detailed case histories, but omissions might have left the reader in some doubt as to how the results were obtained. The basic constitution of the infant is taken into account as one possible variant among others, such as trauma resulting from repeated hospitalizations.

If one were to be critical of the case illustrations, such criticism might have to do with the omission of a study of why these couples did or did not plan their families. Although some attention is paid to the ages of the siblings and the ordinal position of the subject children, the authors do not seem to have asked the simple question, "What size family do you plan?" There also seems to have been a timidity on the part of the researchers to offer more active guidance to the parents. Perhaps this was not one of their intents, but it would seem appropriate in a study of this kind to know about family planning or the lack of it and about the communication between mother and father in regard to it. One would like to know whether active intervention of a preventive type could have been useful; however, since all of these parents were volunteers and could have refused to participate in the study at any time, the authors are to be congratulated for keeping so many involved.

The main and important thrust of this book remains clear: interaction between mother and infant in the first year of life is basic to future character and personality formation. Equally important is the fact that the personalities of the mothers and fathers continue along the same lines as were drawn in the first year of life. The book is to be recommended to anyone working with infants and children—child and general psychiatrists, psychologists, social and public health workers, nurses, and others in related fields.

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1. Brody S, Axelrad S: Anxiety and Ego Formation in Infancy. New York, International Universities Press, 1971

STUART M. FINCH, M.D.
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Psychoanalysis and the History of the Individual, *by Hans W. Loewald, M.D. New Haven, Conn., Yale University Press, 1978, 77 pp., $7.95.*

This book is an edited version of the Freud-Kanzer lectures given at Yale University by Dr. Loewald. The author tackles such difficult and knotty psychoanalytic problems as morality, transference love, and religion. He notes that most analysts have continued in Freud's tradition and, as a result, are not comfortable exploring morality, love as opposed to sexuality, and religion.

Toward the end of the book, the author remarks that these lectures gave him an opportunity to say things he has had on his mind for a long time. This honest reevaluation of accepted ideas is reminiscent of Foulks (1), Grotjahn (2), and Mandell (3). The presentation is graceful and easily read. It is a disciplined but fanciful flight of imagination and intellect, a presentation of ideas rather than personal feelings and reactions. In this respect Loewald differs slightly from Foulks, Grotjahn, and Mandell. Loewald's overall approach seems

detached, objective, and free-flowing. At first I thought he was a philosopher. He is an analyst, but he seems to be at that point in life when men and women try to objectively evaluate themselves, their lives, and their professions.

Essentially, Loewald concludes that man must accept his id. This is the essence of morality. Such acceptance does not mean burying guilt or creating "fancy sublimations" because these things cannot be done. The id must be acknowledged as a part of oneself. Its manifestations cannot be blamed on, nor are they the responsibility of, society, God, parents, or anything else. Man is ultimately responsible. His moral task is to bring the id into the more lucid levels of mature thinking. The task is not to give up, deplore, undo, or deny it. It must be seen, understood, accepted, and worked on.

Transference and transference love, if analytically interpreted and worked through, should allow the patient a free, fanciful, libidinal, symbol-forming relationship among his id, ego, and superego as well as between himself or herself and object representations.

Dr. Loewald is impatient with most analysts' overly simplistic views of religion. He blames this on Freud's discomfort with religion. Neither Freud nor subsequent analysts seriously tackled this question. Loewald notes the similarity between sexual and religious ecstasy and the extreme vagueness conveyed by the term "sublimation." Religious experience and religious myths are important expressions of unconscious mentation. They are not just dependency, magical thinking, denial, or other defenses. Currently, religious feelings are more deeply repressed than was Victorian sexuality, and they are therefore more deserving of analytic attention.

This brief summary does not convey how the book forces one to think about issues not yet adequately explored by psychoanalysis. The reminder is subtly disturbing. I recommend the book to all psychoanalysts and to those psychiatrists with special philosophical and metapsychological interests.

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1. Foulks SH: Group Analytic Psychotherapy. New York, Science Publishers, 1976
2. Grotjahn M: The Art and Technique of Analytic Group Therapy. New York, Jason Aronson, 1977
3. Mandell A: Coming of Middle Age. New York, Summit Books, 1977

FRANK M. KLINE, M.D.
Long Beach, Calif.

Crisis Intervention As Psychotherapy, *by Charles Patrick Ewing. New York, N.Y., Oxford University Press, 1978, 125 pp., $7.95; $3.50 (paper).*

Crisis intervention is quite widely believed to be a good thing—ubiquitous as a term but not necessarily as a recognizable practice. One wonders whether part of its success might be due to its amorphous adaptability. It has become, for some, one of those vague vogue words (such as "family," "hotline," "access," "community," and "counseling") which one can add to the description of nearly any clinical or teaching program, like monosodium glutamate, to spice it up and make the flavor more acceptable without actually altering the contents. "There is a lot of it about," as one says during influenza epidemics, but one is less than clear exactly what it is.

Dr. Ewing's book covers quite a lot of territory relatively superficially, at the level of an extended review article, without really getting its teeth into the subject as one might have hoped. He presents a definition of crisis intervention that is a paraphrase of the popular definition of psychotherapy, adding only that its techniques are "derived from the established principles of crisis theory." Yet nowhere does one get any clear idea of any principle, practice, or technique that is unique to crisis intervention. It is never clear what distinguishes crisis intervention from any other variety of short-term psychotherapy. Indeed, there is a very loud silence on this point in the book. There is no mention at all of the highly relevant allied work on short-term and brief psychotherapy, and the work of Sifneos and Malan seems studiously ignored. This greatly reduces the clarity of the book's attempt to place crisis intervention in the general field of psychotherapy.

Ewing starts by describing basic crisis intervention theory, which seems a rather fragile foundation for what is to be built on it and claimed for it and somewhat lacking in validation. He describes some general principles of clinical practice (in no way distinct from other short-term therapies) and some current uses of crisis intervention in child and family therapy as well as with special client groups. There is a short review of some studies evaluating such programs, some thoughts on possible future developments, and a suggested model for clinical practice.

One chapter discusses the selection of appropriate clients, and is, probably appropriately, uncertain of criteria. There is a suggestion that crisis intervention may be better suited to clients of lower socioeconomic status than some other psychotherapeutic modalities. Otherwise the usual suspicion that the ideal client for any therapy is someone who is not too sick, only recently disturbed, highly motivated, and likely to do equally well if given macrame lessons applies. Comments like "Crisis theory . . . presupposes that crises are self-limiting and last for only a period of weeks" suggest either naïve theory or special pleading by choosing the crises likely to respond well to any intervention, while ignoring chronic crisis states or crises so overwhelming that they last for more than a few weeks. This is also in conflict with a good deal of high quality work studying crises like bereavement, which, although undoubtedly crises, are very rarely if ever resolved within weeks or even months, unless one is very easily satisfied with superficial criteria for "resolution."

The final disappointment about this book is the lack of a really critical approach to the issues raised. It will be comforting for those already convinced of the importance of crisis intervention techniques. Those who still wait to be convinced that there is a substantive and substantiated theoretical background and a distinctive and effective clinical methodology in crisis intervention are still waiting.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Mental Health Almanac, *edited by Robert D. Allen and Marsha K. Cartier. New York, N.Y., Garland STPM Press, 1978, 403 pp., $17.50.*

The Child in His Family, Vol. 5: Children and Their Parents in a Changing World, *edited by E. James Anthony, M.D., and Colette Chiland, M.D., Ph.D. New York, N.Y., John Wiley & Sons, 1978, 440 pp., $23.95.*

Bioethics and Human Rights, *edited by Elsie L. Bandman, R.N., Ed.D., and Bertram Bandman, Ph.D. Boston, Mass., Little, Brown and Co., 1978, 366 pp., $9.95 (paper).*

Counseling Metapsychology, *by Arthur Burton, Ph.D. Sacramento, Calif., Hamilton Psyche Press, 1978, 158 pp., $14.50.*

Relaxation: A Comprehensive Manual for Adults, Children, and Children with Special Needs, *by Joseph R. Cautela and June Groden; illustrations by Phyllis Hawkes. Champaign, Ill., Research Press Co., 1978, 91 pp., $6.95 (spiral-bound).*

The Practicing Physician's Approach to Headache, 2nd ed., *by Seymour Diamond, M.D., and Donald J. Dalessio, M.D. Baltimore, Md., Williams & Wilkins Co., 1978, 148 pp., $21.50.*

The Rorschach: A Comprehensive System, Vol. 2, *by John E. Exner, Jr., Ph.D. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1978, 422 pp., $32.95.*

Healing: Implications for Psychotherapy, *edited by James L. Fosshage, Ph.D., and Paul Olsen, Ph.D., with Kenneth A. Frank, Ph.D., Henry Grayson, Ph.D., Clemens A. Loew, Ph.D., Henry Lowenheim, Ph.D., and Glen Boles, Ph.D. New York, N.Y., Human Sciences Press, 1978, 377 pp., $15.95.*

What Do Women Want: Self-Discovery Through Fantasy, *by Lucy Freeman. New York, N.Y., Human Sciences Press, 1978, 187 pp., $9.95.*

Down's Syndrome and the Family: The Early Years, *by Ann Gath. London, England, Academic Press (Harcourt Brace Jovanovich), 1978, 128 pp., $13.25.*

Treatment in Crisis Situations, *by Naomi Golan. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 253 pp., no price listed.*

Psychiatric Aspects of Imprisonment, *by John Gunn, Graham Robertson, Susanne Dell, and Cynthia Way. London, England, Academic Press (Harcourt Brace Jovanovich), 1978, 312 pp., $25.00.*

Dying: A Psychoanalytic Study with Special Reference to Individual Creativity and Defensive Organizations, *by Tor-Bjorn Hagglund. New York, N.Y., International Universities Press, 1978, 253 pp., $15.00.*

The Care and Training of the Mentally Handicapped: A Manual for the Caring Professions, *by Charles H. Hall, S.R.N., William I. Fraser, M.D., and Ronald C. MacGillivary, M.D. London, England, John Wright & Sons, 1978, 278 pp., $19.95.*

Progress in Behavior Modification, Vol. 6, *edited by Michel Hersen, Richard M. Eisler, and Peter M. Miller. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1978, 248 pp., $18.50.*

Clinical Pharmacology of Psychotherapeutic Drugs, *by Leo E. Hollister, M.D. New York, N.Y., Churchill Livingstone, 1978, 226 pp., $19.50.*

Minimal Brain Dysfunction: Fact or Fiction. Advances in Biological Psychiatry, Vol. 1, *edited by A.F. Kalverboer, H.M. van Praag, and J. Mendlewicz. Basel, Switzerland, S. Karger, 1978, 107 pp., $25.50 (paper).*

Behavioral Approaches to Alcoholism, *edited by G. Alan Marlatt and Peter E. Nathan. New Brunswick, N.J., Rutgers Center of Alcohol Studies Publications Division, 1978, 209 pp., $6.00 (paper).*

The Career Resource Center, *by John Meerbach, Ed.D. New York, N.Y., Human Sciences Press, 1978, 149 pp., $12.95.*

The Politics of Pain, *by Helen Neal. New York, N.Y., McGraw-Hill Book Co., 1978, 212 pp., $9.95.*

Minds and Motion: The Riddle of Psychokinesis, *by D. Scott Rogo. New York, N.Y., Taplinger Publishing Co., 1978, 266 pp., $11.95.*

The Disposable Parent: The Case for Joint Custody, *by Mel Roman and William Haddad with the assistance of Susan Manso. New York, N.Y., Holt, Rinehart and Winston, 1978, 206 pp., $8.95.*

Homosexuality, *by Charles W. Socarides, M.D. New York, N.Y., Jason Aronson, 1978, 626 pp., $30.00.*

Expanding Dimensions of Consciousness, *edited by A. Arthur Sugerman and Ralph E. Tarter. New York, N.Y., Springer Publishing Co., 1978, 297 pp., $16.95.*

Psychosocial Therapy, *by Francis J. Turner. New York, N.Y., Free Press (Macmillan Publishing Co.), 1978, 240 pp., no price listed.*

Transcultural Counseling: Needs, Programs, and Techniques, *edited by Garry R. Walz, Ph.D., and Libby Benjamin, Ph.D. New York, N.Y., Human Sciences Press, 1978, 213 pp., $13.95.*

Understanding Sexual Attacks: A Study Based Upon a Group of Rapists Undergoing Psychotherapy, *by D.J. West, C. Roy, and Florence L. Nichols. London, England, Heinemann Educational Books, 1978, 175 pp., $25.95; $8.95 (paper).*

Foundations' Fund Fellowships for Sabbatical Research in Psychiatry and Its Basic Sciences

The Foundations' Fund for Research in Psychiatry announces a limited program of support for scholars on sabbatical leave in order to further their research and contribute to the knowledge of psychiatric diagnosis, treatment, and prevention. The sabbatical must be spent away from the home institution at an internationally recognized institution.

Applications are open to distinguished and creative investigators with demonstrated research contributions in the field who hold full-time positions in professional schools and graduate departments of universities or equivalent institutes of research. Applicants must be U.S. or Canadian citizens or permanent residents of the U.S. or Canada.

The deadline for receipt of applications is May 1 of the year preceding the proposed sabbatical.

Information may be obtained from:

Foundations' Fund for Research in Psychiatry
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New Haven, Connecticut 06511

ANNUAL INDEX

Following is the comprehensive index for volume 135 of the *Journal*, covering all material from January 1978 through December 1978. The complete citation for each article in the author index is listed under the name of the first author. Joint authors are listed alphabetically with a cross-reference to the first author; cross-references containing more than one first author name separated by semicolons indicate multiple articles by the joint author. Book reviews appear in the author index under the name of the reviewer and in the subject index under the heading Books Reviewed; the books are arranged alphabetically by the surname of the book's first author.

Effective this year, listings in the subject index are according to key words rather than subject categories. In addition, all entries are in abbreviated style. Each entry in the subject index is identified by the surname of the first author only; semicolons indicate multiple works (e.g., letters to the editor). Two-letter abbreviations for the months are used, and entries other than regular articles, brief communications, and clinical and research reports are given the following designations: book review (bk rev), letter to the editor (ltr to ed), editorial (edt), in memoriam (in mem), and official actions (off acts).

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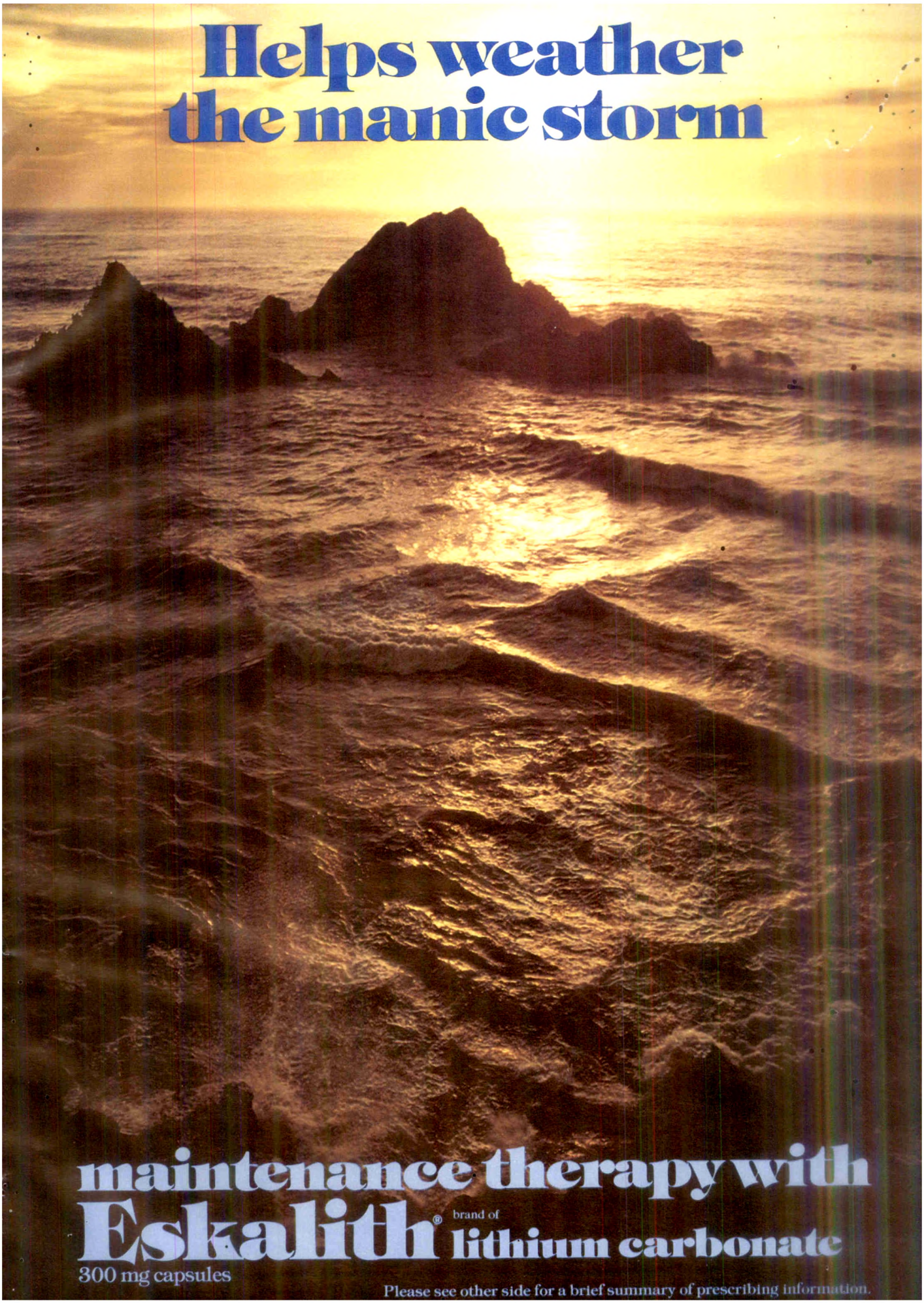
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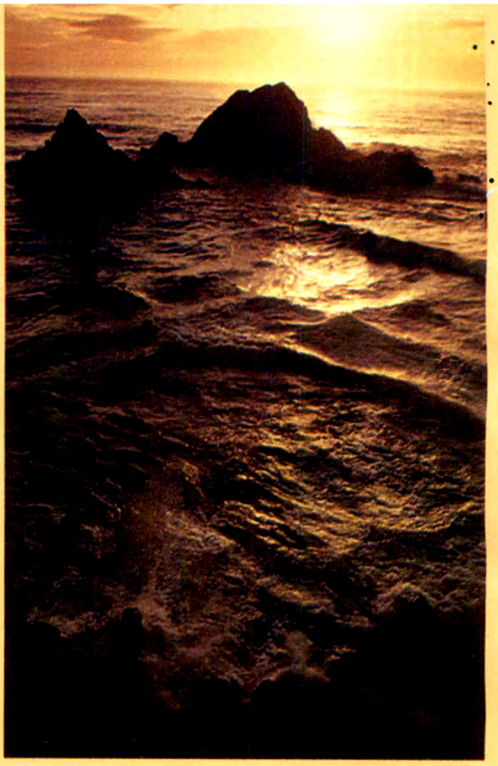
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WARNING
Lithium toxicity is closely related to serum lithium levels, and can occur at doses close to therapeutic levels. Facilities for prompt and accurate serum lithium determinations should be available before initiating therapy.

Indications: Treatment of manic episodes of manic-depressive illness. Maintenance therapy prevents or diminishes the intensity of subsequent episodes in manic-depressive patients with a history of mania.

Warnings: Lithium should generally not be given to patients with significant renal or cardiovascular disease, severe debilitation or dehydration, sodium depletion, or to patients receiving diuretics.

Lithium therapy has been reported in some cases to be associated with morphologic changes in the kidneys.

Caution patient and family to watch for diarrhea, vomiting, tremor, mild ataxia, drowsiness, or muscular weakness as signs of lithium toxicity, and to discontinue therapy and contact a physician should they occur. Patients receiving combined therapy with lithium and an antipsychotic should be monitored closely for early evidence of neurologic toxicity and treatment discontinued promptly if such signs appear. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery).

Lithium should not be used in pregnancy, especially during the first trimester, unless potential benefits outweigh possible hazards.

Not recommended for children under 12.

Precautions: Lithium tolerance is greater during the acute manic phase and decreases when manic symptoms subside.

Lithium therapy may lead to sodium depletion. Normal diet (including salt) and adequate fluid intake (2500-3000 ml.) must be maintained, at least during initial stabilization period. Protracted sweating or diarrhea can decrease tolerance; in such cases, administer supplemental fluid and salt.

Sweating, diarrhea, and concomitant infection with elevated temperatures may require temporary reduction or cessation of dosage.

Adverse Reactions: Mild to moderate toxic reactions may occur at serum lithium levels from 1.5 to 2.5 mEq./l., and moderate to severe reactions at levels from 2.0 to 2.5 mEq./l. Fine hand tremor, polyuria, and mild thirst may occur during initial therapy and persist. Transient and mild nausea and general discomfort also appear during initial therapy. These effects usually subside with continued treatment or temporary reduction or cessation of dosage. If persistent, discontinue dosage.

Diarrhea, vomiting, drowsiness, muscular weakness, and lack of coordination may be early signs of toxicity and may occur at levels below 2.0 mEq./l. At higher levels, ataxia, giddiness, tinnitus, blurred vision, and a large output of dilute urine may be seen. Serum levels above 3.0 mEq./l. may produce a complex clinical picture, involving multiple organs and systems. Serum levels should not exceed 2.0 mEq./l. during acute phase.

The following reactions appear to be related to serum lithium levels, including levels within the therapeutic range: **Neuromuscular**—tremor, muscle hyperirritability (fasciculations, twitching, clonic movements of whole limbs), ataxia, choreo-athetotic movements, hyperactive deep tendon reflex; **Central Nervous System**—blackout spells, epileptiform seizures, slurred speech, dizziness, vertigo, incontinence of urine or feces, somnolence, psychomotor retardation, restlessness, confusion, stupor, coma; **Cardiovascular**—cardiac arrhythmia, hypotension, peripheral circulatory collapse; **Gastrointestinal**—anorexia, nausea, vomiting, diarrhea; **Genitourinary**—albuminuria, oliguria, polyuria, glycosuria; **Dermatologic**—drying and thinning of hair, alopecia, anesthesia of skin, chronic folliculitis, exacerbation of psoriasis, xerosis cutis; **Autonomic**—blurred vision, dry mouth; **Thyroid Abnormalities**—euthyroid goiter and/or hypothyroidism (including myxedema) with lower T_3 and T_4. I^{131} uptake may be elevated; **EEG Changes**—diffuse slowing, widening of the frequency spectrum, potentiation and disorganization of background rhythm; **EKG Changes**—reversible flattening, isoelectricity or inversion of T-waves; **Miscellaneous**—fatigue, lethargy, transient scotomata, dehydration, weight loss, tendency to sleep.

Reactions unrelated to dosage include: transient EEG and EKG changes, leukocytosis, headache, diffuse nontoxic goiter with or without hypothyroidism, transient hyperglycemia, generalized pruritus with or without rash, cutaneous ulcers, albuminuria, worsening of organic brain syndromes, excessive weight gain, edematous swelling of ankles or wrists, thirst or polyuria, sometimes resembling diabetes insipidus, and metallic taste. A single case of a syndrome resembling Raynaud's has been reported.

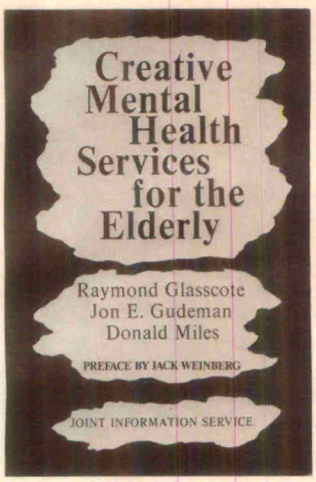
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The Joint Information Service of the American Psychiatric Association and the Mental Health Association releases Two New Volumes That

Focus on the Elderly



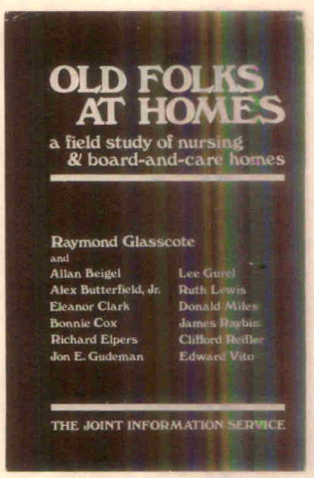
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- a carefully coordinated system of "respite hospitalization," which promotes the health of the elderly while allowing maximum use of hospital beds and family resources
- a "Lucy Booth," patterned after the Peanuts comic strip, which provides easy access to many kinds of services
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Concerned about the very bad reputation that nursing homes have received from many quarters in recent years, the Joint Information Service set up a field study to visit a systematically chosen sample of nursing homes, and board-and-care homes as well, to see to what extent the care of and the quality of life for the patients differed from the geriatrics units of state hospitals. Altogether, sixty nursing homes and 31 board-and-care homes in ten locales were visited, in each case by the senior author accompanied by two mental health professionals.

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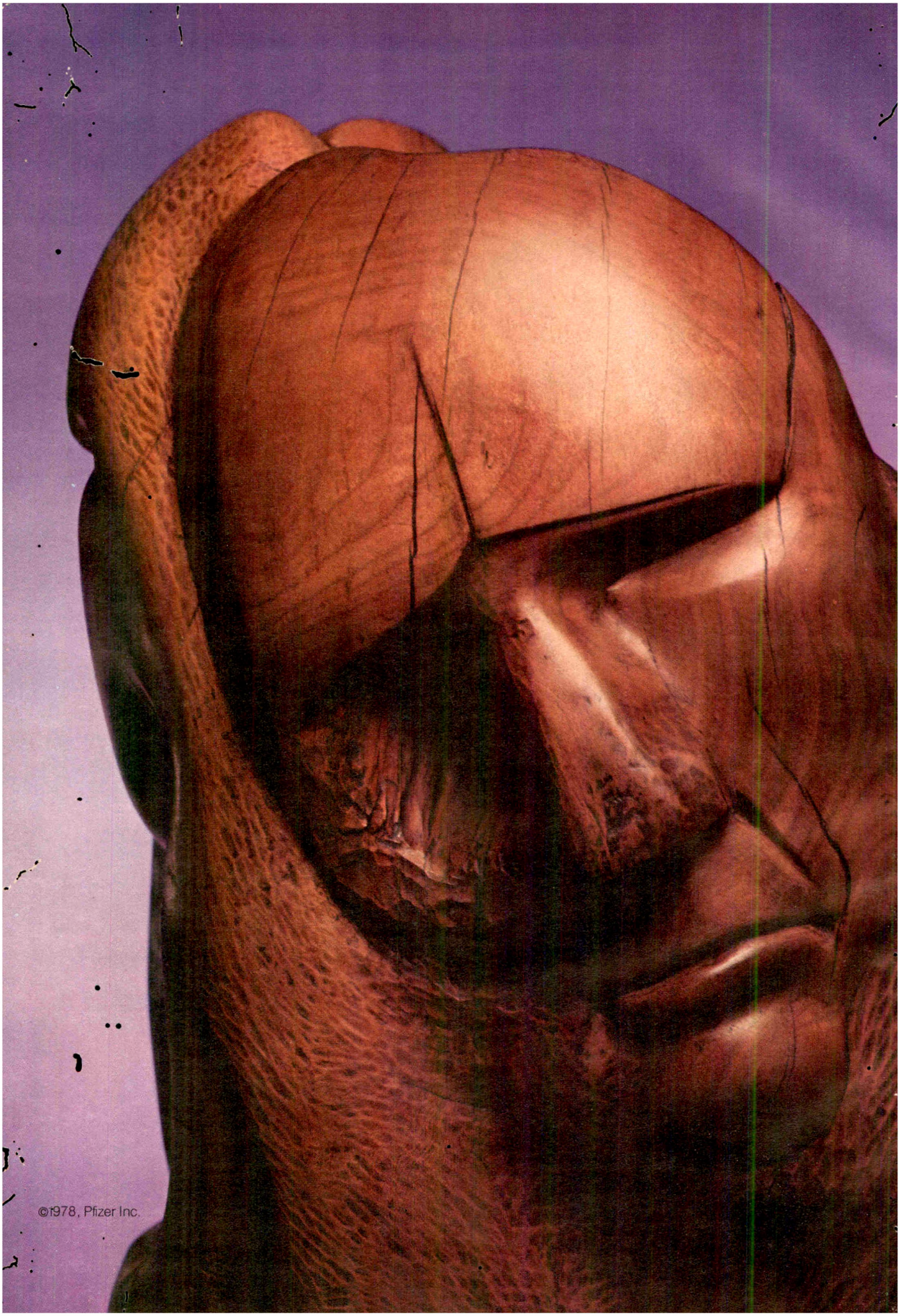
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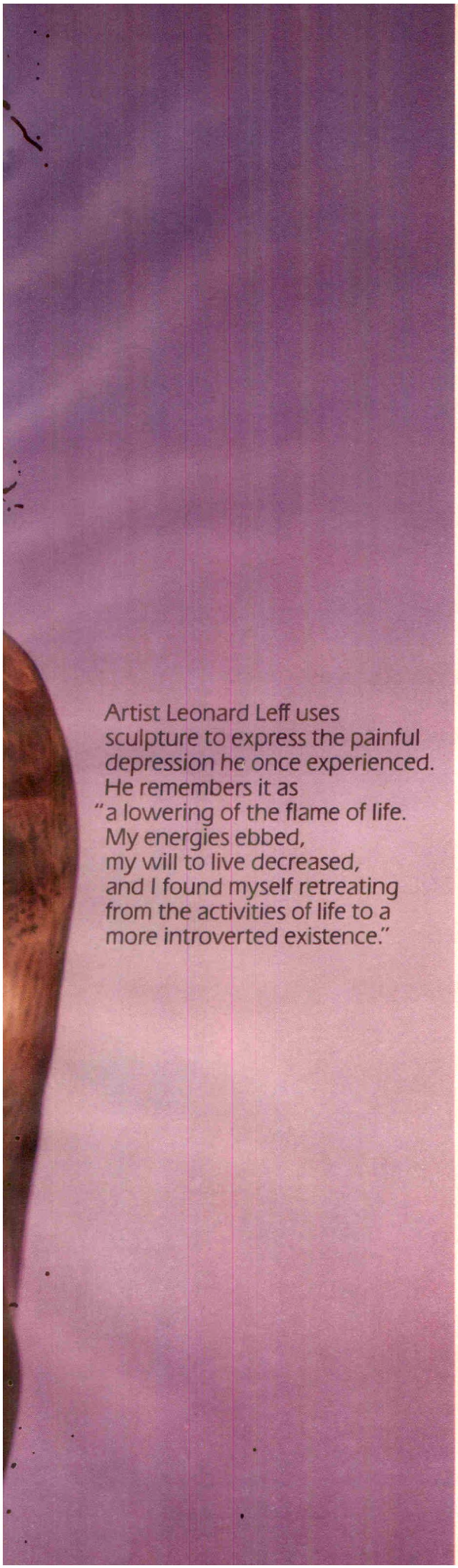
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Artist Leonard Leff uses sculpture to express the painful depression he once experienced. He remembers it as "a lowering of the flame of life. My energies ebbed, my will to live decreased, and I found myself retreating from the activities of life to a more introverted existence."

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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

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to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (*paralytic ileus*), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water, but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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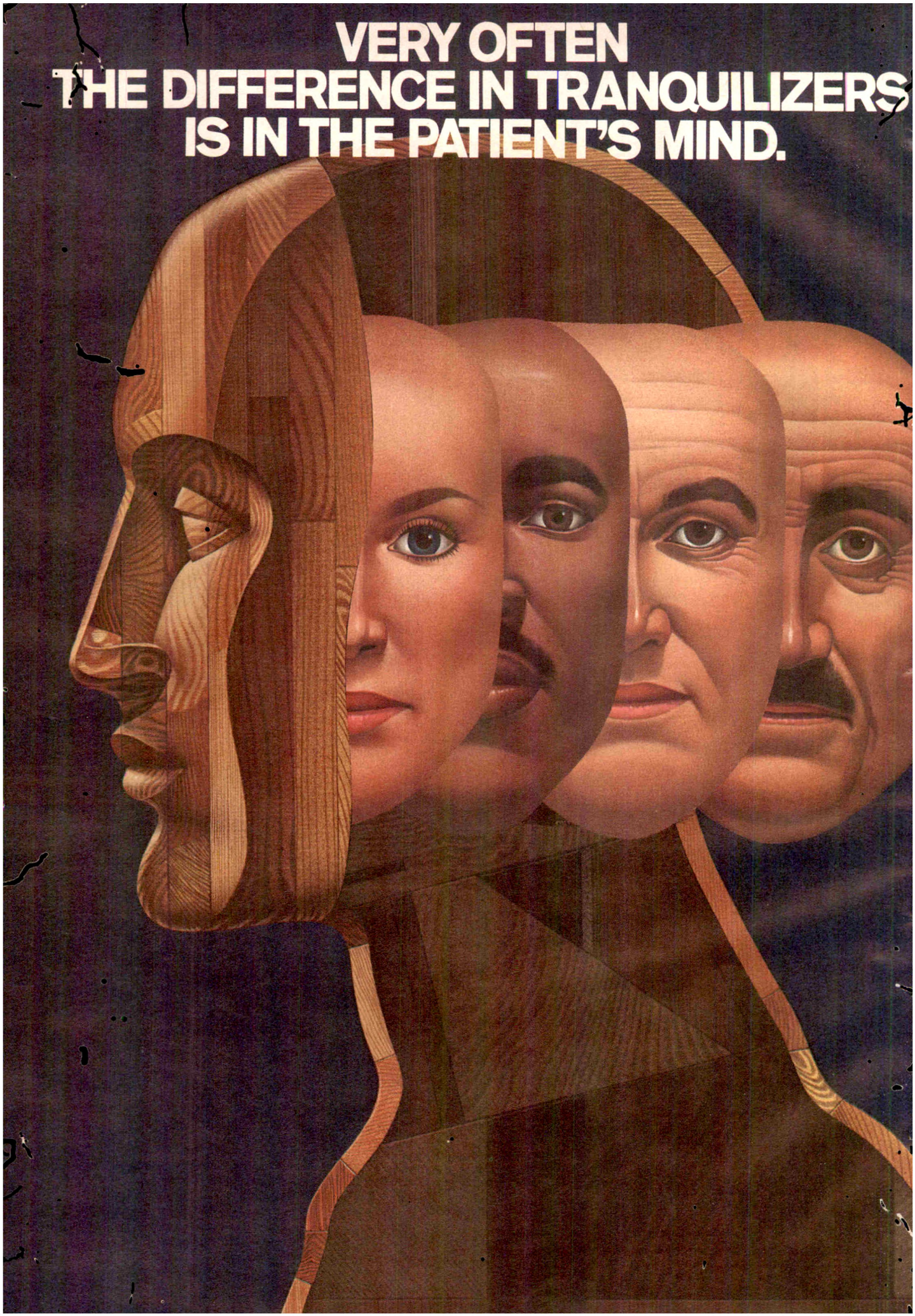
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AZENE® controls anxiety yet avoids excessive sedation in most patients. If sedation is a problem, the flexibility of the nighttime dosing may reduce or alleviate this problem. AZENE® is thus a logical choice for patients who want relief of anxiety without troublesome sedation.

*Of course, patients should be advised against engaging in hazardous activities that require mental alertness.

Please see brief summary of prescribing information on the following page.

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3.25 mg
6.5 mg
13 mg
capsules

CLORAZEPATE MONOPOTASSIUM C IV

A LOGICAL ADJUNCT TO PSYCHOTHERAPY

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AZENE®

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CLORAZEPATE MONOPOTASSIUM ℂ℣

BRIEF SUMMARY OF PRESCRIBING INFORMATION

CONTRAINDICATIONS AZENE® is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS AZENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on AZENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since AZENE (clorazepate monopotassium) has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, AZENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepates. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Tolerance developed within 3 to 9 days in dogs. Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. AZENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

AZENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Periodic blood counts and liver function tests are advisable for patients on AZENE for prolonged periods. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS The side effect most frequently reported with the use of benzodiazepines is drowsiness. Less commonly reported side effects are: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects include insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed with clorazepates.

DRUG INTERACTIONS If AZENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that clorazepates prolong the sleeping time after hexobarbital or after ethyl alcohol, increase the inhibitory effects of chlorpromazine, but do not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If AZENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

HOW SUPPLIED AZENE (clorazepate monopotassium) is supplied as capsules in three dosage strengths: 3.25 mg capsules (light tan-colored) in bottles of 100 and 500. 6.5 mg capsules (orange-colored) in bottles of 100 and 500. 13 mg capsules (blue-colored) in bottles of 100. 6092-3 BS

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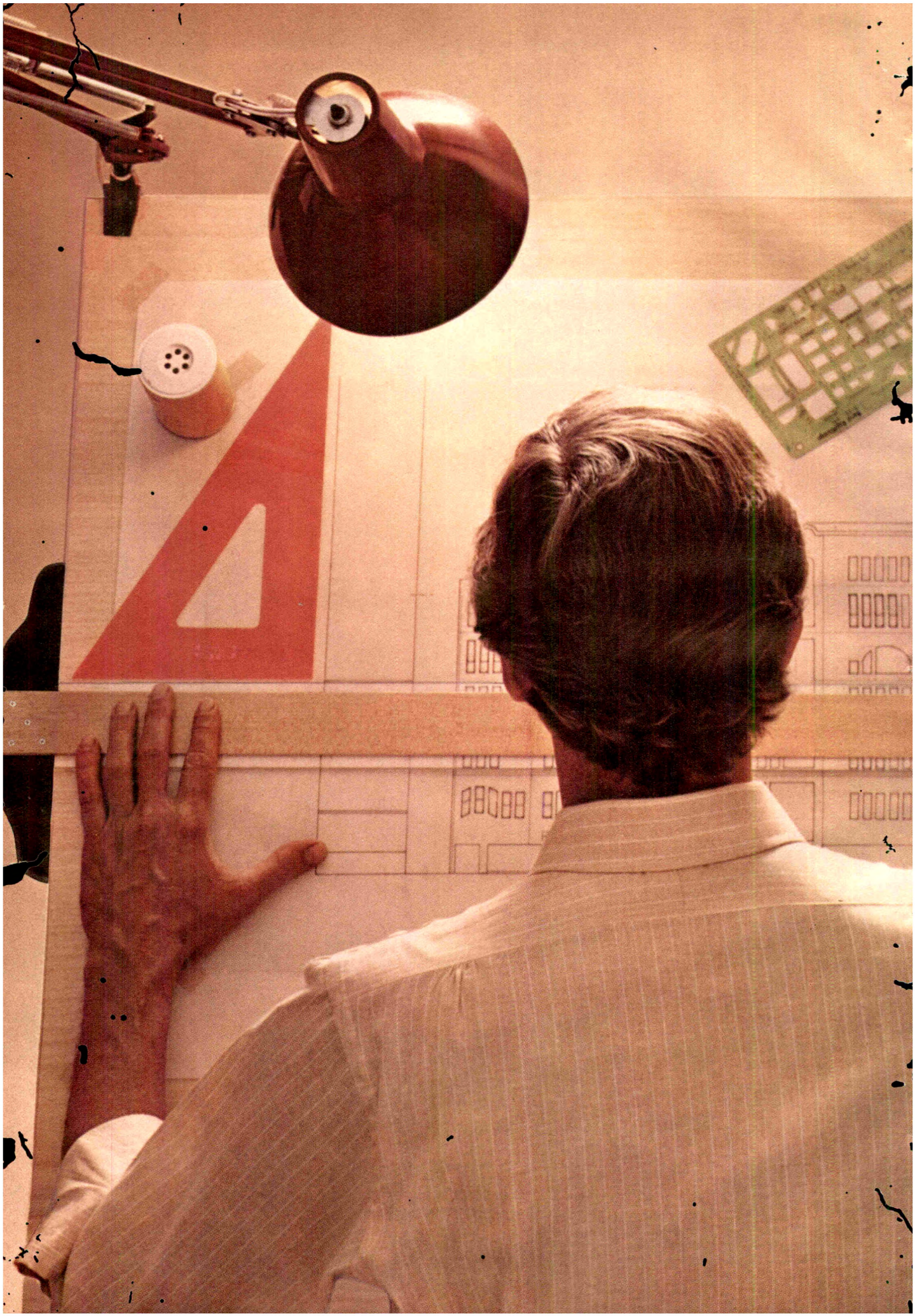
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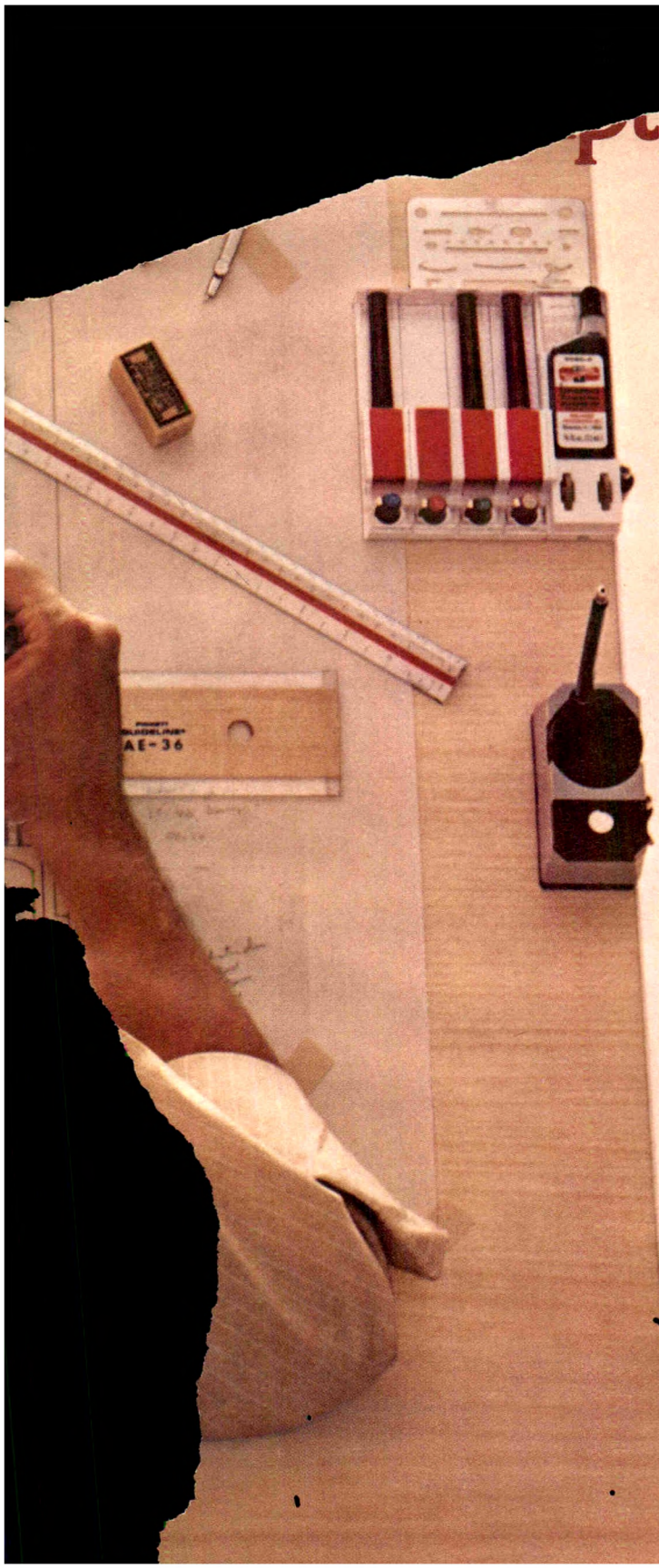
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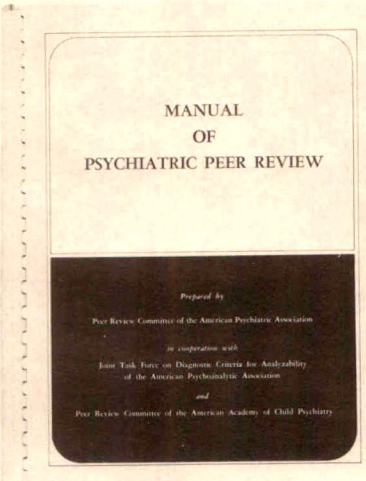
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"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

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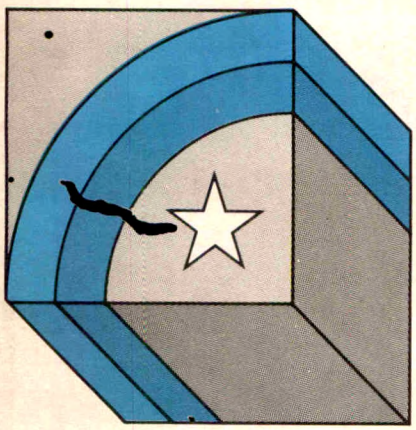
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Brief Summary

ACTIONS — Pharmacologically, clorazepate dipotassium has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine. (See CLINICAL PHARMACOLOGY section.)

INDICATIONS — TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS — TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS — TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS — In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In debilitated patients, the initial dose should be small, and increments made gradually, in accordance with the response of the patient, to avoid ataxia or excessive sedation.

ADVERSE REACTIONS — The side effect most frequently reported was drowsiness commonly reported (in descending order of occurrence) dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects include insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION

For the symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For the symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg. TRANXENE initially, followed by 30 to 60 mg. in divided doses; 2nd 24 hours, 45 to 90 mg. in divided doses; 3rd 24 hours, 22.5 to 45 mg. in divided doses; 4th day, 15 to 30 mg. in divided doses. Thereafter gradually reduce to 7.5 to 15 mg. daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS — If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE — Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE (clorazepate dipotassium) as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY — Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption — Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED — TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 30 (NDC 0074-3418-30), 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 30 (NDC 0074-2699-30) and 100 (NDC 0074-2699-13).

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Not drowsy by day, yet anxiety controlled

Troublesome drowsiness uncommon. Tranxene lets you manage anxiety in most patients without problem sedation. Any initial drowsiness is usually transient. By avoiding excessive sedation, Tranxene helps you calm patients without compromising their capacities.*

CAPSULES: 3.75, 7.5, 15 mg.
TABLETS: 11.25, 22.5 mg.

Tranxene®

(CLORAZEPATE DIPOTASSIUM)

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ABBOTT

*Of course patients should be cautioned against hazardous tasks requiring mental alertness.

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See overleaf for prescribing information.